Investor Presentation

June 2024

Disclaimer

Forward-Looking Statements

This presentation contains forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended, including statements regarding the future performance of 23andMe's businesses in consumer genetics and therapeutics and the growth and potential of its proprietary research platform. All statements, other than statements of historical fact, included or incorporated in this presentation, including statements regarding 23andMe's strategy, financial position, funding for continued operations, cash reserves, projected costs, plans, and objectives of management, are forward-looking statements. The words "believes," "anticipates," "estimates," "plans," "expects," "intends," "may," "could," "should," "potential," "likely," "projects," "continue," "will," "schedule," and "would" or, in each case, their negative or other variations or comparable terminology, are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. These forward-looking statements are predictions based on 23andMe's current expectations and projections about future events and various assumptions. 23andMe cannot guarantee that it will actually achieve the plans, intentions, or expectations disclosed in its forward-looking statements and you should not place undue reliance on 23andMe's forward-looking statements. The forward-looking statements contained herein are also subject generally to other risks and uncertainties that are described from time to time in the Company's filings with the Securities and Exchange Commission, including under Item 1A, "Risk Factors" in the Company's most recent Annual Report on Form 10-K, as filed with the Securities and Exchange Commission, and as revised and updated by our Quarterly Reports on Form 10-Q and Current Reports on Form 8-K. These forward-looking statements involve a number of risks, uncertainties (many of which are beyond the control of 23andMe), or other assumptions that may cause actual results or performance to be materially different from those expressed or implied by these forward-looking statements. Investors are cautioned not to place undue reliance on any such forward-looking statements, which speak only as of the date they are made. Except as required by law, 23andMe does not undertake any obligation to update or revise any forward-looking statements whether as a result of new information, future events, or otherwise.

Use of Non-GAAP Financial Measures

To supplement the 23andMe's unaudited condensed consolidated statements of operations and unaudited condensed consolidated balance sheets, which are prepared in conformity with generally accepted accounting principles in the United States of America ("GAAP"), this presentation also includes references to Adjusted EBITDA, which is a non-GAAP financial measure that 23andMe defines as net income (loss) before net interest income (expense), net other income (expense), changes in fair value of warrant liabilities, income tax benefit, depreciation and amortization of fixed assets, amortization of internal use software, amortization of acquired intangible assets, goodwill and intangible assets impairment, non-cash stock-based compensation expense, acquisition-related costs, and expenses related to restructuring and other charges, if applicable, for the period. 23andMe has provided a reconciliation of net loss, the most directly comparable GAAP financial measure, to Adjusted EBITDA at the end of this presentation.

Adjusted EBITDA is a key measure used by 23andMe's management and the board of directors to understand and evaluate operating performance and trends, to prepare and approve 23andMe's annual budget and to develop short- and long-term operating plans. 23andMe provides Adjusted EBITDA because 23andMe believes it is frequently used by analysts, investors and other interested parties to evaluate companies in its industry and it facilitates comparisons on a consistent basis across reporting periods. Further, 23andMe believes it is helpful in highlighting trends in its operating results because it excludes items that are not indicative of 23andMe's core operating performance. In particular, 23andMe believes that the exclusion of the items eliminated in calculating Adjusted EBITDA provides useful measures for period-to-period comparisons of 23andMe's business. Accordingly, 23andMe believes that Adjusted EBITDA provides useful information in understanding and evaluating operating results in the same manner as 23andMe's management and board of directors.

In evaluating Adjusted EBITDA, you should be aware that in the future 23andMe will incur expenses similar to the adjustments in this presentation. 23andMe's presentation of Adjusted EBITDA should not be construed as an inference that future results will be unaffected by these expenses or any unusual or non-recurring items. Adjusted EBITDA should not be considered in isolation of, or as an alternative to, measures prepared in accordance with GAAP. Other companies, including companies in the same industry, may calculate similarly-titled non-GAAP financial measures differently or may use other measures to evaluate their performance, all of which could reduce the usefulness of Adjusted EBITDA as a tool for comparison. There are a number of limitations related to the use of these non-GAAP financial measures rather than net loss, which is the most directly comparable financial measure calculated in accordance with GAAP. Some of the limitations of Adjusted EBITDA include (i) Adjusted EBITDA does not properly reflect capital commitments to be paid in the future, and (ii) although depreciation and amortization are non-cash charges, the underlying assets may need to be replaced and Adjusted EBITDA does not reflect these capital expenditures. When evaluating 23andMe's performance, you should consider Adjusted EBITDA alongside other financial performance measures, including net loss and other GAAP results.

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Industry and Market Data

This Presentation relies on and refers to certain information and statistics based on 23andMe's management's estimates, and/or obtained from third party sources which it believes to be reliable. 23andMe has not independently verified the accuracy or completeness of any such third party information.

Our mission is to help people access, understand, and benefit from the human genome.

23andMe Customers
from Around the World

We are building value with three business verticals based on genetics

To achieve our three-part mission, we are executing across three different businesses.

Consumer

Personalized Health: genome, exome, lab (blood) work

Telehealth & Telepharmacy (Lemonaid Health)

Ancestry & DNA Relatives

Recurring subscription revenue

Research

Worlds largest re-contactable genetic and phenotypic data engine

Database licensing

Target discovery

Commercial and pharma services

Therapeutics

Genetics-informed targets, biologically validated

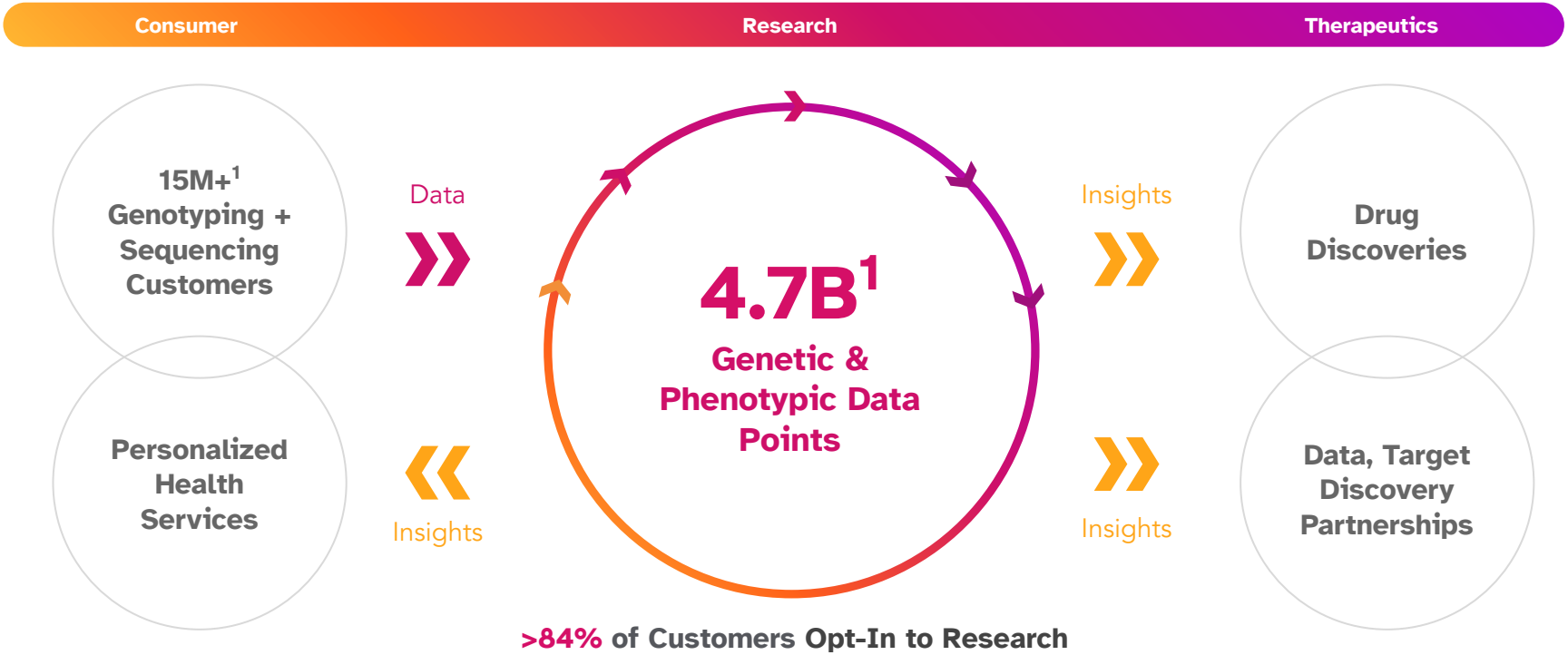
Lead IO asset ‘610 enrolling phase 2A

IO asset ‘1473 enrolling Phase 1

Early-stage Immunology and Inflammation pipeline

They power our consumer-driven healthcare flywheel

All three businesses are powered by our dynamic health data engine, allowing us to run hundreds of billions of association tests per year to build the future of genetics-driven healthcare.



1. as of March 31, 2024.

Our unprecedented scale enables impactful, novel, personalized health

With our growing database, we are uniquely positioned to understand human biology across areas of consumer health, research and therapeutics unlike any other genetics program globally.

23andMe	15M+[1]
REGENERON	~2M+
MILLION VETERAN PROGRAM	1M
OUR FUTURE HEALTH	800,000+
ALL OF US	540,000+
UK BIOBANK	500,000
DECODE GENETICS	500,000
FINNGEN	473,000+

1. Genotyped customers as of March 31, 2024.

1

Consumer

Transforming Healthcare with Genetic Health Services at Scale

A recent study[1] showed that **1 in 25** people have a **medically actionable** genetic variant[2] that is associated with reduced lifespan.

1. https://www.nejm.org/doi/pdf/10.1056/NEJMoa2300792
2. https://www.ncbi.nlm.nih.gov/clinvar/docs/acmg/

Genetics plays a role in 8 of the 10 leading causes of death in the US[1]

1. Heart disease

2. Cancer

3. Accidents (unintentional injuries)

4. COVID-19

5. Stroke (cerebrovascular diseases)

6. Chronic lower respiratory diseases

7. Alzheimer's disease

8. Diabetes

9. Nephritis, nephrotic syndrome, and nephrosis

10. Chronic liver disease and cirrhosis

= Addressed by 23andMe genetic report

1. https://www.cdc.gov/nchs/data/databriefs/db492-tables.pdf#4

Risk prediction and prevention can drive better health outcomes

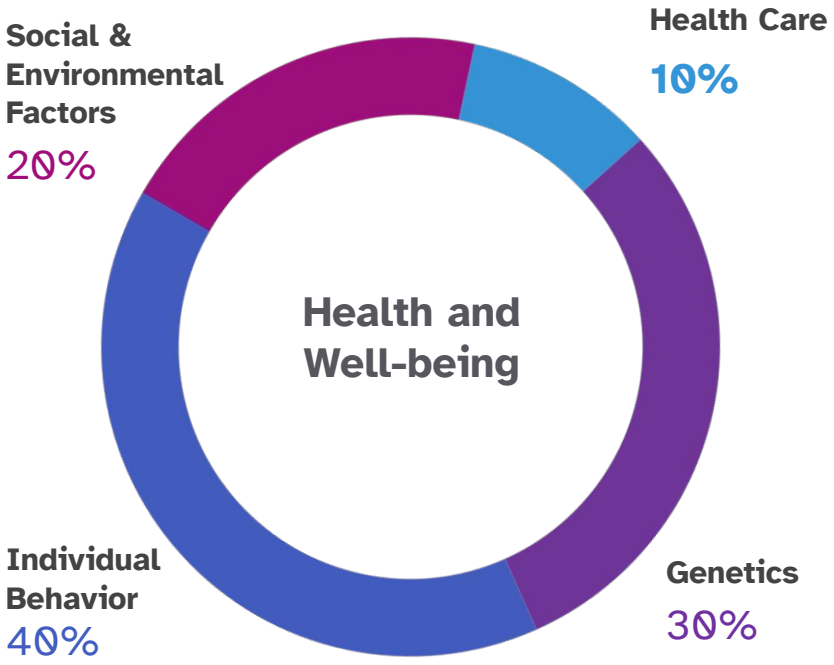
1. Heart disease	**80% preventable**[1]
2. Cancer	**40% preventable**[2]
3. Accidents (unintentional injuries)	
4. COVID-19	
5. Stroke (cerebrovascular diseases)	**80% preventable**[1]
6. Chronic lower respiratory diseases	**39% preventable (emphysema >90%)**[3]
7. Alzheimer's disease	**Up to 40% preventable**[4]
8. Diabetes	**80% preventable**[5]
9. Nephritis, nephrotic syndrome, and nephrosis	**CKD up to 50% preventable**[6]
10. Chronic liver disease and cirrhosis	**90% are preventable**[7]

= Addressed by 23andMe genetic report

1. https://www.sciencedaily.com/releases/2019/01/190131084238.htm
2. https://acsjournals.onlinelibrary.wiley.com/doi/10.3322/caac.21440
3. https://www.cdc.gov/mmwr/preview/mmwrhtml/mm6317a1.htm
4. https://www.cdc.gov/aging/publications/features/dementia-risk-reduction-june-2022/
5. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3109209/
6. https://pubmed.ncbi.nlm.nih.gov/23171953/
7. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10131973/pdf/JFMPC-12-419.pdf

Today's health care system only has a 10% impact[1] on our health and well being.

Impact of Different Factors on Risk of Premature Death[1]



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

23andMe is helping people identify their genetic risks...and take action

Monica, 23andMe Customer
Found she had increased risk for Breast and Ovarian Cancer.

28K+

with **BRCA1/BRCA2 variant** with up to 85% higher genetic health risk for breast and ovarian cancer.

Robert, 23andMe Customer
Found he had increased likelihood of developing Type 2 Diabetes.

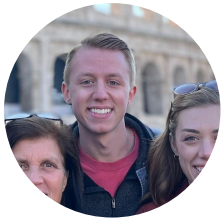
4M+

with higher likelihood of **type 2 diabetes**.

Kim, 23andMe Customer
Found she had an increased likelihood of developing Coronary Artery Disease.

2.2M+

with higher likelihood with **coronary artery disease**.



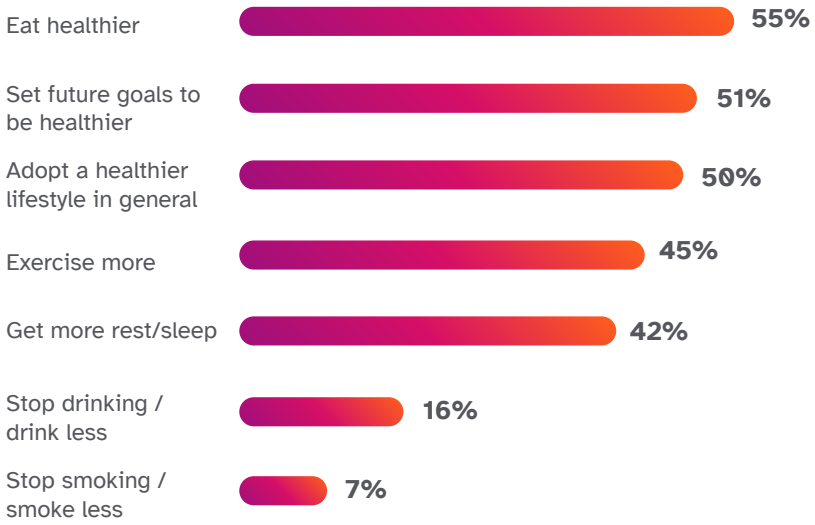
Andrew, 23andMe Customer
Found he had increased risk for Hereditary Thrombophilia.

1M+

at high genetic health risk for **hereditary thrombophilia** (harmful blood clots)

All stats current as of May 21, 2024 from 23andMe Database.

76% of customers report taking a positive health action after learning about their genetics[1]



1. Based on 2019 online survey, designed by 23andMe and M/A/R/C Research, of 1,046 23andMe Health + Ancestry customers.

Our success is driven by strong engagement and trust

Providing a meaningful, engaging and fun experience.

84%
US customers consent to research

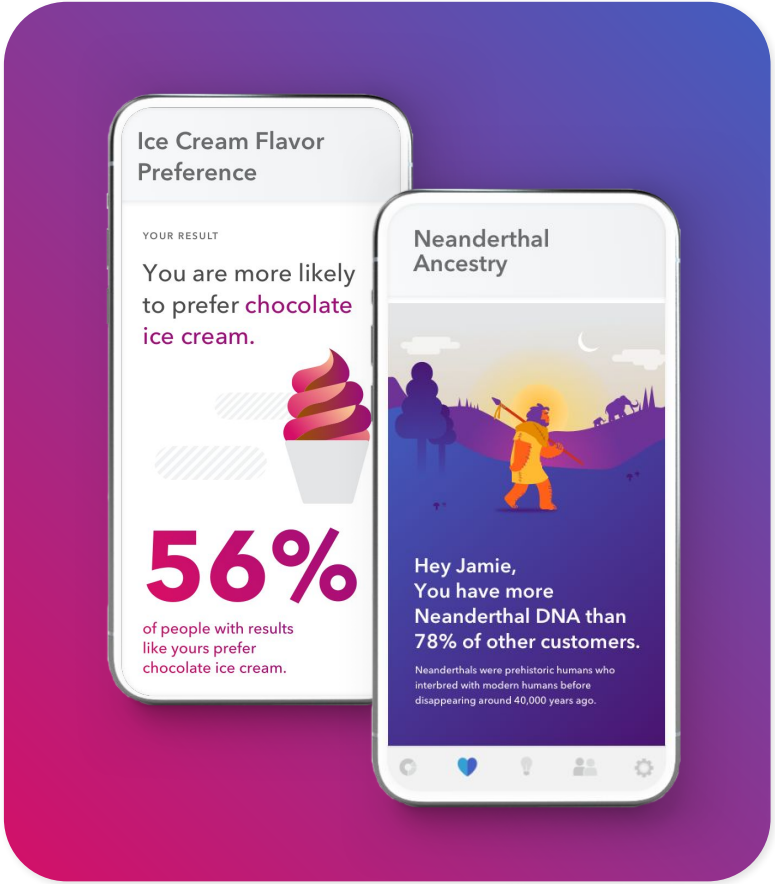
15K
research surveys completed daily

4.7B
phenotypic data points

230+
published research papers

8.9M
genotyped customers logged-in in past year

50%
pre-2020 customers logged-in in past year



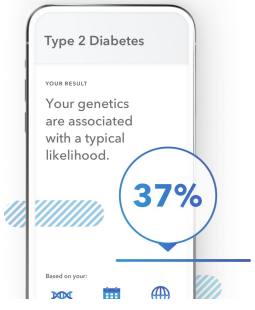
All stats current as of May 21, 2024

Turning personalized health learnings into actionable insights

23andMe Personal Genetic Services

9 FDA Authorizations

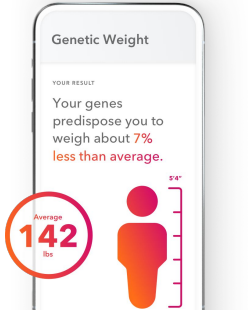
Health Predispositions [1]



30+ reports including:

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

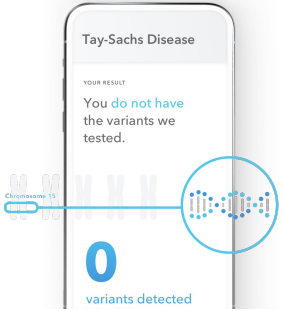
Wellness [2]



10 reports including:

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

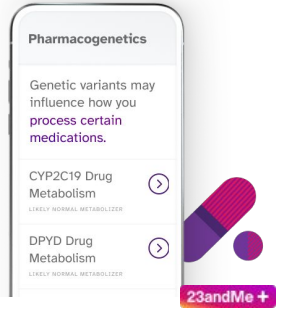
Carrier Status



40+ reports including:

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

Pharmacogenetics



6 reports including:

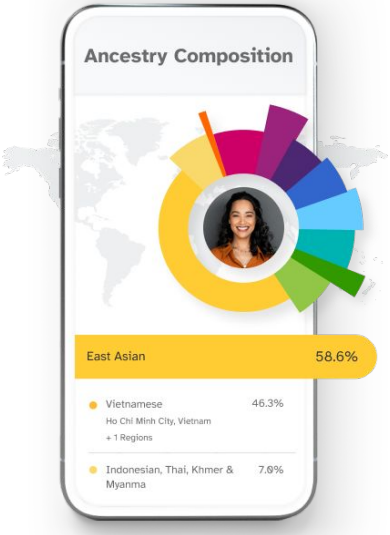
SLCO1B1 Drug Transport
  e.g., simvastatin
CYP2C19 Drug Metabolism
  e.g., citalopram and clopidogrel
DPYD Drug Metabolism

1. Includes FDA Authorized Genetic Health Risk Reports and Wellness Reports for Genetic Likelihood Powered by 23andMe Research.
2. Wellness information does not require FDA Authorization.

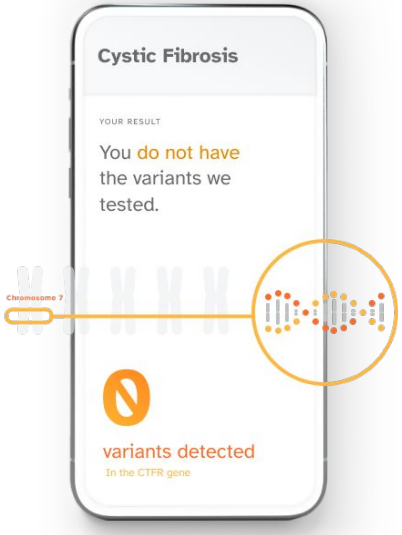
We have a genetic service for every type of customer

Memberships

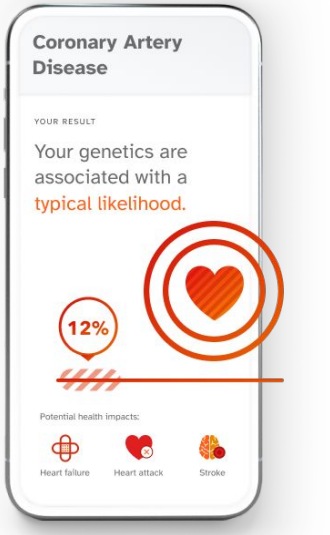
Ancestry Service



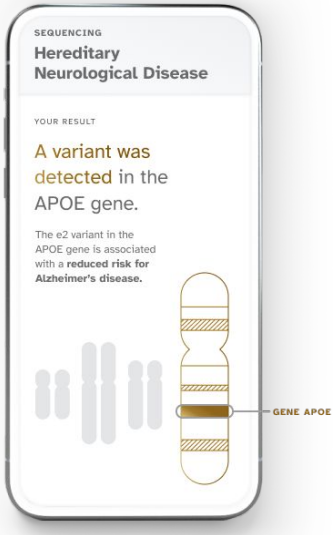
Health + Ancestry



23andMe+ Premium™

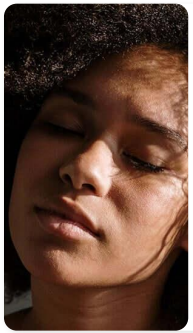


23andMe+ Total Health™



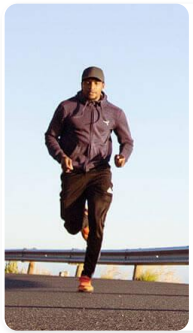
We offer direct access to care with *Lemonaid Health Telehealth Services*

With a growing menu of options



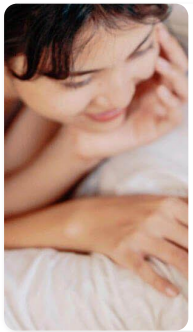
Mental Health

Anxiety
Depression
Insomnia
Seasonal Affective Disorder



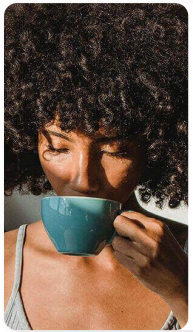
Men's Health

Erectile Dysfunction
Premature Ejaculation
Hair Loss



Women's Health

Birth Control
Morning-After Pill
UTI
Hot Flashes



General Health

Cold Sores
Genital Herpes
Sinus Infection
Primary Care Complete
AND MORE

Skin

Acne
Dark Spots

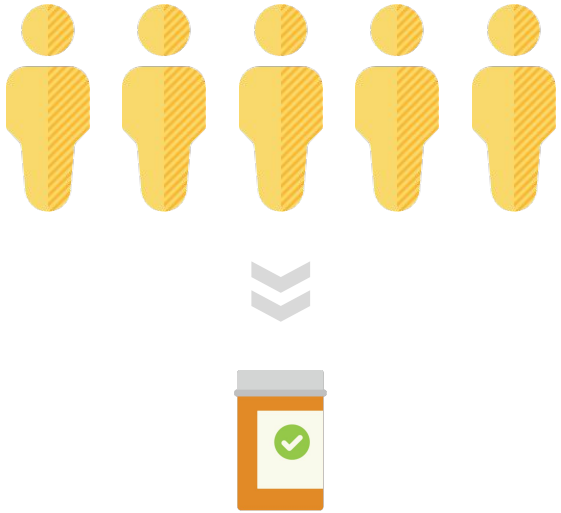
Testing

STD Test
A1C Blood Sugar Test
Cholesterol Test
Blood Type Test

23andMe helps consumers take a proactive approach to their health

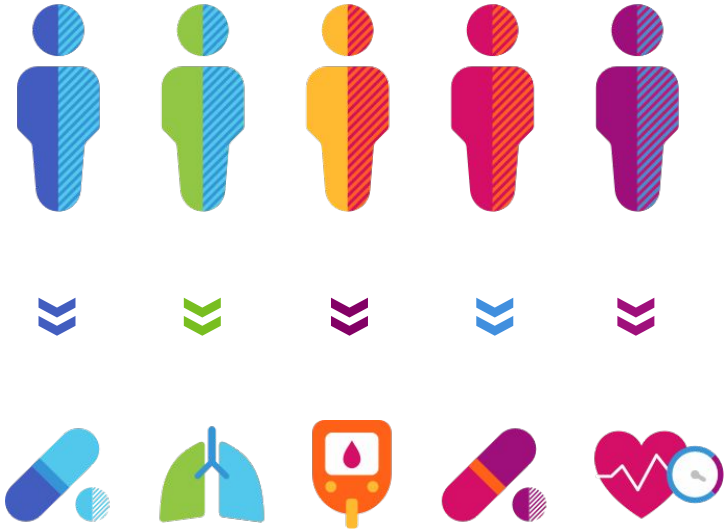
Practice of Medicine Today

Reactive - no customization until symptomatic



23andMe

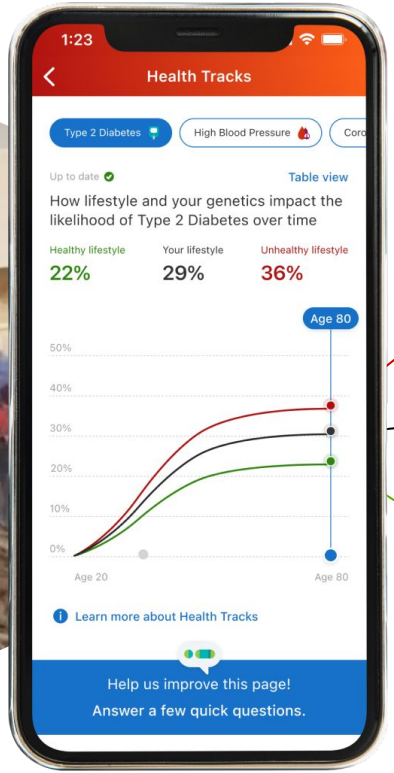
Proactive - truly individualized from the beginning



Giving everyone the opportunity to change their health trajectory

"I can't change the DNA but I can change what I do on a daily basis to help mitigate that."

Tracy
23andMe Customer
Discovered she has a higher genetic likelihood for developing Type 2 Diabetes.



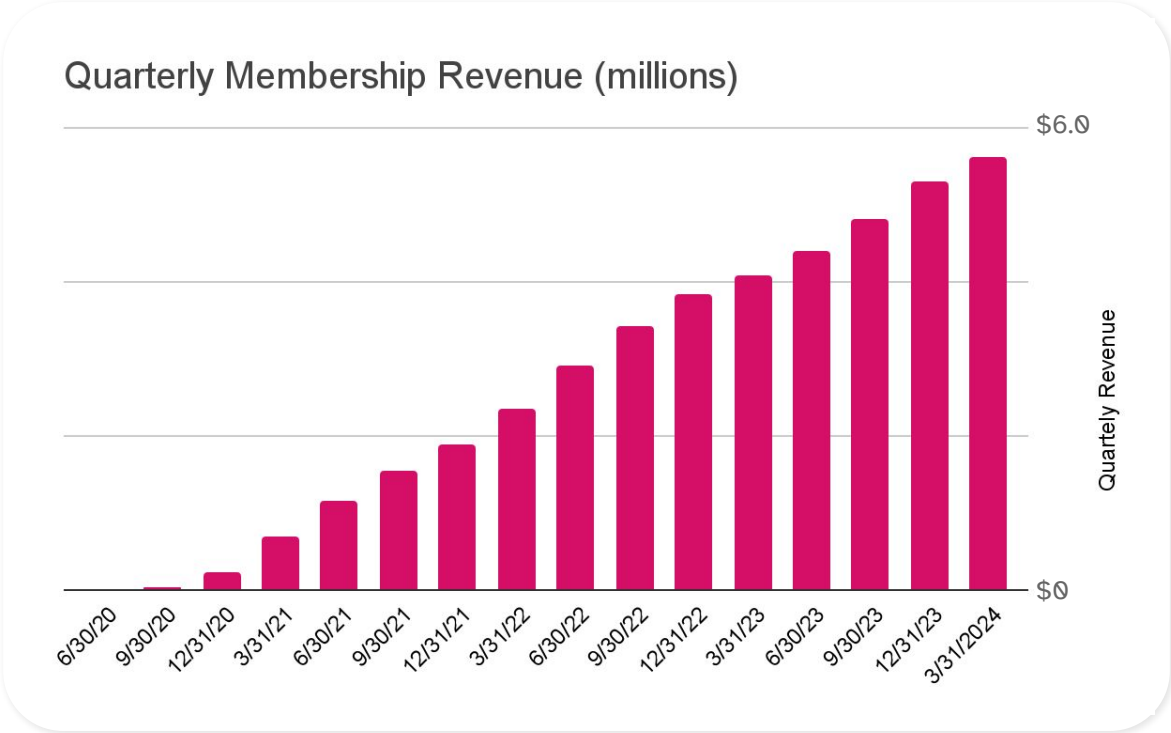
Unhealthy lifestyle
A regression of lifestyle would lead to a Type 2 diabetes risk 36% by age 60

Current lifestyle
Continuing with existing lifestyle results in a Type 2 diabetes risk 29% by age 60

Healthy lifestyle
Lifestyle changes can reduce Type 2 diabetes risk to 22% by age 60

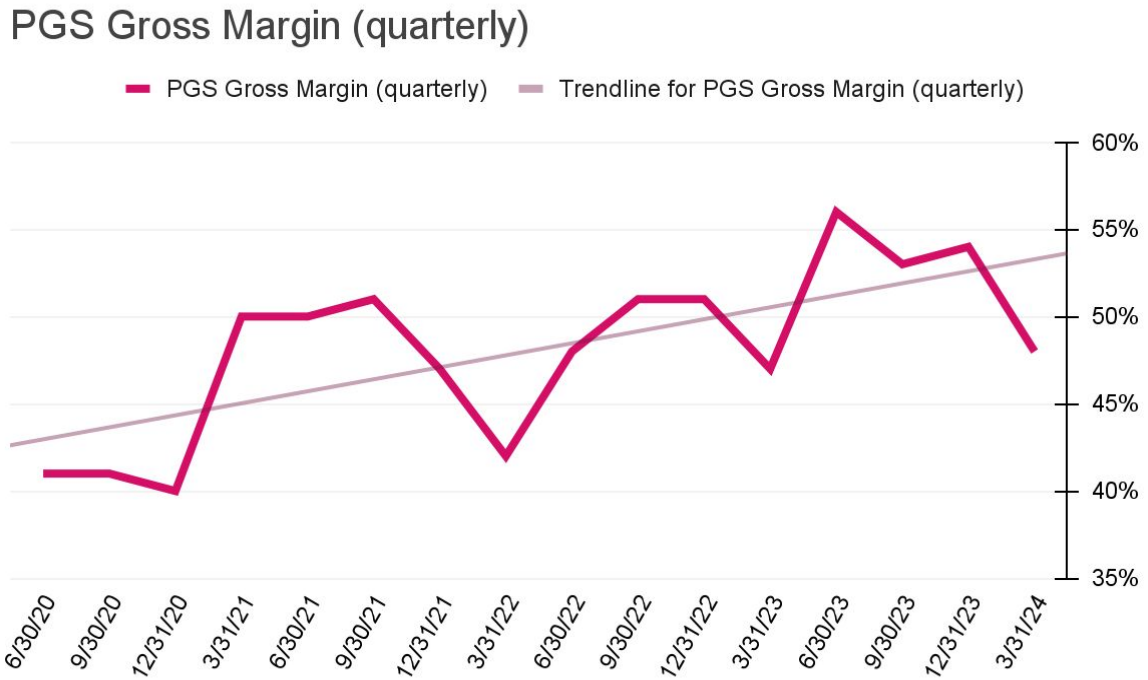
23andMe White Paper: Health Tracks: Time to Event Modeling of Common Conditions Using Polygenic Scores and Lifestyle Factors.
https://permalinks.23andme.com/pdf/23_24-HealthTracksMethodology.pdf

We are prioritizing membership revenue growth



- Prioritizing growth in sustainable, recurring revenue business
- Building out value-add features and products
- Recently launched Health Action Plan™, Health Tracks™ and 23andMe+ Total Health™
- FY 2024 PGS revenue of $168M with subscription revenue of $20M

Improving margins and driving toward profitability



- Steadily improving gross margin despite seasonality
- Margin tailwinds from increasing subscription revenue and price optimization
- Strong new product uptake would further positively impact consolidated GM over time

We are delivering a healthier future, and we are just getting started

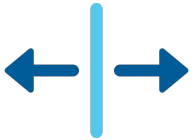
The future is...

All of our current services and support

New Telehealth and Pharmacy services

AI integration into health tracking tools

Precision Prescribing Using Pharmacogenetics



Long-term Engagement

All connected within a single technology platform.

2

Research

Providing Unique Value and Insights for Research Partners

The world's largest recontactable genetic data engine

- Participation is online
- Fully opt-in, and opt-out at any time
- IRB approved
- Everyone can be included in multiple studies

1. as of March 31, 2024.

Scale enables differentiated research across multiple disease areas

Phenotype	Number of Cases[1]
Asthma	1.1M
Autoimmune	
Lupus	58k
Multiple Sclerosis	31.5k
Type 1 Diabetes	38.5k
Solid Tumors	> 1M
Basal Cell	388k
Squamous Cell	214k
Melanoma	125k
Breast	120k
Hematologic Cancers	
NHL	17k
Leukemia	14k

Phenotype	Number of Cases[1]
Retinal Diseases	
AMD	106k
Glaucoma	186k
Rare Diseases	
Scleroderma/SSc	12k
Sarcoidosis	9.3k
Idiopathic Pulmonary Fibrosis	5k
Neurology + Psychiatry	
Depression	1.8M
Parkinson's	33.5k
Essential Tremor	47k

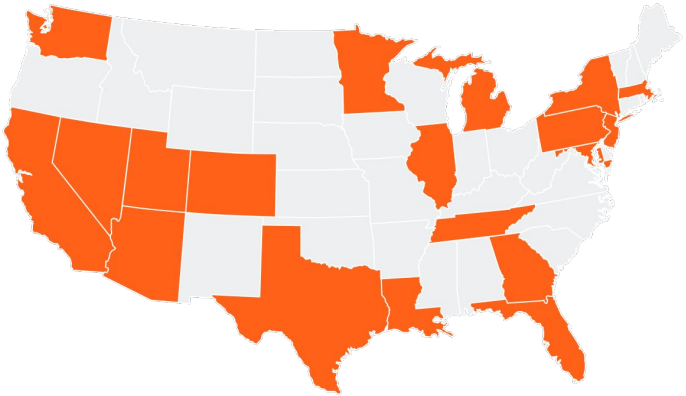
Numbers represent the number of research participants with the condition indicated

1. 23andMe multi-ancestry meta-analysis GWAS as of October 2023

Re-contactable customers participate in health research

- Research participants can be recontacted on the basis of phenotype or genetics for additional data or biosample collection.
- Example: Working with a mobile phlebotomist, we obtained blood draws from >60 human knockouts with a rare loss of function variant
  - Applied clinical lab testing for lipids, liver function, kidney function, glucose levels, heart function, and CBC counts

Geographic distribution of participants



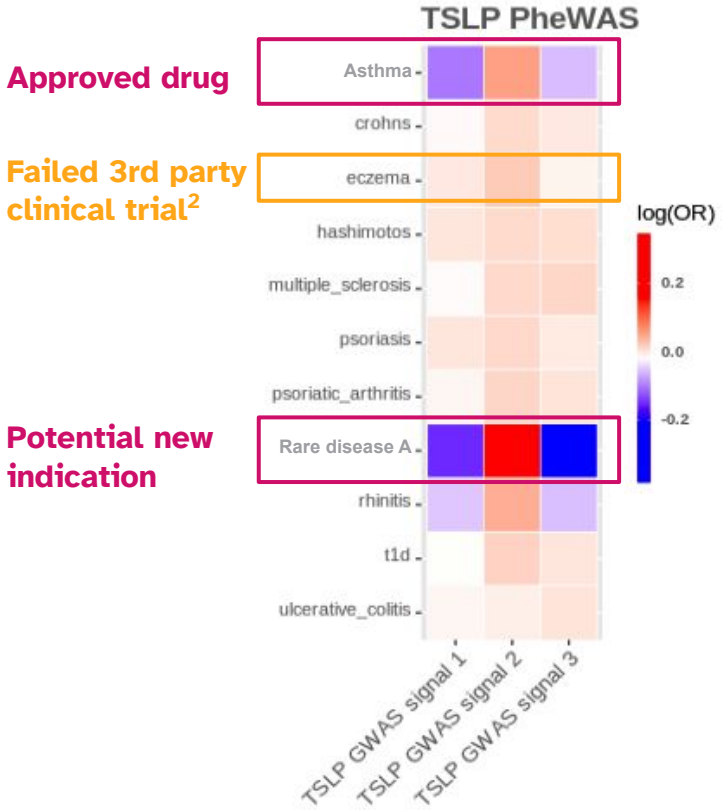
Breadth of phenotyping provides deeper genetic understanding beyond single diseases

Our insights can increase development efficiency and chances of clinical success

Drugs with human genetic support are

2x-3x

more likely to succeed[1]

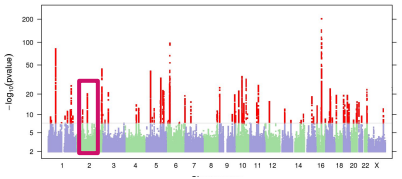


[1]*Nelson et al., 2015 (Nature Genetics); King et al., 2019 (PLOS Genetics).*

[2]https://www.astrazeneca.com/content/dam/az/PDF/2017/Q3/Year-to-date_and_Q3_2017_Results_Announcement.pdf

23andMe's GWAS and PheWAS:

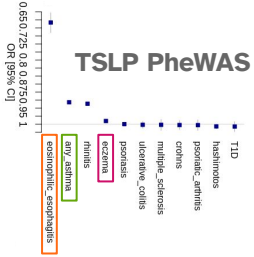
Unparalleled, Proven Resource for Novel Target Discovery



GWAS results are building blocks for target discovery:

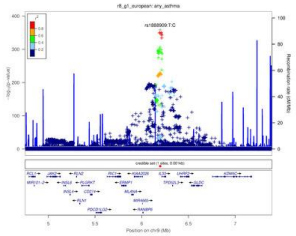
GWAS signals across the whole genome identify gene / phenotype associations and potential drug targets

Additionally, implicated pathways and point to underlying disease biology



23andMe runs GWAS in >1,000 phenotypes

PheWAS (Phenome-Wide Association Study) captures pleiotropic effects of genetic variants and points to possible unwanted toxicities or potential indication expansions



23andMe developed major methodological improvements to interrogate biology via GWAS

GWAS signal-to-gene mapping, including novel ML methods and experimental / FxG validation

Improved imputation panels and strategic whole exome sequencing approaches

A new paradigm for 23andMe research:

2017 — Mid-2023 — Late-2023 — Future

Exclusive drug discovery and development collaboration with GlaxoSmithKline (GSK)

- $25-50M annual contract fee
- Co-development of targets
- Over 50 targets discovered
- Limited 23andMe control of costs
- Resource intensive
- Difficult to forecast due to cost sharing

Non-exclusive research collaborations

- Database access, focused target discovery, portfolio optimization
- Full 23andMe control of costs
- Deal specific resource scaling
- Higher margin
- Easy to forecast
- Ex: GSK -$20M/yr database access

Exploring multiple types of collaborations and partnerships

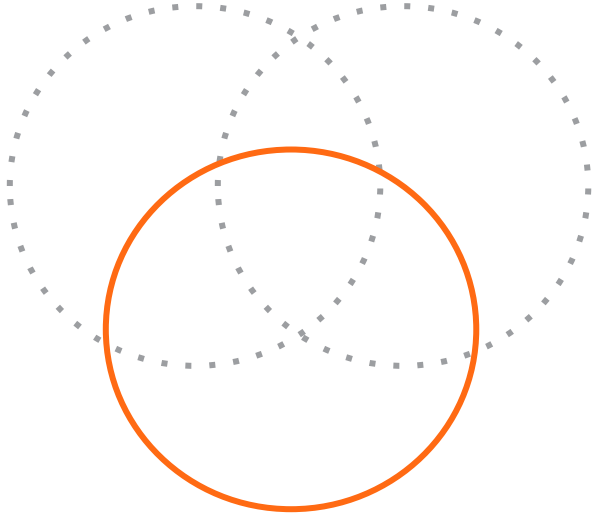
Potential Deal Types	Database Access	Target Discovery*	Portfolio Optimization
Capabilities and Structure	• Non-exclusive deals • Annual access fee • **Example:** GSK paying $20M for 6th year of access	• Multiple targets in a therapeutic area • Upfronts • Royalties • Milestones	• Portfolio screening • Indication validation • Patient population optimization
Target Partners	Pharma / Biotech	Pharma / Biotech	Pharma / Biotech

*Also pursuing other capabilities and structures

23andMe is well placed to realize the potential of AI in health and genetics

We are investing in AI to drive the next wave of insights and value-creation for our customers and partners

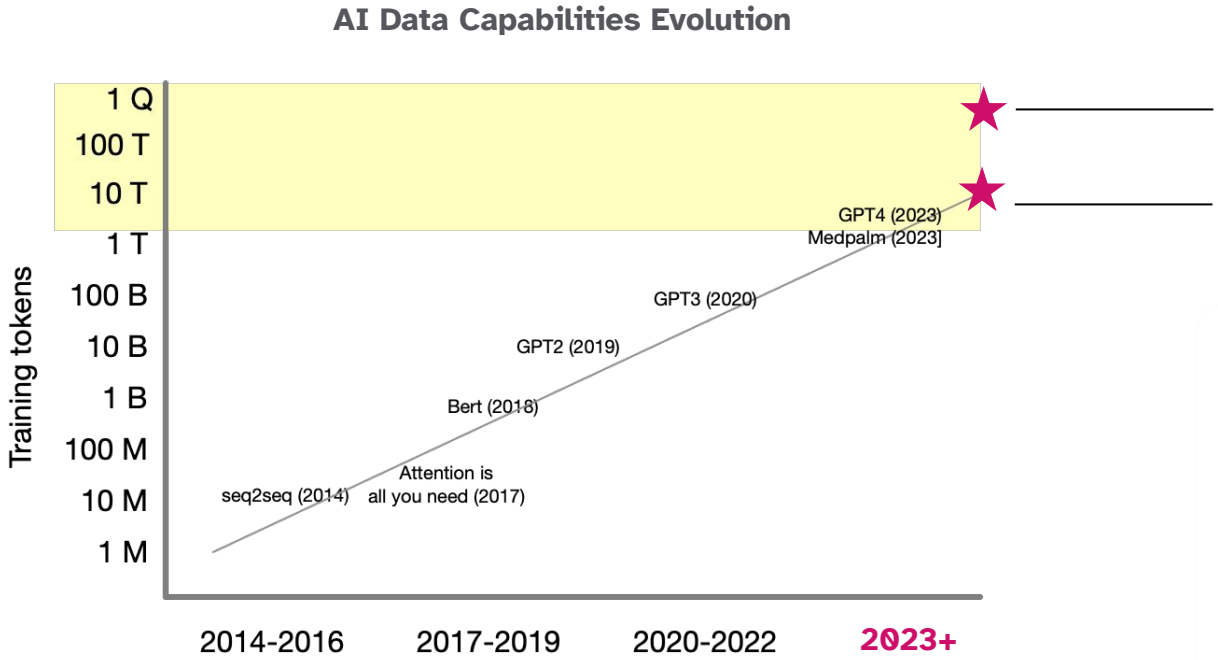
AI/ML expertise



AI technologies, models for health and pharma

Large-scale, relevant, *unique data*

Advances in AI methods can now handle the scale of our data



23andMe data today

~100M imputed SNPs x ~100s Phenotypic labels x ~10M Individuals

~1M genotyped SNPs x ~100s Phenotypic labels x ~10M Individuals

The next 5-10 years:
Genetic & Phenotypic data + Wearables + Omics → **(truly) predictive models of health and disease**

*number of tokens estimated from primary papers, press-releases, and other public information

Foundational pillars of our AI strategy will support future innovation

Health Prediction:
Health language models

Health events models

Wearable models

Health trajectories and recommendations

Biological insights:
Genome language models

Protein language model

DNA language model

Disease causality and therapeutic insights

3

Therapeutics

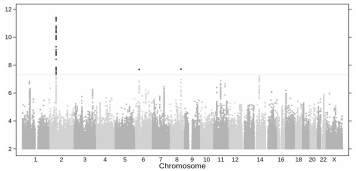
Turning Data at Scale into New Treatments for Patients

The evolution of 23andMe Therapeutics

2015 — Today

2015

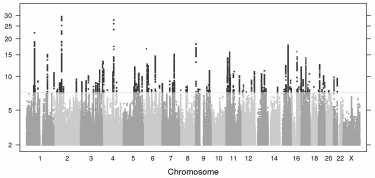
23andMe Tx Began



Multiple programs identified to be brought forward independently

July 2018 - July 2023

GSK Collaboration

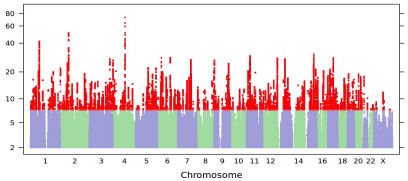


Incredibly productive multi-modality drug discovery collaboration with GSK across many therapeutic areas

50+ programs

August 2023 - Today

Full-fledged Biotech



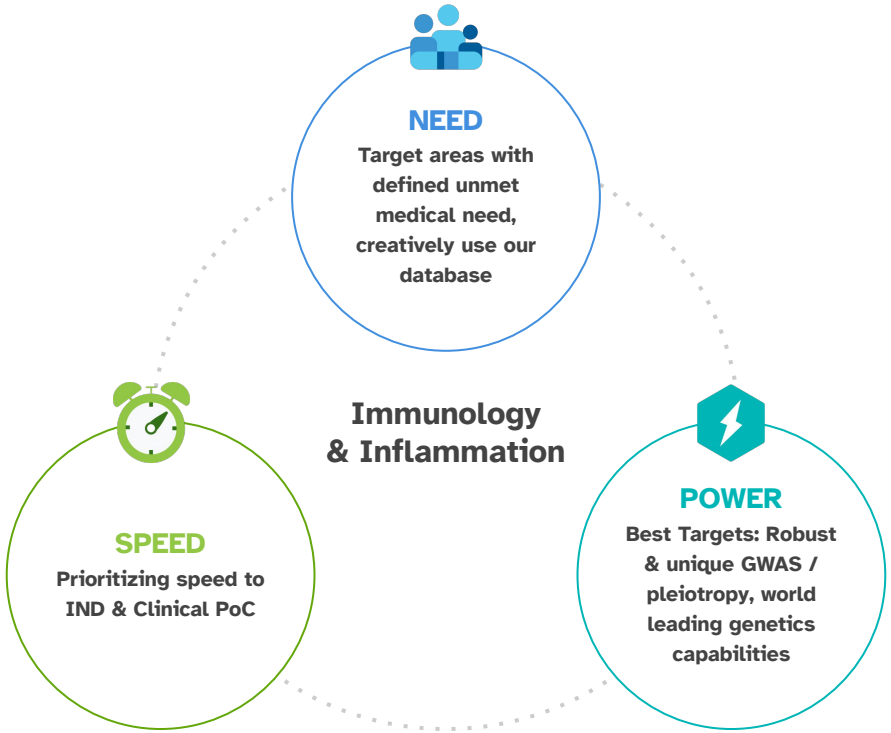
Two novel, clinical stage Oncology antibody assets

Discovery focus on Immunology and Inflammation

In-silico target discovery, functional genomics, antibody design and wet-lab validation

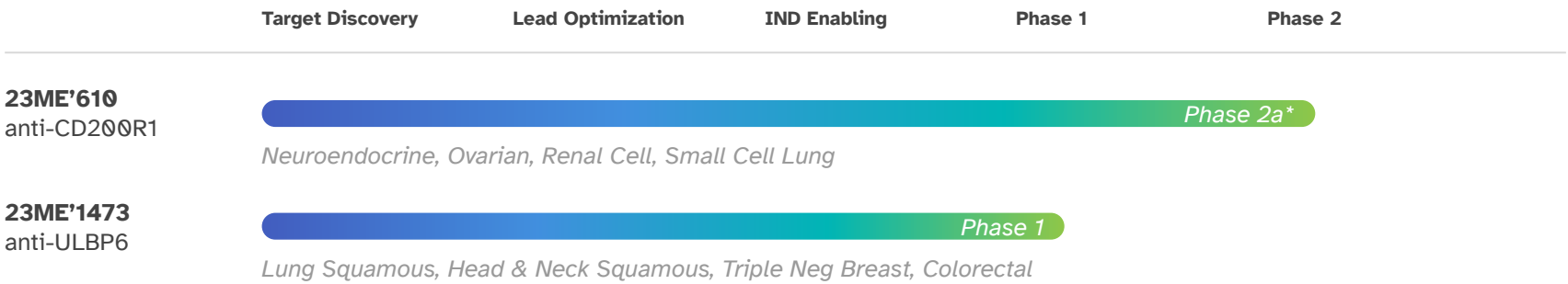
Our Therapeutics discovery platform

Capitalizing on 23andMe's Capabilities & Genetic Advantage



23andMe Therapeutics development pipeline:

First-in-class potential in oncology



23ME'610/anti-CD200R1

- Potent monotherapy Ab with PK/PD/tolerability profile indicating excellent combination potential
- Ph2a monotherapy basket (including neuroendocrine and ovarian) ongoing, with emerging clinical benefit
- Tumor CD200 as potential prognostic biomarker for optimal patient identification
- **Ph2a monotherapy data throughout 2024**

23ME'1473/anti-ULBP6

- Effector-enhanced Ab with dual NK-activating MOA
- Targets major resistance mechanisms hampering IO
- **Ph1 ongoing**

*Note: As of January 2024, '610 is in the Ph2a portion of the Phase 1/2a clinical trial.

23ME-00610*

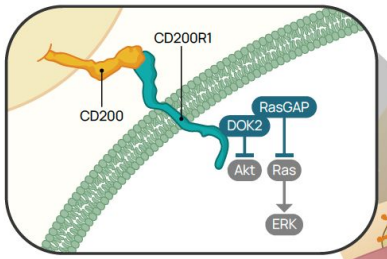
Anti-CD200R1 Antibody for Hard-to-Treat Solid Tumors

Phase 1/2a

**Development ongoing in multiple relapsed/refractory solid tumors (including neuroendocrine and ovarian)*

CD200-CD200R1 immune suppression

CD200-CD200R1 binding initiates signaling via DOK2 and RasGAP to downregulate proinflammatory cytokine production, contributing to an immunosuppressive TME.



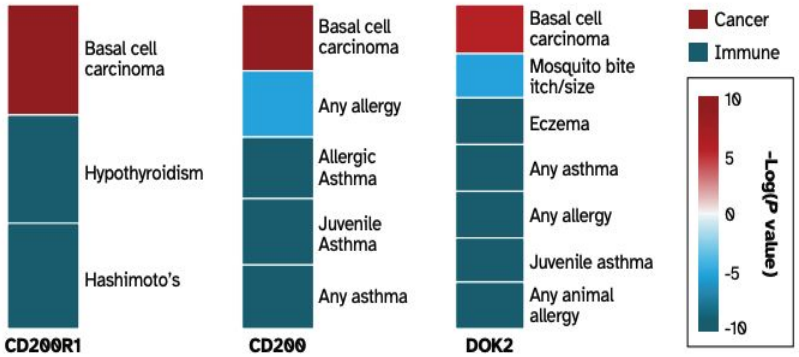
23ME-00610-mediated reversal of immune suppression

Binding of 23ME-00610 to CD200R1 frees up downstream signaling, which may rescue immune cells and reactivate anti-tumor immune responses.

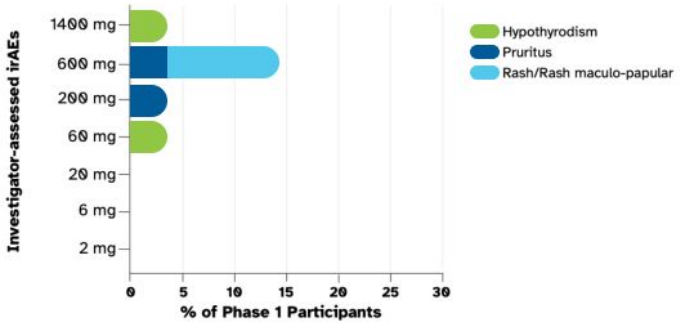
Abbreviations: Akt: protein kinase B; CD: cluster of differentiation; DOK: docking protein; ERK: extracellular signal-regulated kinase; IFN: interferon; IL: interleukin; NK: natural killer; RasGAP: Ras-specific GTPase-activating proteins; TME: tumor microenvironment

’610: geno-phenotypic data unveils novel immune processes that bear out from *in silico* to the clinic

CD200/R1 is a dominant immune checkpoint



Genetic data tracks AE profile observed in clinic (Ph 1/2a) with anti-CD200R1

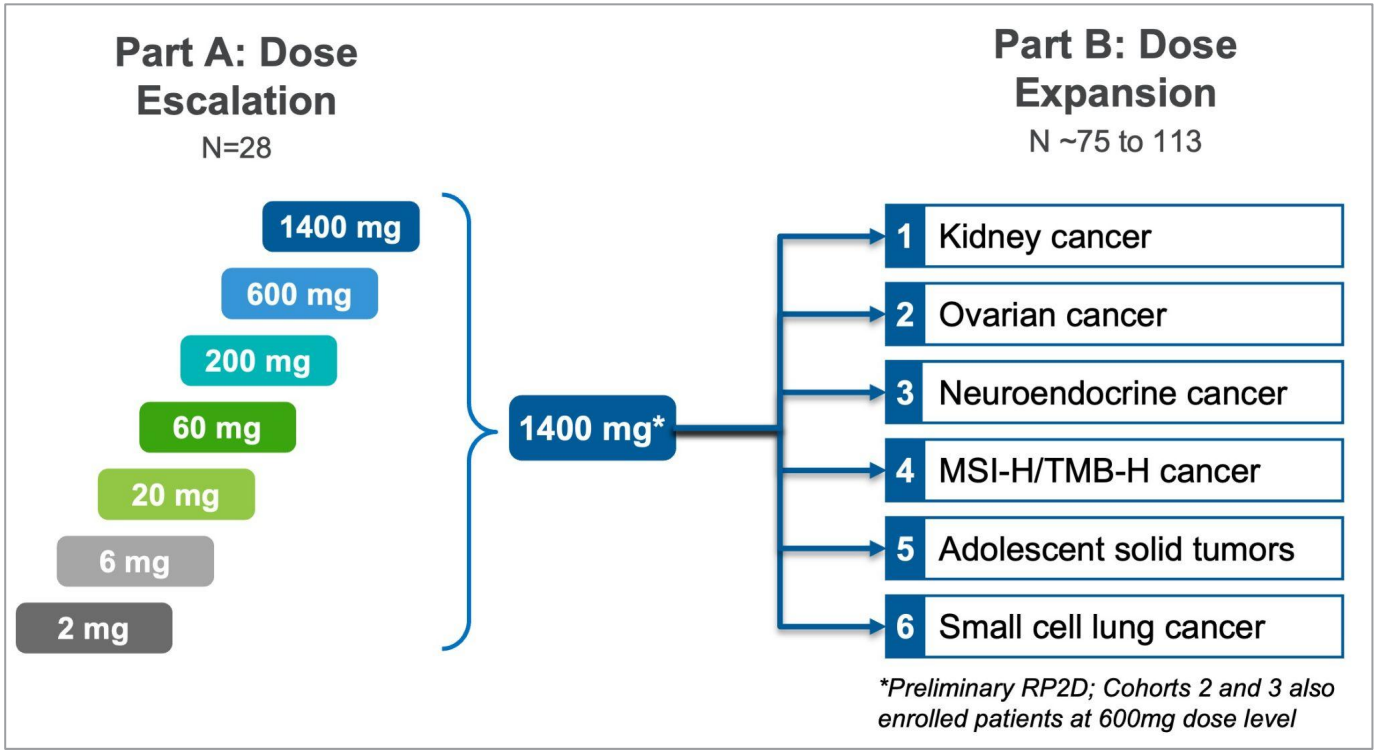


Investigator-assessed immune-related adverse events seen in >5% of patients in cohort

Disease-modifying potential across a broad spectrum of “cold” neoplasms (e.g., neuroendocrine, ovarian)

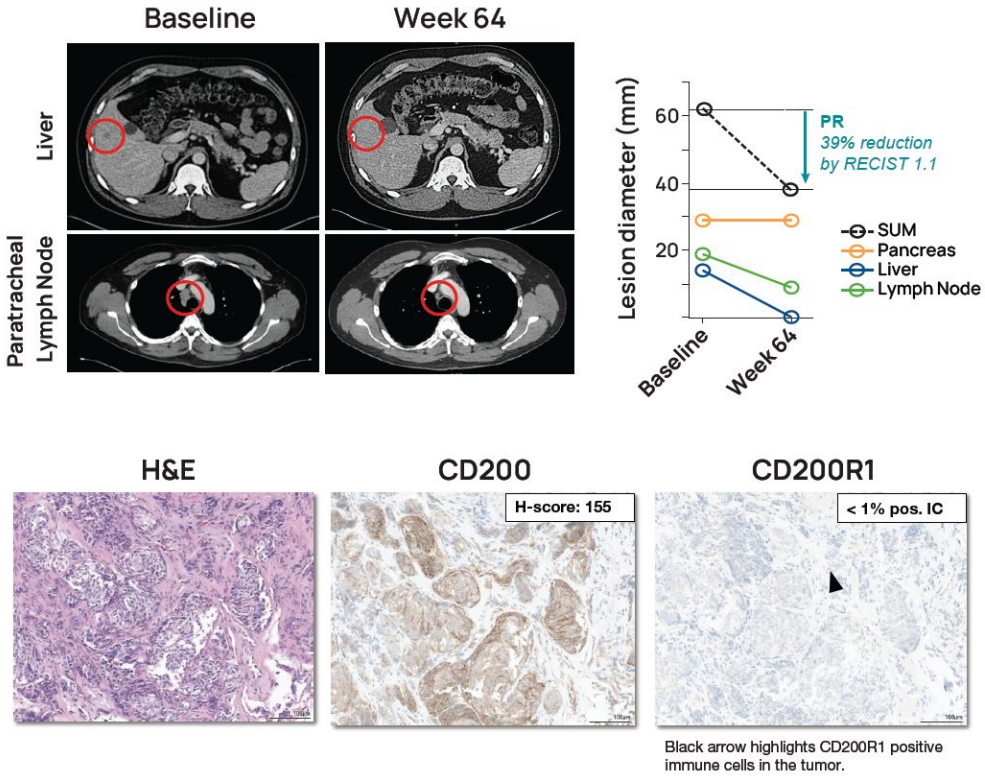
Rasco, D, et al., 2023, SITC Annual Meeting #619

’610 ph1/2a clinical trial design



Further study details , including I / E criteria, at clinicaltrials.gov

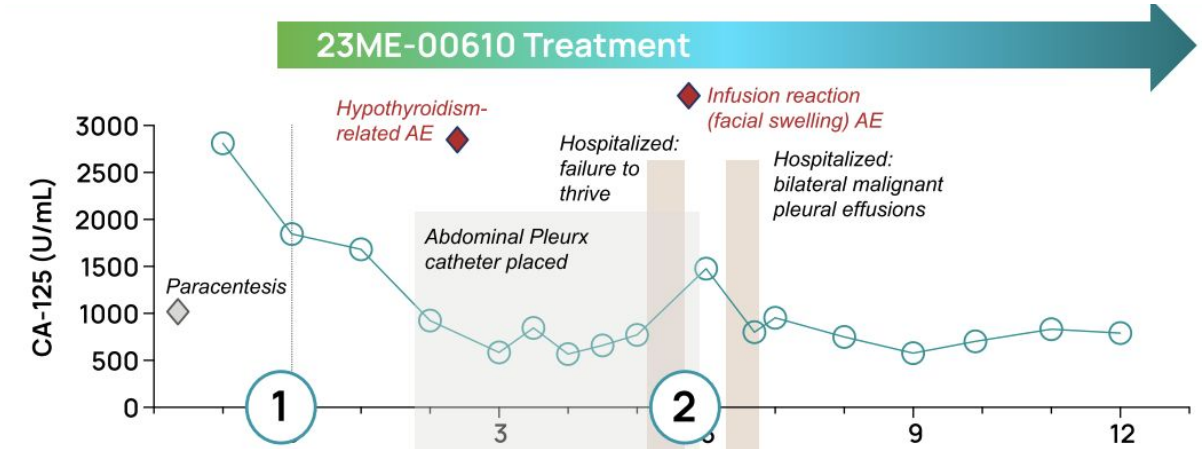
’610 preliminary clinical activity: NET patient vignette

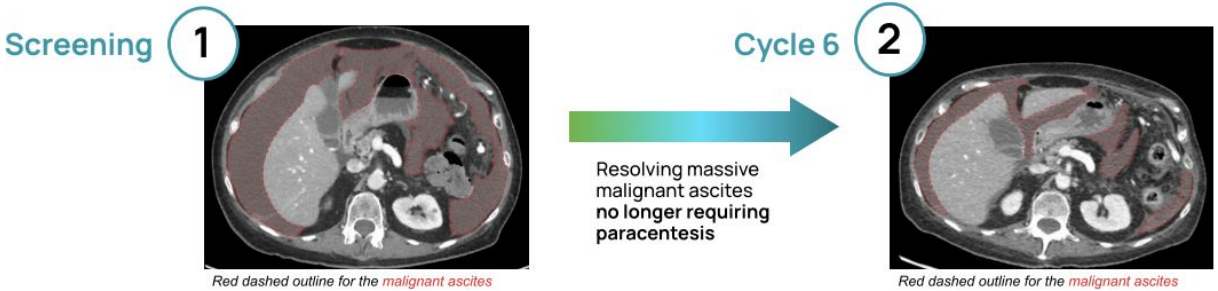


Confirmed PR in CD200-high pancreatic well-differentiated neuroendocrine tumor (pNET);

21 months on treatment

’610 preliminary clinical activity: ovarian patient vignette

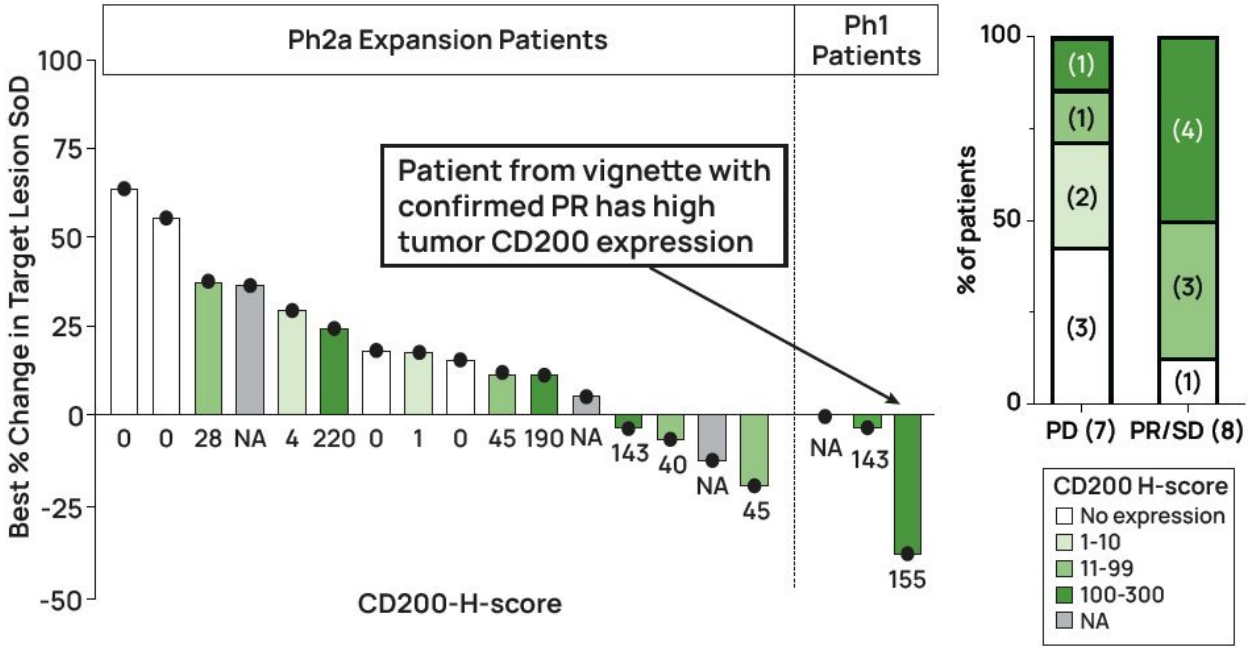




Clinical benefit in mesonephric adenocarcinoma:

- Decreasing CA-125
- Substantial decrease of malignant ascites
- Measurable tumor reduction
- Durable treatment duration (> 12 cycles)

Tumor CD200 emerging as putative biomarker for ’610 clinical activity



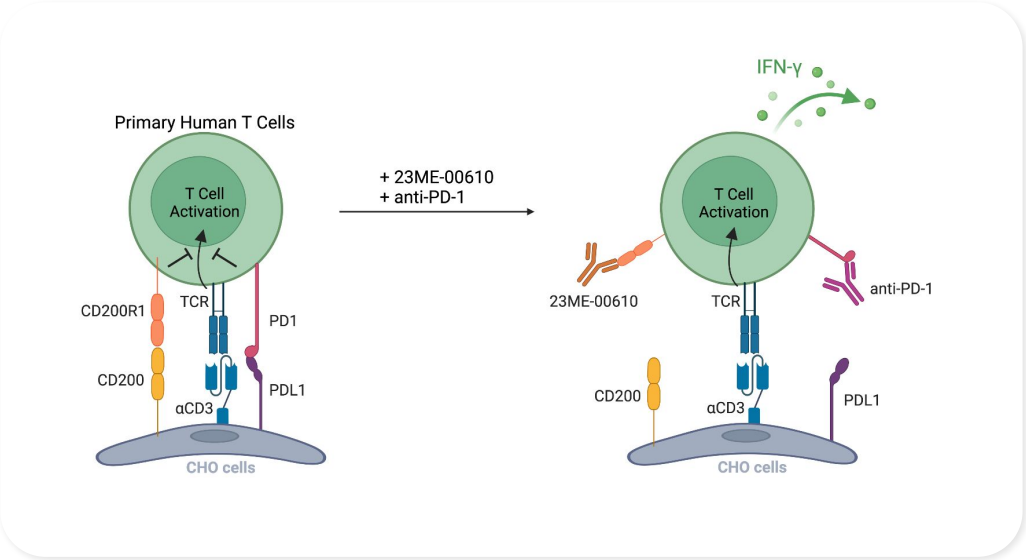
Moderate-to-high tumor CD200 expression associated with higher probability of clinical benefit

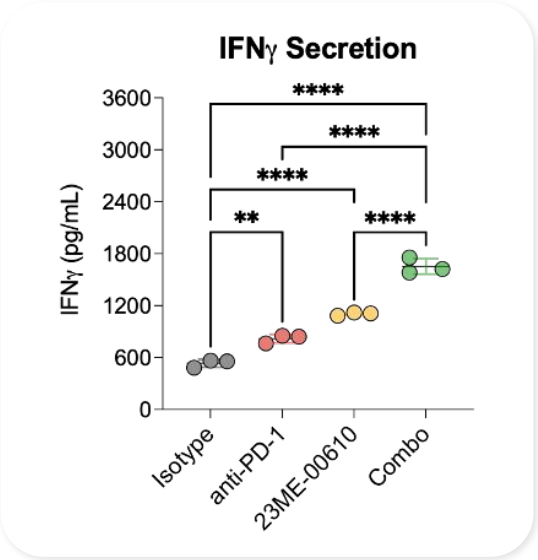
(PR or durable SD)

ASCO 2024

SoD, sum of target lesions; NA, not available; () = number of patients. 4 patients without archival tissue for IHC (“NA”) were not included in the summary statistics (ie, right panel)

’610 has combination potential with a-PD-1





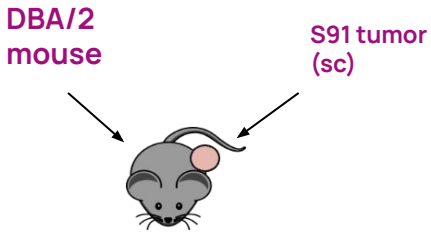
AACR 2024

- 23ME-00610 differentially enhanced IFNγ secretion from cancer patient PBMCs relative to anti-PD-1
- 23ME-00610 enhanced both T and NK cell anti-tumor activity

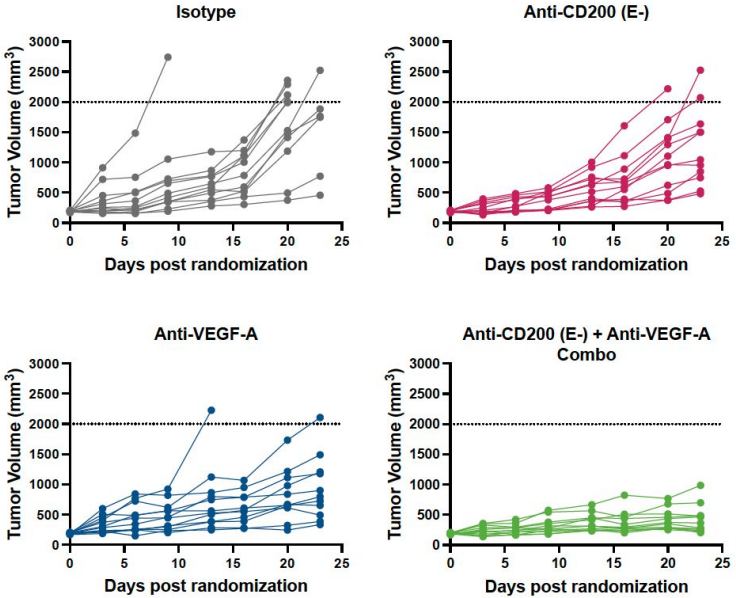
2 ug/mL per antibody. Representative data from one of four donors tested. Statistics: Ordinary one-way ANOVA with Tukey's multiple comparisons test, **p<0.01, ****p<0.0001

’610 has combination potential with a-VEGF

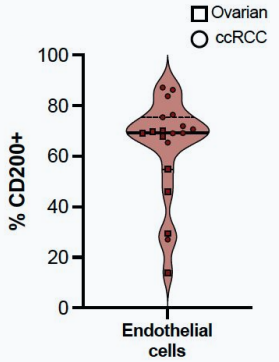
Combo better than single agents for tumor growth inhibition ($p < 0.001$)



N=12/gp. 2qw dosing from 200 mm^3



AACR 2024



CD200 is expressed on endothelial cells, incl. within human tumors

‘610 summary

- Single agent activity seen in Phase 1/2a, with **durable efficacy at highly tolerable doses with prolonged treatment durations**
    - ASCO 2024: Confirmed PR in PNET; tumor reduction and clinical benefit data in OC
- Tumor CD200 emerging as **potential efficacy biomarker**
- PK/PD, safety profile and preclinical data support **combination potential with anti-PD-1, anti-VEGF**

23ME-01473

Genetically validated NK Cell Activator (Anti-ULBP6)

Antibody for Solid Tumors

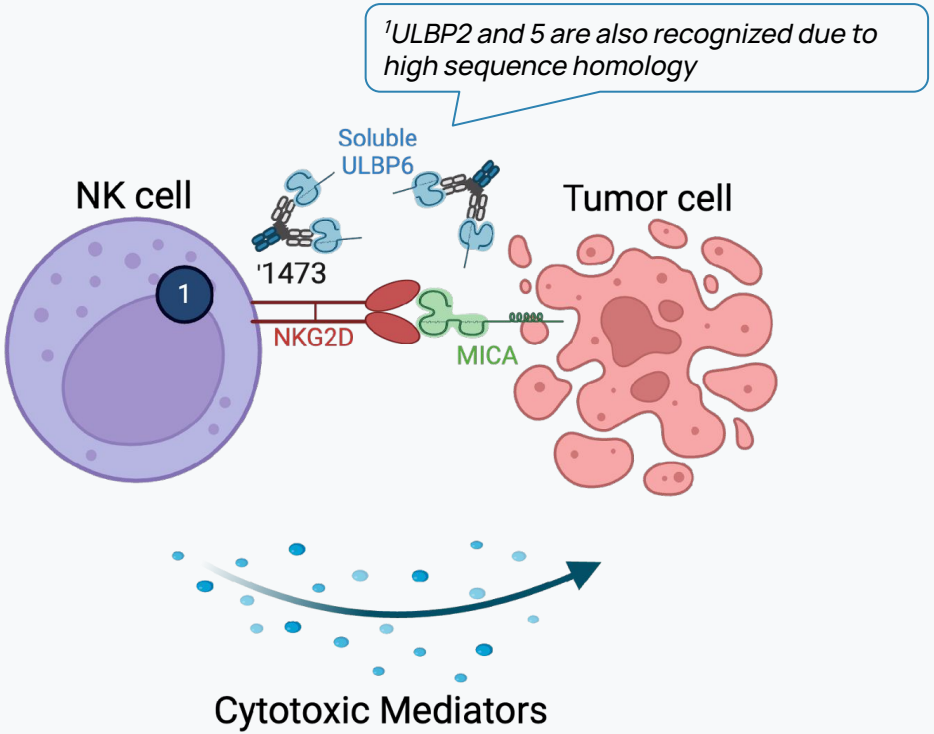
Phase 1 Ongoing

Targeting ULBP6: genetics-first approach with potential to address I/O resistance

23ME-01473, anti-ULBP6[1] humanized monoclonal antibody has dual synergistic MoAs to **fully unleash NK cell activity**

MoA 1: Block soluble ULBP6 to reinvigorate NKG2D axis

MoA 2: Block membrane ULBP6 + Fc-enhanced effector function to maximize ADCC

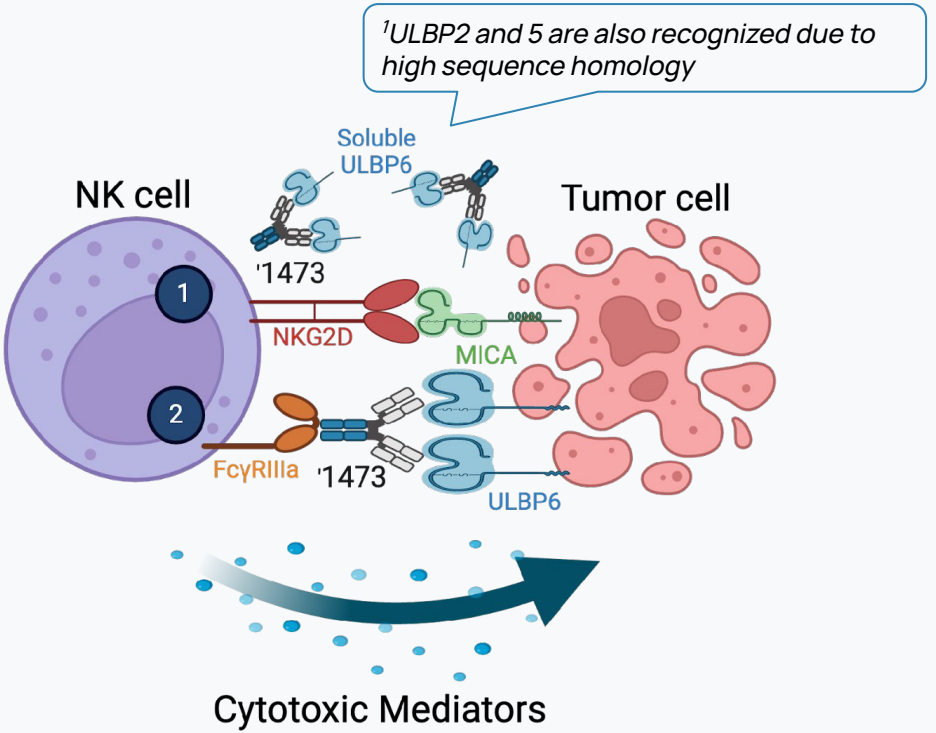


Targeting ULBP6: genetics-first approach with potential to address I/O resistance

23ME-01473, anti-ULBP6[1] humanized monoclonal antibody has dual synergistic MoAs to **fully unleash NK cell activity**

MoA 1: Block soluble ULBP6 to reinvigorate NKG2D axis

MoA 2: Block membrane ULBP6 + Fc-enhanced effector function to maximize ADCC



1473's dual MoA overcomes limitations of other NK-modulating approaches

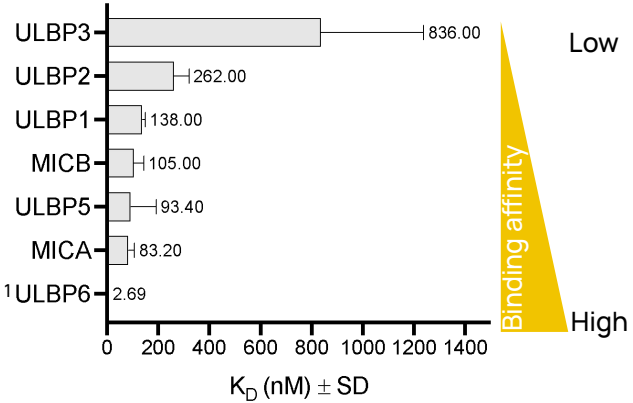
NK Modulator Success Criteria	Engineered NK-cells (e.g., CAR-NK, Allogenic-NK)	Cell-harnessing Tx. (e.g., ICIs, mAbs)	Modulating TME (e.g., cytokines: IL-2, IL-15)	Dragonfly platforms (TriNKETs & cytokines)	23ME-01473
Achieve effective therapeutic index					✔ Highly targeted, IO-like safety potential
Increase targeting ability of NK cells					✔ High binding affinity for ULBP6
Promote sufficient NK cell recruitment					✔ Removing shed ULBP6 *(MOA1)* → increased NK cell availability/persistence
Reactivate suppressed NK cells					✔ Engineered FcγR *(MoA 2)*
Convenient dosing					✔ May be dosed Q3W

Note: * Anti-drug antibodies which may result in a loss of efficacy

Source: Expert interviews; St-Pierre et al., *Cancers* (2021); Zhang et al., *Front Immunol.* (2023); Demaria et al., *EJI* (2021); Yu, *Cancers* (2023); [1] Moscarelli J et. al. *Transplant Cell Ther.* (2022) ; [2] Tarannum, M., Romee, R., *Stem Cell Res Ther* (2021); [3] Khan M, *Front Immunol.* (2020); [4] Tinker, Anna V et al. *AACR* (2019); [5] Chu, J., et. al., *J Transl Med* (2022); [6] Gutierrez et al., *Cell* (2023)

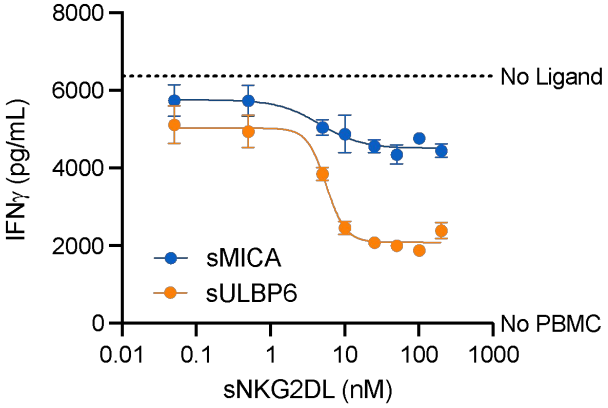
As highest-affinity NKG2D ligand, ULBP6 is a critical regulator of anti-tumor immunity

ULBP6 has the highest binding affinity for NKG2D

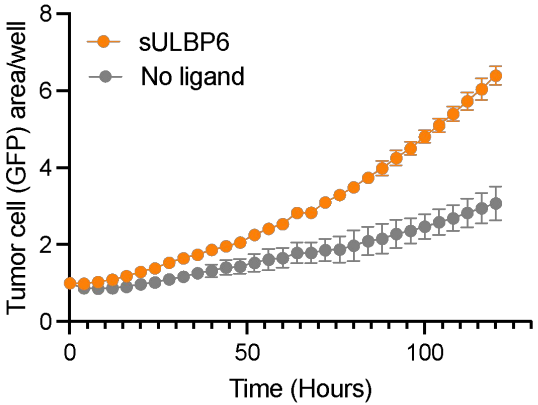


*No binding with ULBP4

Soluble ULBP6 is highly immunosuppressive



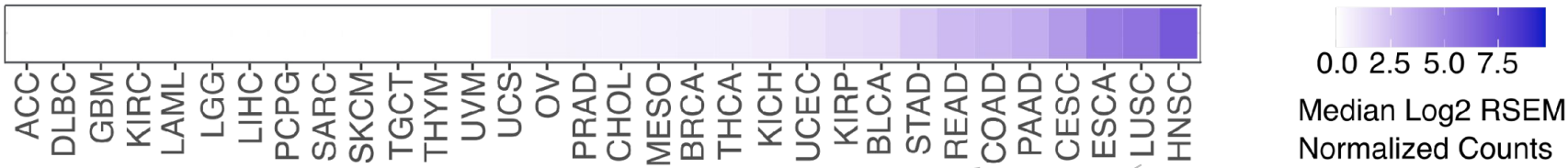
Soluble ULBP6 promotes tumor cell growth



[1]ULBP6 isoform 1

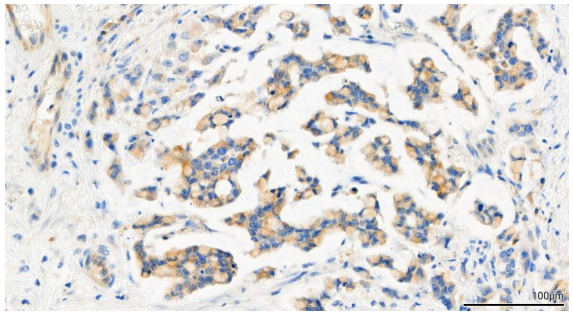
ULBP6 is highly expressed in squamous cell carcinomas & subset of adenocarcinomas

ULBP6 (*RAET1L*) mRNA expression in TCGA

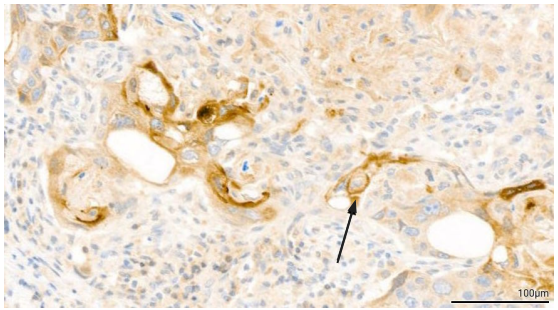


ULBP6 IHC[1]

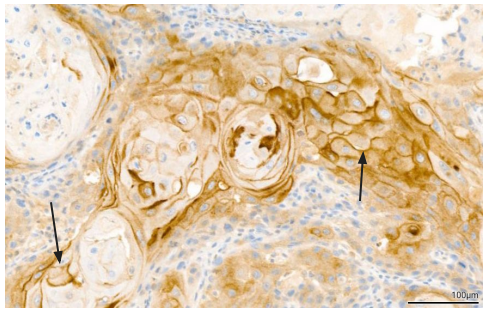
Colorectal



Lung squamous



Head & neck squamous



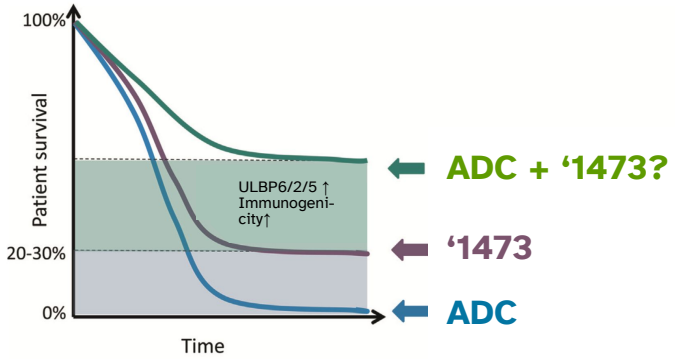
[1]ULBP2 and 5 are also recognized due to high sequence homology and highly expressed in squamous cell carcinomas
Arrows = membranous staining

‘1473 has combination potential with multiple modalities, including ADCs

Various MOA-based areas of potential clinical synergy

- NK/T potentiation: ‘1473 expected to act on NKG2D+ NK and (antigen experienced) T cells, additive/synergistic potential if combined with:
  - ADC/chemo/radiation: Agents increasing tumor immunogenicity [1]
  - T cell stimulators: (e.g. a-PD-(L)1)
  - Cytokines: prolonging NK persistence
- Target upregulation:
  - ADC/chemo/radiation: NKG2D ligands are upregulated by cellular stress including exposure to cytotoxic agents [2]

Potential impact of ADC and 23ME-01473 combination



Modified from: Gerber et al 2016 Biochem Pharma

1: Zhang et al 2022 Front Oncol; Heinhuis 2019 Ann Oncol
2: Jones et al 2022 Cancers

‘1473 summary

- Purposefully designed dual-MoA mAb against ULBP6, tailored to activate NK and T cells addressing major needs unmet with other IO therapies
- Potential dose expansion cohorts in:
  - Squamous cell tumors (head & neck, lung)
  - Additional ULBP6-high tumors (CRC, TNBC)
  - Phase 1b combinations with other checkpoint inhibitors, synergistic mechanisms
- Phase 1 dose escalation ongoing
  - Tissue and genetic biomarker characterization of treated patients

Immunology Discovery

Immunology / Inflammation (I&I) remains a biotech frontier

Immune system is highly complex

Highly polygenic diseases with complex, diverse tissue dysfunction and clinical phenotypes across individuals

Many conditions are severe, chronic, with morbidity and high unmet need

Few solid therapeutic hypotheses

Mostly coarse, subjective clinical labels with no actionable causal nodes

Poor disease subtyping / precision approach relative to other TAs (e.g., oncology)

Poor clinical translation

Non-predictive target-drug-patient choices → poor clinical outcomes after hundreds of $MM invested

23andMe: bringing unprecedented power to I&I discovery

Ultra-powered for precision

Genetics-based deconvolution of I&I complexity, starting with respiratory disease

Powered by world's largest database of human genomic and phenotypic health information

- 15M genotyped individuals
- >4B phenotypic datapoints

High-confidence target-drug-indication decisions

Ab program P032, dual-MOA pipeline-in-a-drug potential (asthma+)

Ab program P023, FIC potential in sarcoidosis

Multiple prioritized targets with pan-modality druggability (incl. small molecule, siRNA)

Translation-focused stack and team

Genetically driven roadmap for translation, potentially >2-3x PoS*

Integrated R&D stack across:

- computational biology
- functional genomics
- antibody engineering
- early clinical development

Pharma veterans with hit-to-clinic success for Amgen, Genentech, GSK

*Minikel et al. *Nature* (2024)

We survey >150 immune disease phenotypes

~700 novel hits in asthma alone

Disease	23andMe GWAS cases	Public GWAS cases	23andMe loci beyond largest public GWAS
Asthma	**1.1M**	**154k**	**697**
COPD	83k	36k	171
Atopic dermatitis	716k	65k	502
Psoriasis	278k	19k	319
Severe acne	535k	34k	735
Urticaria	461k	41k	386
Hidradenitis	31k	1.6k	114
IBD	117k	60k	54

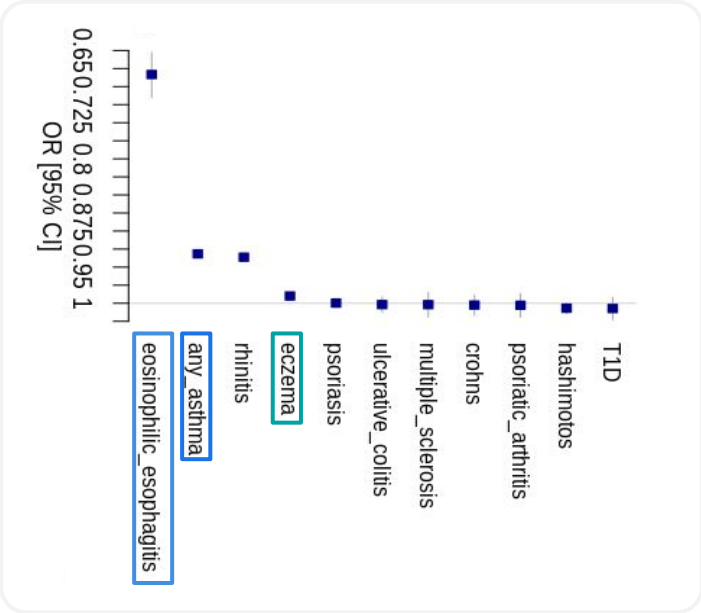
[1] 23andMe multi-ancestry meta-analysis GWAS as of October 2023

Respiratory | Skin | Bowel

We have a uniquely robust dataset credentialing our target selection

Case study: TSLP and indication (mis)pairing

TSLP PheWAS*

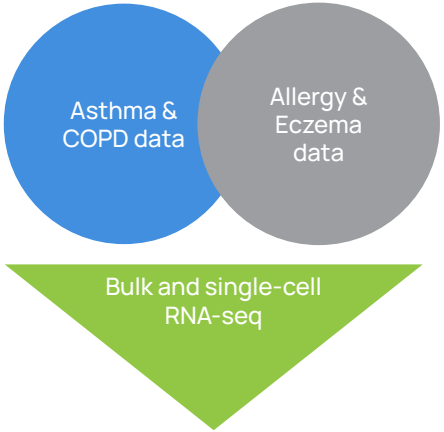


- 23andMe runs GWAS in >1,000 phenotypes, which increases discrimination power for target-indication pairing
- We observe a clear genetic signal linking TSLP to **asthma**
- Amgen clinical trials of anti-TSLP mAb as **eczema** target failed. **We do not observe a statistically significant genetic signal linking TSLP to eczema**
- We observe a strong genetic signal linking TSLP to **eosinophilic esophagitis** → potential indication expansion in a rare disease

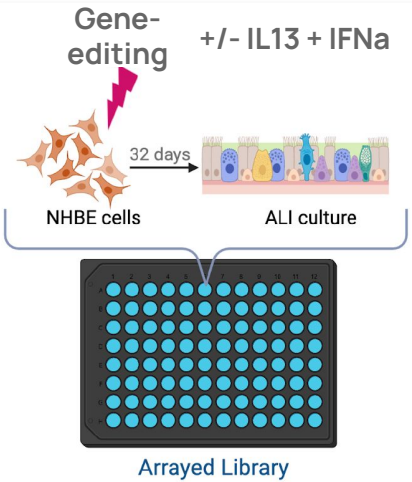
*PheWAS (Phenome-Wide Association Study) captures pleiotropic effects of genetic variants and points to possible unwanted toxicities or potential indication expansions

We use human *and* phenotype-relevant cellular data to validate genetic insights

23andMe genetics

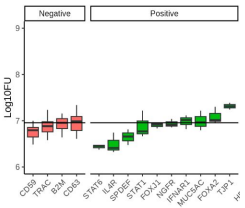


***In vitro* functional genomics**

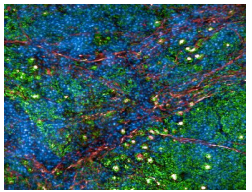


Disease-relevant readouts

Barrier function

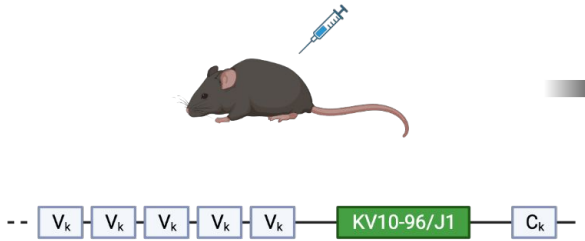


Cell composition & mucin production

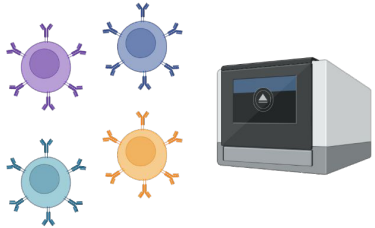


Several high-confidence hits identified from 200+ tested genes, several with effect sizes similar to IL4R deletion (target of dupilumab)

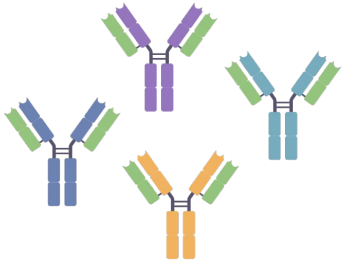
Our In-House Expertise in Antibody and Protein Engineering Enables Differentiated Therapeutic Generation



Our proprietary common light chain (cLC) mouse*



Antibody^{+} B cell cloning and NextGen sequencing

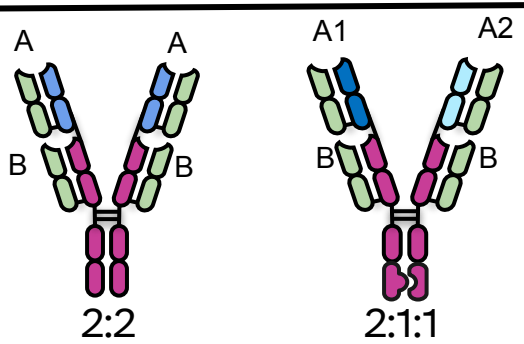


High throughput cLC antibody production, humanization and triage

Easy to format bi/multi-specifics that enable desired activity

- **Superior developability for discovery and development in comparison to bispecifics without common LC**
- P032 current options: 2:2 and 2:1:1 formats with biparatopic anti-Target 1 arms (A1, A2) and anti-Target 2 arm (B)

Deep experience in protein engineering, biochemistry, structural biology (ex-Genentech leadership)



*Rong et al. *Antibodies (2024)*

P032: a novel program with pipeline-in-a-product potential

Asset Overview

- Genetic support for pathway components in multiple immune diseases
- P032 will be an effectorless bi-specific IgG1 mAb that blocks the activity of three cytokines
- Strong translational derisking (internal & external biology support, including in the clinic)

Commercial Rationale

- **Indication potential**: multiple-immune related diseases validated in target pathways
- Substantial unmet medical need remains in large, non-Th2 subtypes within asthma, COPD
- Biologics targeting these single cytokines leave room for considerable improvement

Scientific Rationale

- P032 poised to block three key cytokines from signaling and contributing to disease
- Our unique cLC mouse enables the generation of multiple bispecific antibody formats with downstream manufacturability advantages

P023: a unique, novel-MoA antibody for granulomatous disease

Asset Overview

- P023 target: A compelling and unique genetic association with sarcoidosis
- P023: a monovalent IgG1 effectorless mAb binds target to block ligand activation
- Lead molecule selected; cyno PK studies completed, PD studies ongoing

Commercial Rationale

- **Indication potential:** Sarcoidosis, Crohn's, Multiple Sclerosis, other granulomatous diseases
- Substantial unmet medical need; biologics (off-label) do not address underlying disease

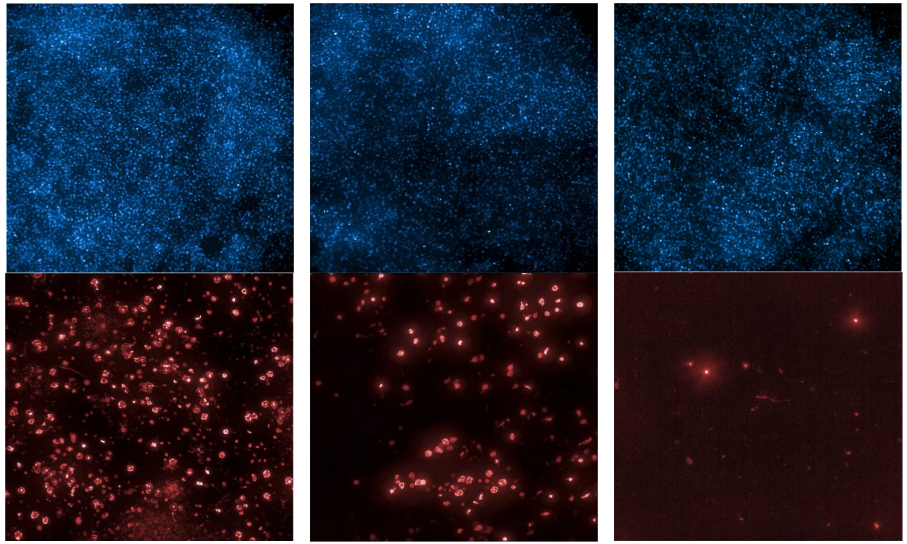
Scientific Rationale

- P023 target is a genetically validated target in sarcoidosis and other granulomatosis indications
- Numerous genetic variants with reasonable effect size and allele frequency
- P023 target neutralization expected to both prevent and resolve granuloma formation to prevent organ damage and meaningfully improve QoL

Gene X: example of a high-confidence, novel respiratory target

Gene X

Alters mucus-producing cell differentiation

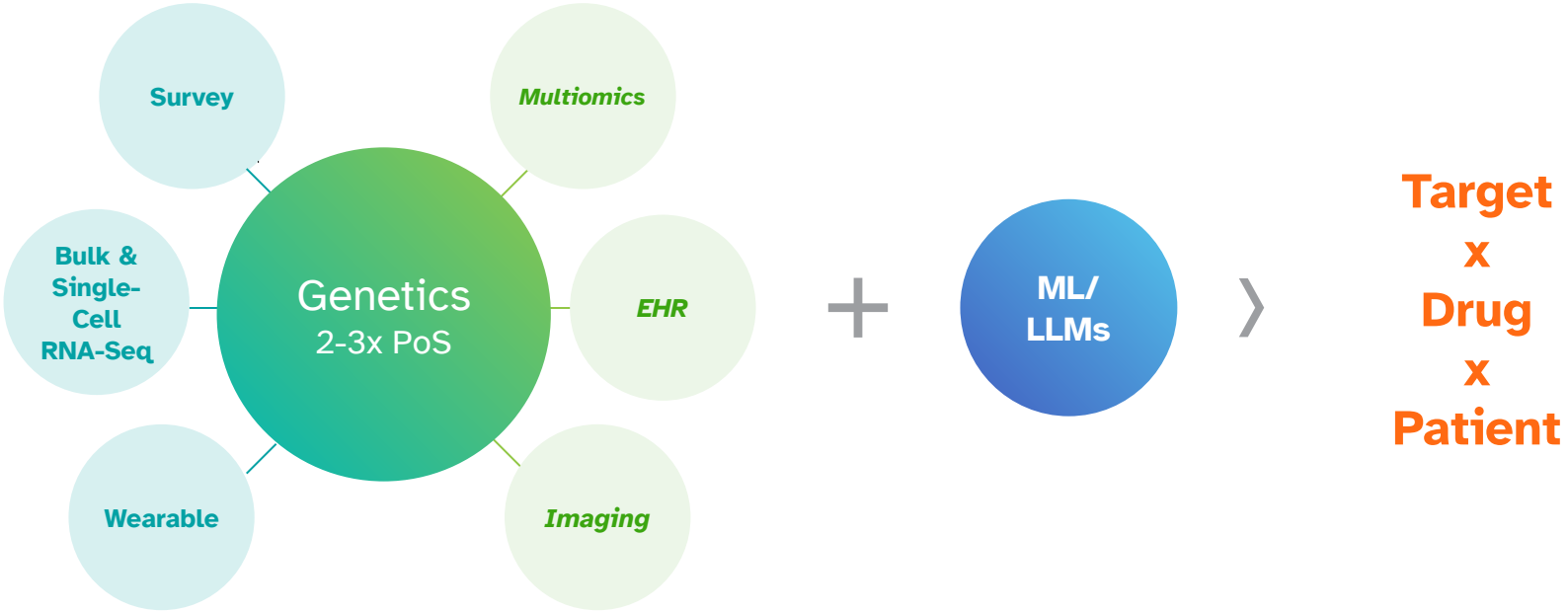


Gene X is a potential siRNA target; other pathway members are mAb-tractable

Coming up: broadening target discovery to other I&I cell types

	Cell type	Disease opportunities	Data available
	Bronchial epithelia	Respiratory: asthma, COPD	210 genes
	Fibroblasts	Respiratory: asthma, COPD, IPF	Emerging
	Keratinocytes	Skin: eczema, acne, hs	Emerging

Future vision: multi-modal data + custom ML → precision I&I



For More Detailed Information on 23andMe Therapeutics:

www.Therapeutics.23andMe.com

and visit our Investors page to view our full Therapeutics investor deck

https://investors.23andme.com/news-events/events-presentations

4

Financials

Solving for fiscally responsible future growth

1

Investing in future growth potential

- Subscription Services
- New reports and insights
- Research partnerships
- Therapeutics

2

Employing a conservative approach to planning

- Prioritizing the minimization of Adjusted EBITDA deficit rather than maximizing top-line growth in our Consumer business (PGS and telehealth).

3

Investing in future growth potential

- Cash of $216 million[1] supports 23andMe's plans for targeted investment in high ROI growth initiatives.

[1] As of March 31, 2024.

Revenue composition

	Three Months Ended March 31,				Year Ended March 31,	
	FY2024		FY2023		FY2024	
(in $M, except percentages)	**Amount**	**Percentage of Revenue**	**Amount**	**Percentage of Revenue**	**Amount**	**Percentage of Revenue**
Consumer Services	$63	*99%*	$81	*88%*	$202	*92%*
Research Services	1	*1%*	11	*12%*	17	*8%*
Therapeutics	-	-		-	-	-
Total Revenue	**$64**	***100%***	**$92**	***100%***	**$220**	***100%***

Consumer services revenue seasonality by fiscal quarter

	Q1	Q2	Q3	Q4	Full Year
FY 2020	24%	24%	21%	31%	100%
FY 2021	18%	21%	22%	39%	100%
FY 2022	22%	20%	21%	38%	100%
FY 2023	22%	25%	22%	31%	100%
FY 2024	28%	23%	20%	29%	100%

Note: Fiscal year ends March 31.

Upcoming value drivers and catalysts

Consumer

New product development, improved subscription value delivery, upgrades and cross-selling health services

Continued customer LTV and margin improvement

Progress toward adjusted EBITDA breakeven

Research

Research collaborations

New GWAS

Imputation and AI-driven innovations

Therapeutics

Initial ‘610 Phase 2A data

‘1473 Phase 1 data

Potential collaborations