23andMe Therapeutics

January 2024

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, database growth, future collaborations, future development of therapeutic programs or products 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.

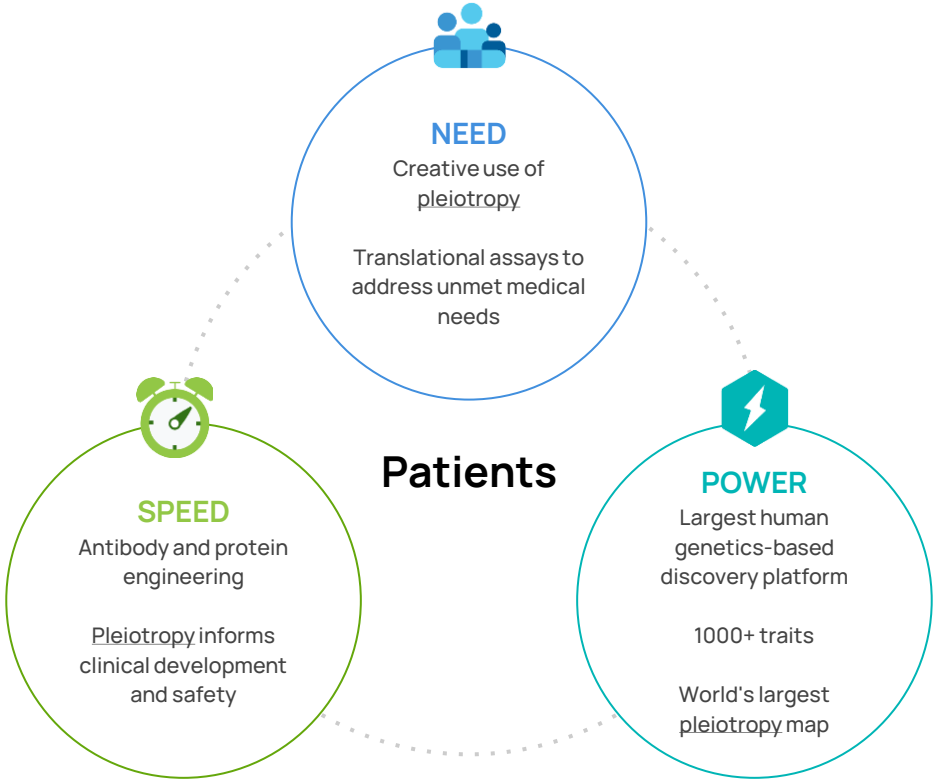
23andMe Therapeutics: Genetics Reimagining R&D

Our Value Proposition

GENETICS

Our credo: Every Day Matters	• **Current focus: Oncology Development, Immunology Discovery** • Fast timelines and early kill decisions from discovery through clinical development to approval
Higher probability of success in the clinic	• Indication selection informed by lifetime genetic risk based on world's largest human genotypic & phenotypic data platform • Genetics (e.g. GWAS, PRS) and biomarkers to optimize target-indication-patient clusters
Forward-thinking expert team	• Experienced, innovative genetics researchers and clinical development team with track record for innovative approvals • Genetics and clinical development scientists to identify higher success programs to bring into the clinic

Using Human Genetics to Create Meaningful Therapeutics for Diseases with High Unmet Need in Oncology and Immunology

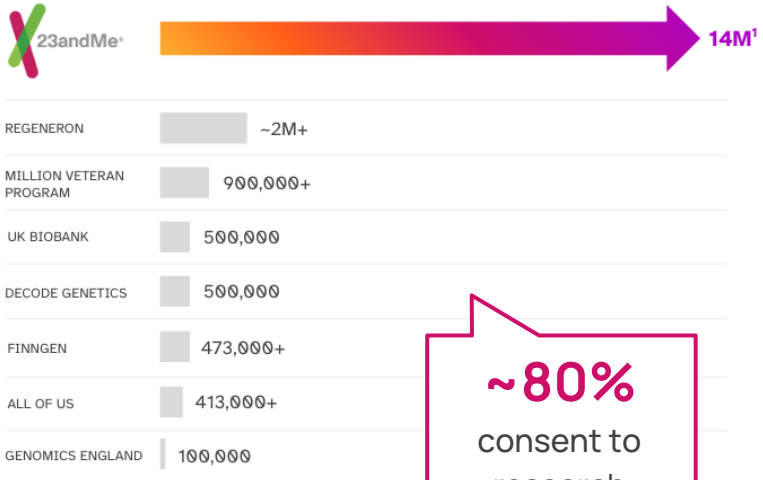


The Power of Our Approach

Leaders in Data

23andMe Has the Largest Recontactable Genetic Database for Target Discovery in the World

Largest, most diverse recontactable database of genotyped + phenotyped individuals



~80% consent to research

[1] As of September 30, 2023.

Pharma partnerships leverage the database for research and recruitment

- **Target discovery**
- **Target validation**
- Patient selection
- Clinical trial recruitment

Drugs with human genetic support are **2x - 3x** more likely to succeed*

GSK · janssen · Genentech · Pfizer

*Publications supporting human genetic evidence for approved drug indications
Nelson et al., 2015 (Nature Genetics); King et al., 2019 (PLOS Genetics)

POWER: Combining Our "I" and "O" Phenotypes Gives Us Broad Statistical Power to Drive Unique Immunological Insights for Oncology Development

IO phenotypes of interest (examples)

"O" Oncology phenotypes	Cases
BCC	410,104
Bladder	15,663
Brain	4,586
Breast	118,632
Colorectal	25,398
Endometrial	17,912
Esophageal	1,134
Head and Neck	8,596
Kidney'	14,934
Leukemia	13,763
Liver	3,077
Lung	12,367
Melanoma	125,364
Myeloma	7,127
NH lymphoma	17,643
Ovarian	13,044
Pancreatic	2,910
Prostate	71,616
SCC	218,805
Stomach	3,508
Thyroid	27,259

Total: 1,133,442

"I" Immune phenotypes	Cases
Vitiligo	60,701
Alopecia areata	56,233
Hashimoto's	186,069
IBD	116,788
Atopic dermatitis	716,447
Poison oak rash	783,604
Allergy	2,053,011
Food allergy	213,185
Asthma	1,128,292
Tonsillectomy	270,499
Toenail Fungus	276,405
Psoriasis	277,525
Hidradenitis suppurativa	31,008
Lupus	58,414

Biological processes of interest captured in "I" phenotypes, not targeted in the clinic yet

- Autoimmunity
- Immune Polarization
- Atopy
- Inflammation
- Chronic Infection
- Tissue Repair

POWER: 23andMe Database Contains >150 Immune Disease Phenotypes With Up To 100s of Novel Genetic Insights Per Disease for Immunology Discovery

Drugs with human genetic support are

2x-3x

more likely to succeed[1]

Disease	23andMe GWAS cases	Public GWAS cases	23andMe hits beyond largest public GWAS
Asthma	1.1M	65k	716
COPD	83k	36k	171
Atopic dermatitis	716k	84k	399
Psoriasis	278k	19k	319
Severe acne	535k	34k	735
Urticaria	461k	41k	386
Hidradenitis	31k	1.6k	148
Rosacea	352k	73k	421
Alopecia areata	56k	3k	67
Vitiligo	61k	4.7k	75
IBD	117k	60k	54

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

Skin | Respiratory | Bowel

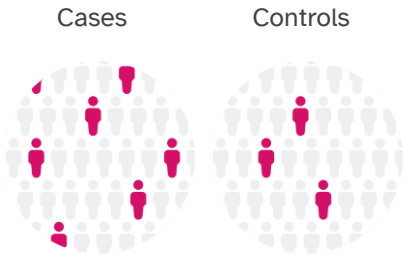
Nelson et al., 2015 (Nature Genetics); King et al., 2019 (PLOS Genetics)

GWAS: The Initial Foundation for Genome Analysis

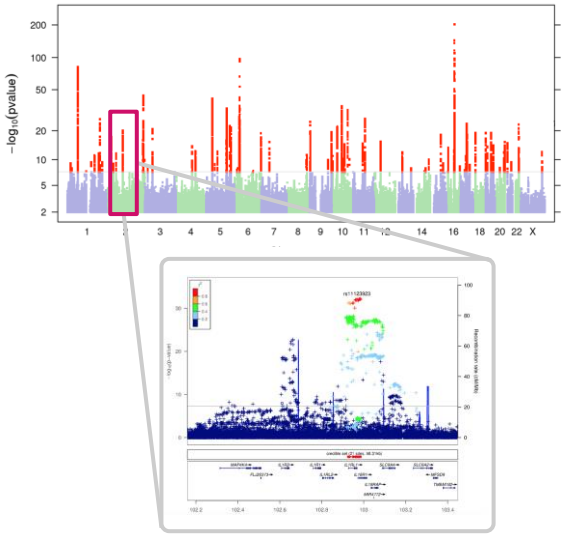
Single Nucleotide Polymorphism (SNP)

GGCCAGCTGGACGAGG

GGCCAGCTGGATGAGG



- GWAS = Genome-Wide Association Study
- SNPs associated with disease found at different frequencies in case vs controls
- Extensive know-how required to get from association to therapeutic target



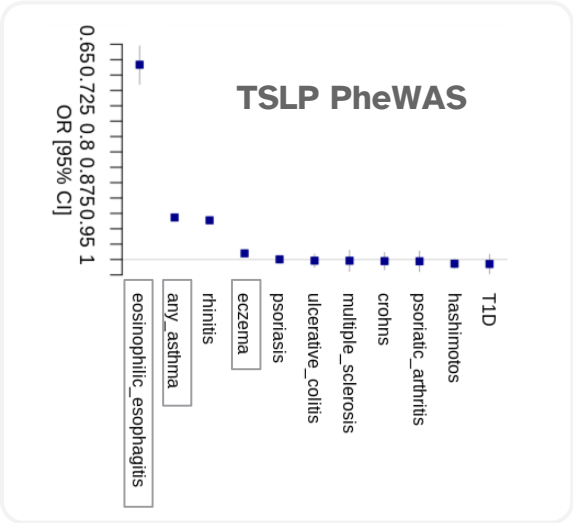
SNPs are tested across the genome and disease associations mapped to specific regions

23andMe®

PheWAS: Breadth of Phenotyping Elucidates Critical Disease Drivers

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



- We observe a clear genetic signal linking TSLP to asthma
- We do not observe signals in phenotypes that would point to safety issues
- 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

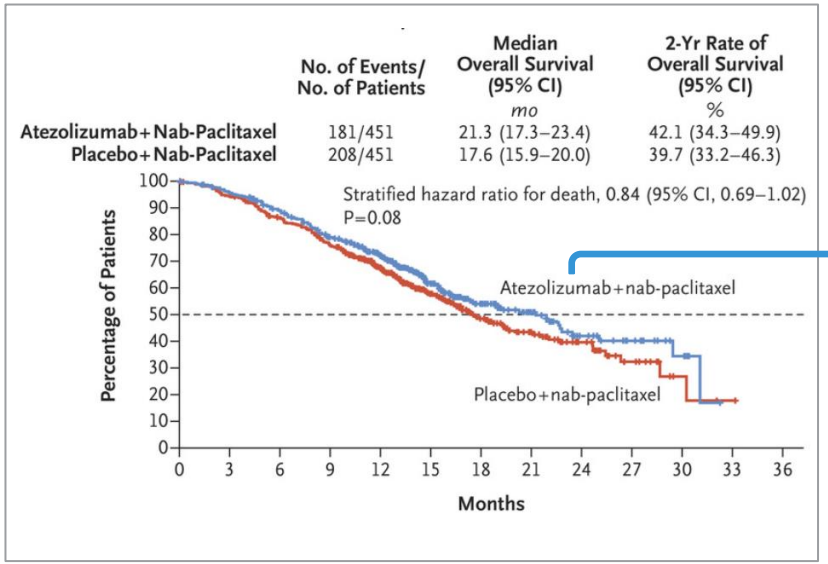
POWER: Immune Genetics Implemented as an IO Clinical Biomarker

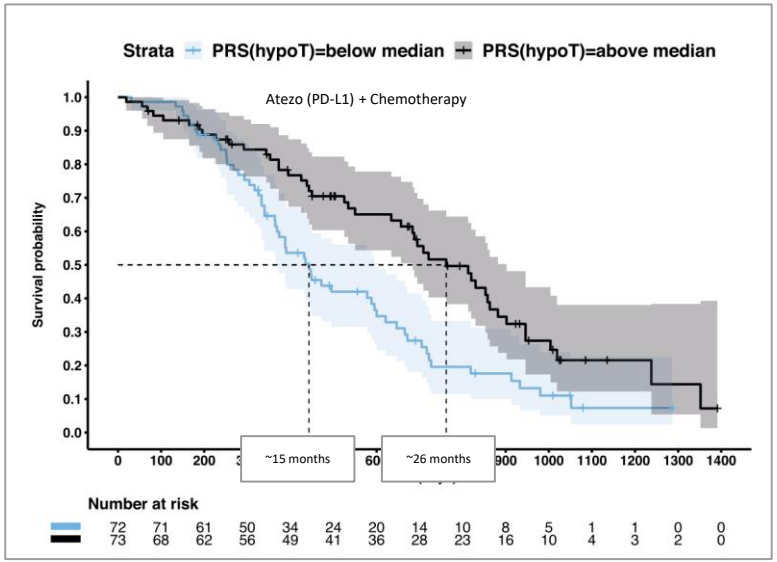
Phase 3 trial failure → Withdrawal of triple-negative breast cancer indication

N~900
HR~0.84

N~150
HR~0.62

Germline genetic score (PRS) for hypothyroidism risk separates survival probability



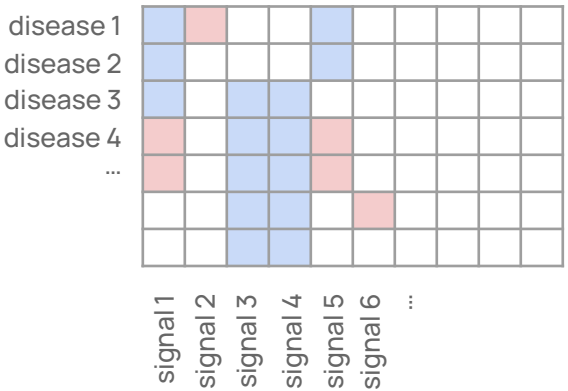


PMID: 30345906;
PMID: 34099659

POWER: Combining Extensive Pleiotropy in the 23andMe Database and Computational Biology for Target Discovery

Genetic insights

GWAS signals / pleiotropy (one variant affecting multiple traits)



Computational Biology ML / AI

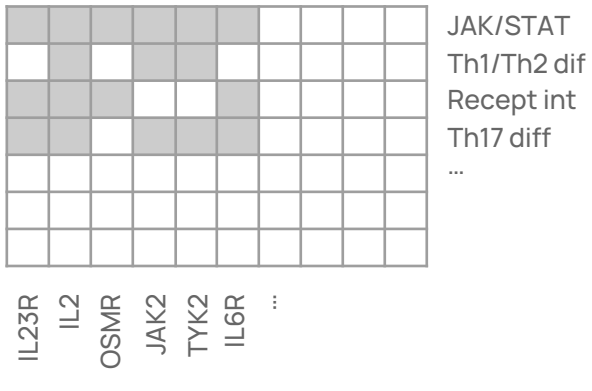
QTL-based and custom ML models for gene mapping and target hypothesis prioritization

Interpretation of GWAS signals making extensive use of pleiotropy and allelic series and to increase reliability of biological conclusions

Analysis of bulk/single cell/differential gene expression

Biological insights

genes, mechanisms, pathways and cell types

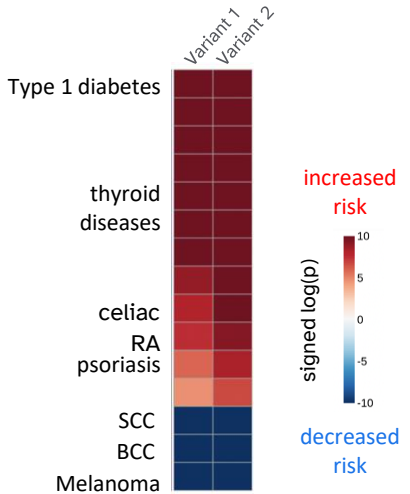


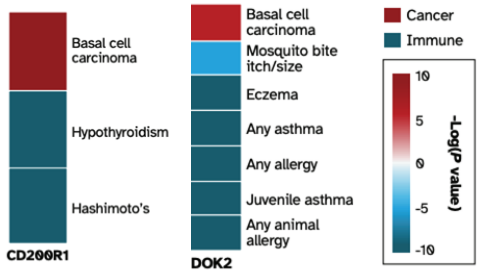
Utilizing the World's Largest Human Pleiotropy Map to Address Unmet Medical Need

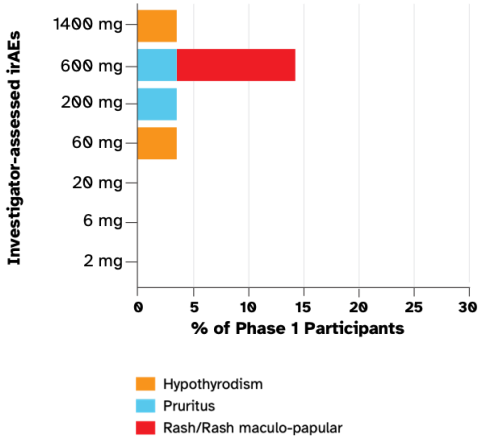
NEED: Our Unique Approach to De-risk Development:

Leveraging Pleiotropy to Characterize Novel Cancer Targets

23andMe "IO Signature" › **23ME-00610 Lead Asset (currently in Ph2a*)** › **Genomic data successfully predicted '610 AE Profile**



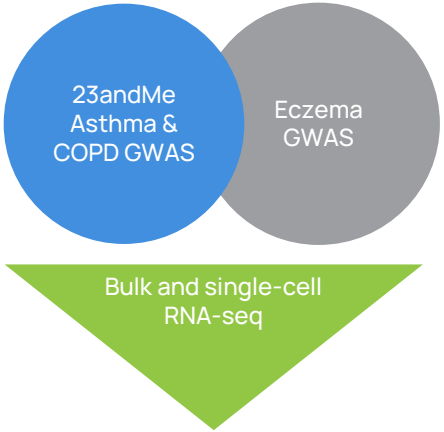




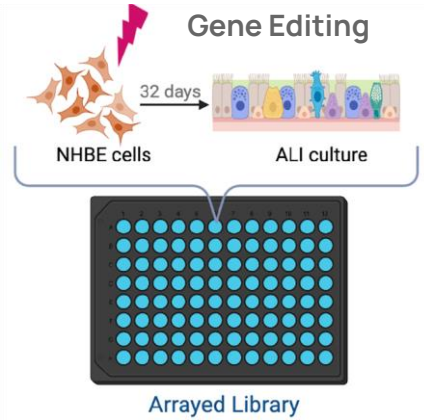
*Currently in Phase 2a portion of Phase 1/2a

NEED: Our FxG Efforts Leverage Pleiotropy to Identify Targets in Defined Areas of Medical Need in Asthma

23andMe genetics

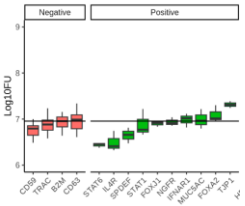


In vitro functional genomics

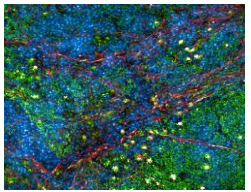


Disease-relevant readouts

Barrier function



Cell composition & mucin production

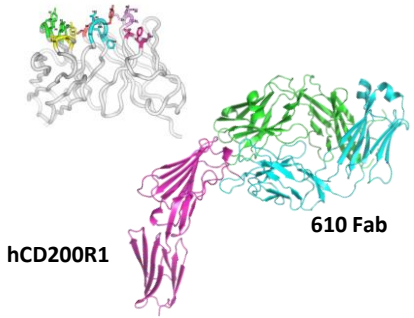


Validated targets with pharmacologically meaningful effects in disease relevant assays

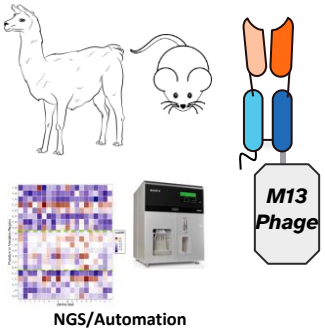
Progression of Therapeutics at Speed

SPEED: Our In-House Expertise in Antibody and Protein Engineering Enables Rapid Therapeutic Generation

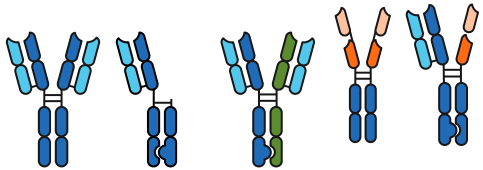
- Experienced Antibody and Protein Engineering group
- Deep experience in protein engineering, biochemistry, structural biology, enabling diverse approaches to antibody discovery, antibody engineering, and automation



Protein engineering and biochemistry



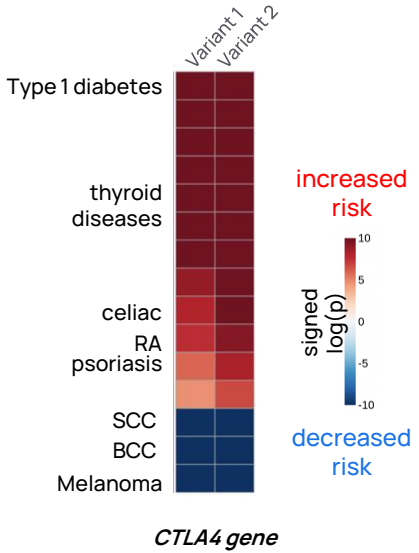
Antibody discovery and optimization

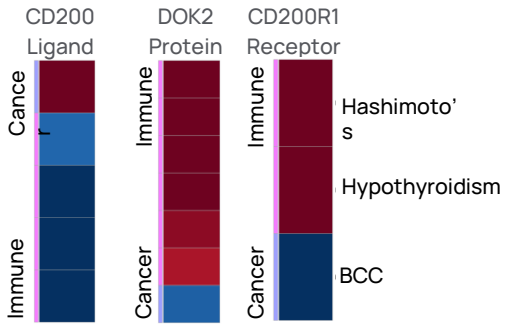


Antibody formats and Fc engineering

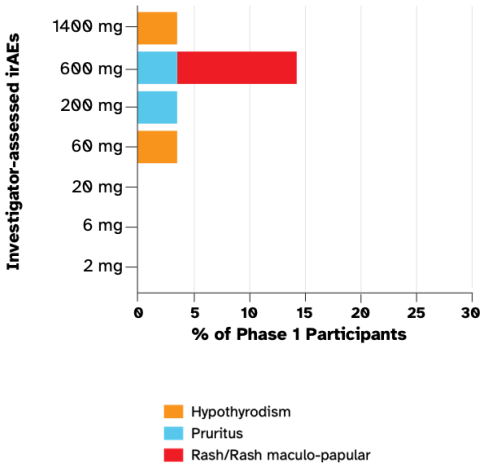
SPEED: Our lead IO program progressed from discovery to the clinic in 5 years

23andMe "IO Signature" › **23ME-00610 Lead Asset (currently in Ph2a of Phase 1/2a trial)** › **Genomic data successfully predicted '610 AE Profile**





- Program initiated: 2016
- Lead molecule generated by 23andMe
- First human dosed: 2021



23andMe Therapeutics: Clinical Development

Experienced Clinical Development Leadership

Jennifer Low, MD, PhD
Head of Development

Genentech LOXO ONCOLOGY

Erivedge (vismodegib)
Vitrakvi (larotrectinib)
Zelboraf (vemurafenib)
Cotellic (cobimetinib)

Maike Schmidt, PhD
Sr Group Head,
Translational Sciences

Genentech FivePrime

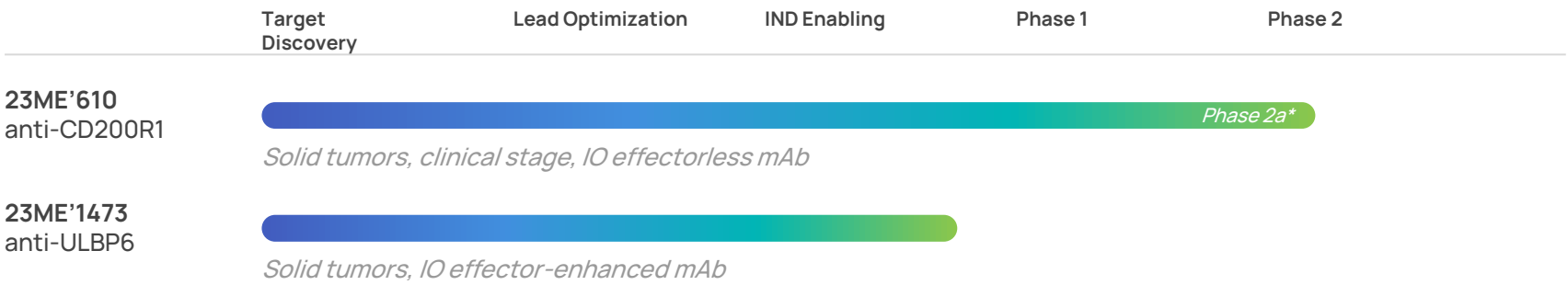
Avastin (bevacizumab)
Tecentriq
(atezolizumab)

Dylan Glatt, PhD
Sr Clinical Pharmacologist,
23ME-00610 PTL

GILEAD

Jyseleca (filgotinib)

23andMe Therapeutics IO Pipeline: First-in-Class Potential



23ME'610/anti-CD200R1

- Targets Innate and Adaptive Immunity
- Potent Ab with great PK/PD
- Phase 1 monotx with on-target AEs
- Ph2a data expected to be presented mid-2024

23ME'1473/anti-ULBP6

- Activator of tumor NK cells
- Effector-enhanced Ab with dual NK-activating MOA

Note: '610 is in Phase 1/2a as of January 2024.

23ME-00610*

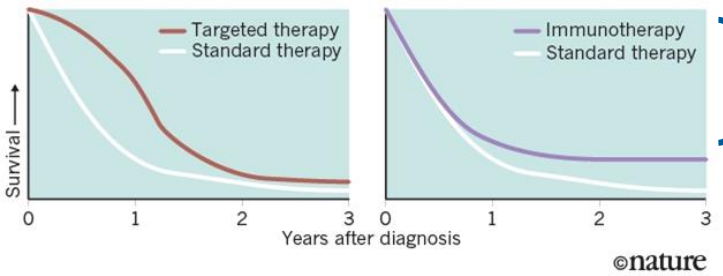
Anti-CD200R1 Antibody for Hard-to-Treat Solid Tumors Phase 1/2a

**Wholly owned; development ongoing in multiple relapsed/refractory solid tumors (including neuroendocrine and ovarian)*

'610 Development Rationale

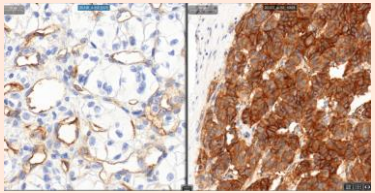
Addressing Critical Unmet Need in Solid Tumors

Patients + Caregivers DESPERATELY seeking survival



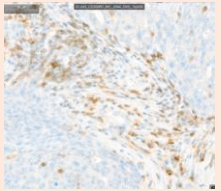
*Potential activity in **>60% of current patients** not deriving efficacy from PD-(L)1 inhibitors*

CD200 — Targeted by samalizumab for CLL and MM, failed to achieve target saturation*



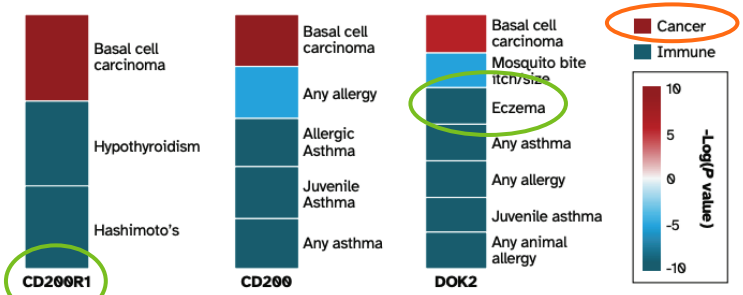
Highly expressed on tumor, stromal, and endothelial cells

CD200R1 — 23ME-00610 ('610) is first-in-class



Restricted immune expression: myeloid > T > B

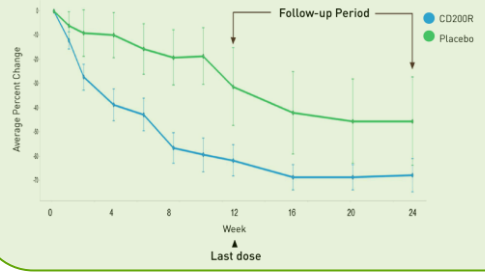
CD200/R1 is a dominant immune checkpoint*



CD200R1 (inhibitory cell surface receptor), CD200 (CD200R1 ligand), and DOK2 (involved in the CD200/CD200R1 signaling pathway).

*PMIDs: 12960329, 23602662, 22264927, 19786546, 15557172, 22491458, 15220441, 34326171, 18081533, 24388216, 11099416

Mean EASI Percent Change from Baseline by Treatment in Atopic Dermatitis*



Lilly's Ucenprubart: *Clinical POC for CD200/R1 agonism in immune disease*

*PMID: 31443741; https://investor.lilly.com/static-files/9efbede9-bd6a-4d7b-823e-2996b1c2d114

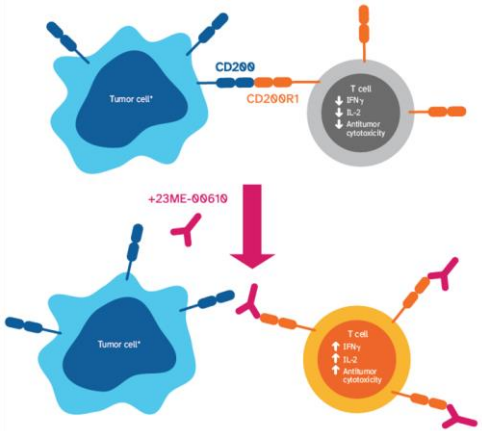
23andMe®

23ME-00610 ('610), a Fully Humanized, Effectorless IgG1, Inhibits Immunosuppressive Signaling via High Affinity Binding to CD200R1

'610 Primary Pharmacology*

- Subnanomolar affinity
- Kills tumor cells in vitro
- Anti-tumor activity in vivo
- Potential for monotherapy
  - *activity on huPBMCs that do not respond to PD-1 antibody*
- Potential for combination

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



'610 Clinical Development*

- Well tolerated up to 1400 mg
- PK supports Q3W (or better)
- Promising therapeutic index, projected dose ≥ ~600 mg
- Monotherapy dev ongoing
  - *Further expansion in NE and OC for safety, PK, PD and dose selection*
- Indication CDPs and TPPs

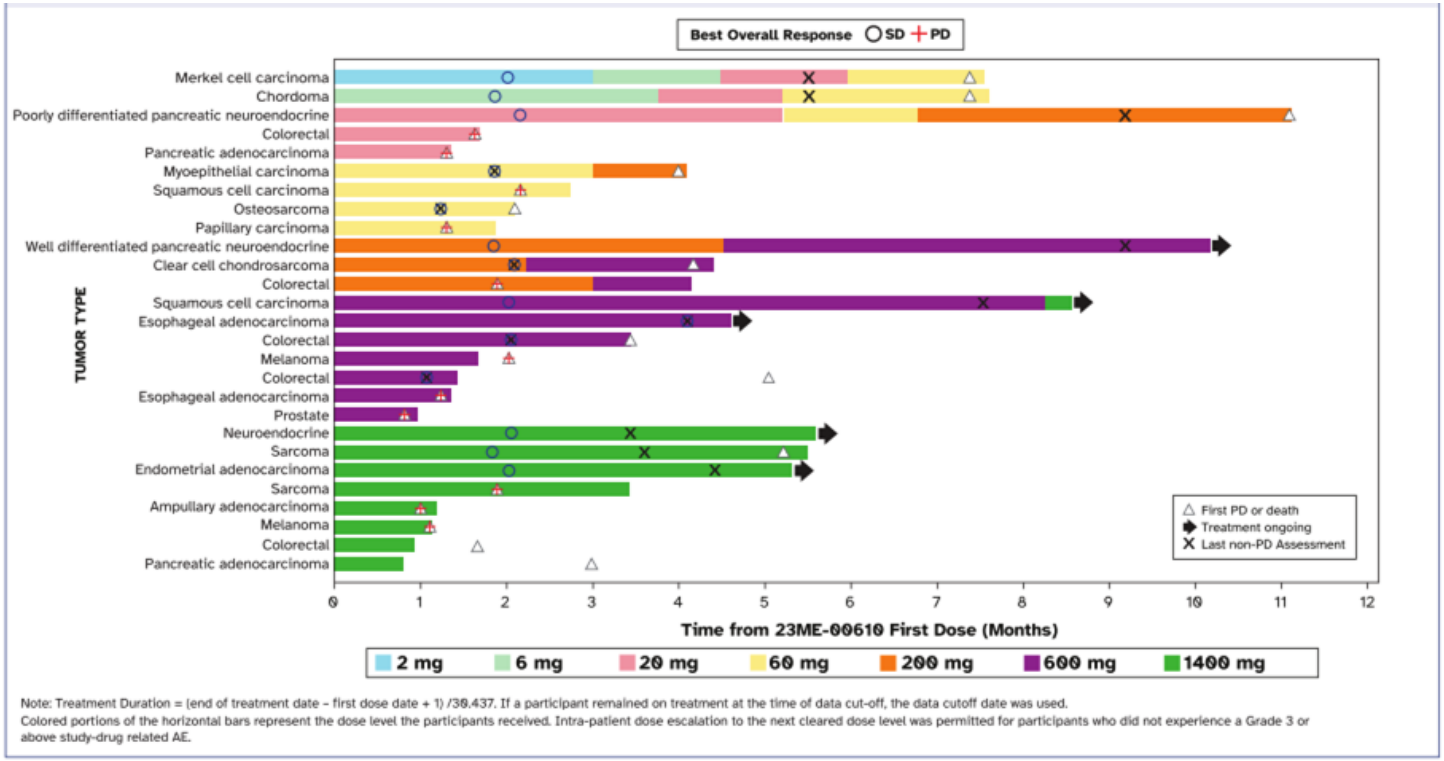
* PMID: 37288324

*CD200-expressing cell types include tumor, stroma and endothelial
IFN, interferon; IL, interleukin

* Rasco, et al., 2023, SITC Annual Meeting #619; Glatt, et al., 2023 SITC Annual Meeting #609

'610 Phase 1 Results: Dose Escalation Duration of Treatment

Stable disease rate across ALL Phase 1 patients is 52% with median duration of 18.6 weeks



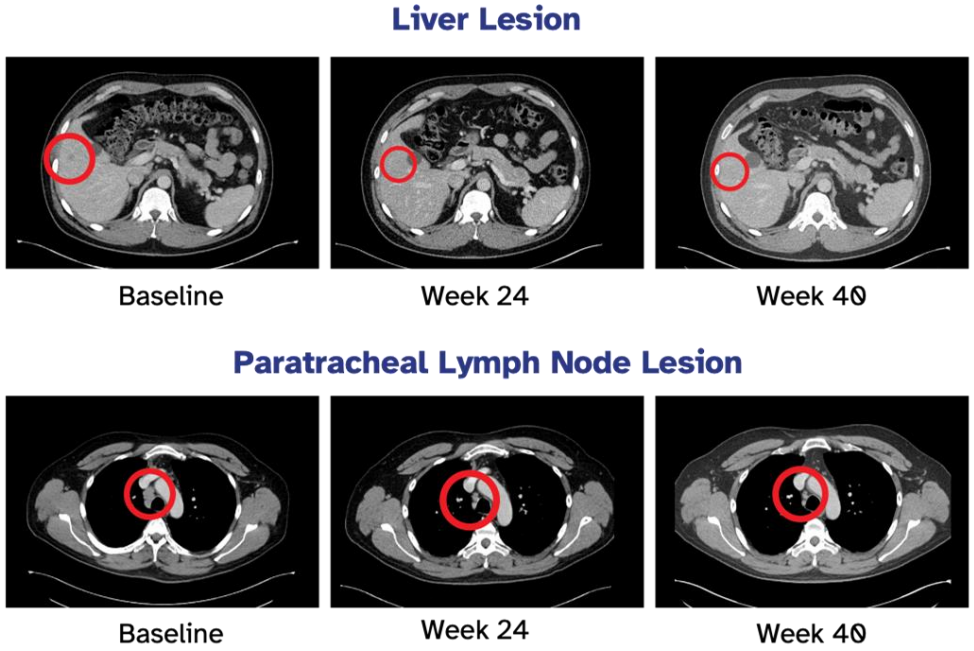
Note: Treatment Duration = (end of treatment date – first dose date + 1) /30.437. If a participant remained on treatment at the time of data cut-off, the data cutoff date was used.
Colored portions of the horizontal bars represent the dose level the participants received. Intra-patient dose escalation to the next cleared dose level was permitted for participants who did not experience a Grade 3 or above study-drug related AE.

May 15, 2023 data cut-off date.

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

23andMe®

'610 Preliminary Clinical Activity in Neuroendocrine Cancer



- 23ME-00610 treatment was well tolerated
- **19%** reduction in target lesions at Week 24 and Week 40 assessment
- **58%** size reduction in longest dimension of paratracheal lesion
- Patient continues on study drug at Cycle 13 with stable disease at time of data cutoff (May 2023)

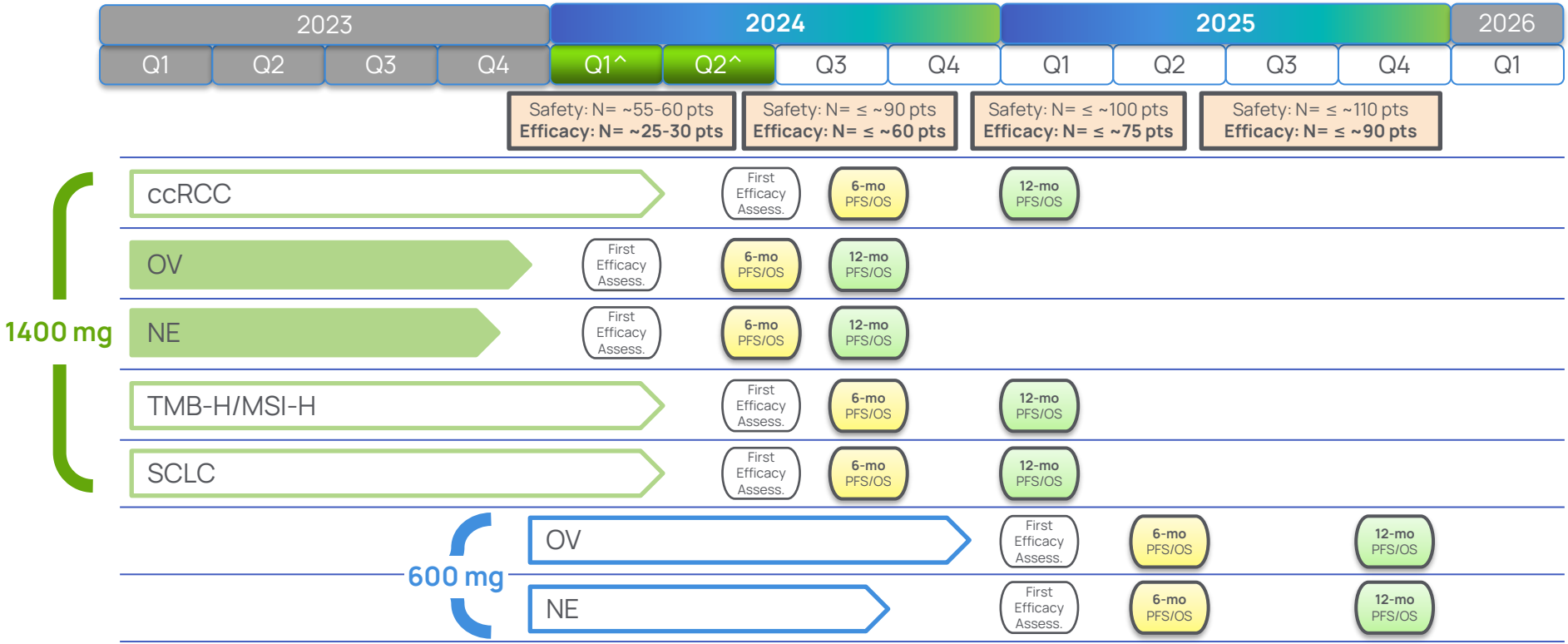
Rasco, D, et al., 2023, SITC Annual Meeting #619

'610 Phase 2a Data: Estimated Timeline*

N=15 in each cohort
Enrolling
Fully Enrolled

First Efficacy Assess = ie., Preliminary ORR, patients continue to be scanned

Safety in Phase 2a Population
Efficacy in Phase 2a Population



^Genotyping, tumor (archival) CD200/R1 IHC, tumor RNAseq, and pre/on-treatment tumor immunophenotyping exploratory analyses **to identify potential correlates with activity**

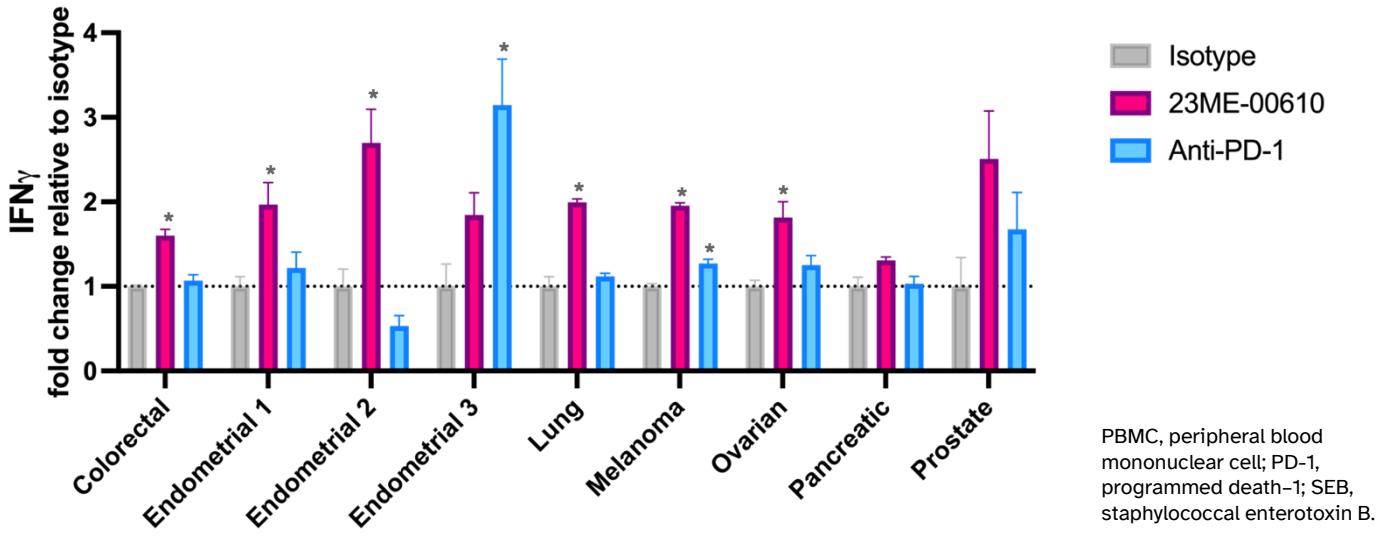
ccRCC = clear cell renal cell carcinoma
OV = ovarian cancer (predominantly non-clear cell histology)
NE = neuroendocrine
TMB-H/MSI-H = tumor mutational burden / microsatellite instability high tumors
SCLC = small cell lung cancer (extensive stage)

*Part of the Phase 1/2a clinical study of '610. Strictly estimated dates for discussion purposes only. Based on calendar year. Subject to change.

23andMe®

'610 Differentiation: Inhibition of CD200R1 Has the Potential to Address **Resistance to Anti-PD1 Therapies**

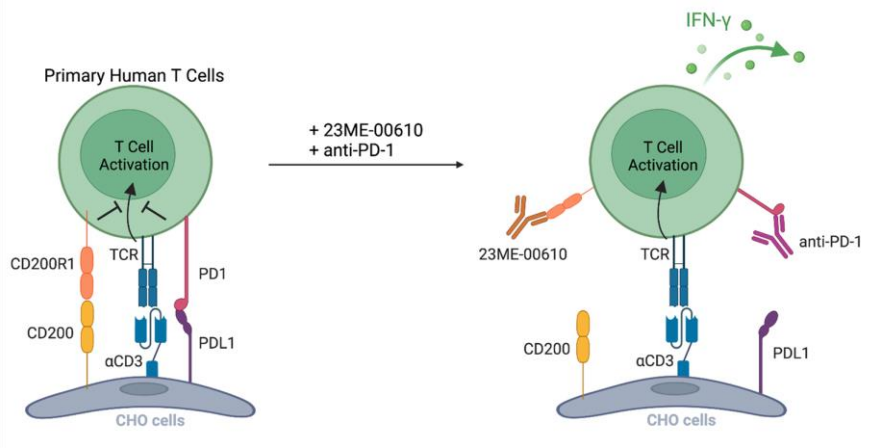
Blocking the CD200R1 pathway enhanced IFNγ production from SEB-stimulated PBMCs compared to isotype control and anti-PD1 in the majority of samples tested

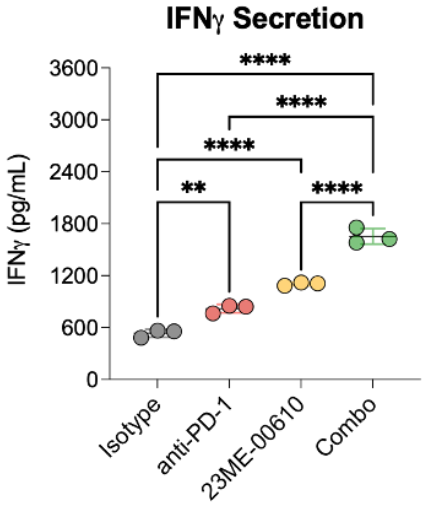


PBMC, peripheral blood mononuclear cell; PD-1, programmed death–1; SEB, staphylococcal enterotoxin B.

PBMCs from each respective patient were incubated with 100 nM of 23ME-00610, anti–PD-1, or isotype control. Cells were stimulated with SEB. IFNγ levels were determined by enzyme-linked immunosorbent assay. Mean biologic triplicates were normalized to isotype control. * p-value<=0.05 compared to control

'610 Differentiated Combo Potential: Anti-**CD200R1 with Anti-PD-1** Potentially Enhances Immune Activation





*23andMe internal data

- **Preliminary data from ex-vivo combination of anti-PD-1 and anti-CD200R1 blockade increased IFNγ (interferon-gamma) secretion from primary human T-cells**

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 Next Steps

- Complete enrollment of Phase 2a Dose Expansion Cohorts
  - Recently expanded **Neuroendocrine**, **Ovarian** cohorts
  - **Initial Phase 2a data** cohorts planned to be presented mid-2024
  - **Clinical development planning for Fast-to-Market strategies**
  - Potential clinical combinations with **assets with complementary mechanisms**, to support earlier line indications

- Seeking partnerships to expand Phase 2a and conduct randomized Phase 2b/3 clinical trials – ***multiple readouts expected in 2024***

23ME-01473

Genetically validated NK Cell Activator (Anti-ULBP6)

Antibody for [Metastatic] Solid Tumors

23ME'1473: Tumor Cell Killing-Enhanced Antibody Targets Major Resistance Mechanisms Hampering Immune Oncology

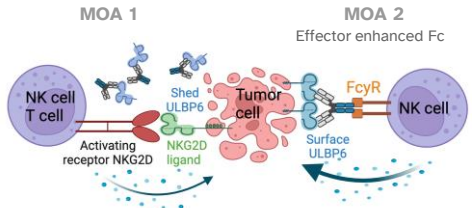
Targeting NK cells and NKG2D shows clinical promise

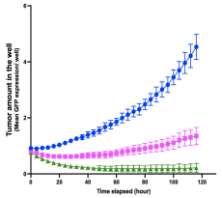
ULBP6 inhibition could benefit patients in broad range of tumor types with neoantigen loss

Tumor type	Tumor ULBP6	Soluble ULBP6	Loss of antigen presentation[1]
HNSC[2]	+++	*Under CDA*	++
CESC[3]	+++	*Under CDA*	+++
Additional tumor types under CDA	+++	*Under CDA*	+++

[1]Dhatchinamoorthy et al., Front Immunol 2021

Dual MOA achieves synergistic NK activation and tumor cell killing





MOA1
NKG2D activation

MOA2
NKG2D activation + ADCC = **23ME-01473**
Effector enhanced

23andMe developed major methodological improvements to targeting ULBP6

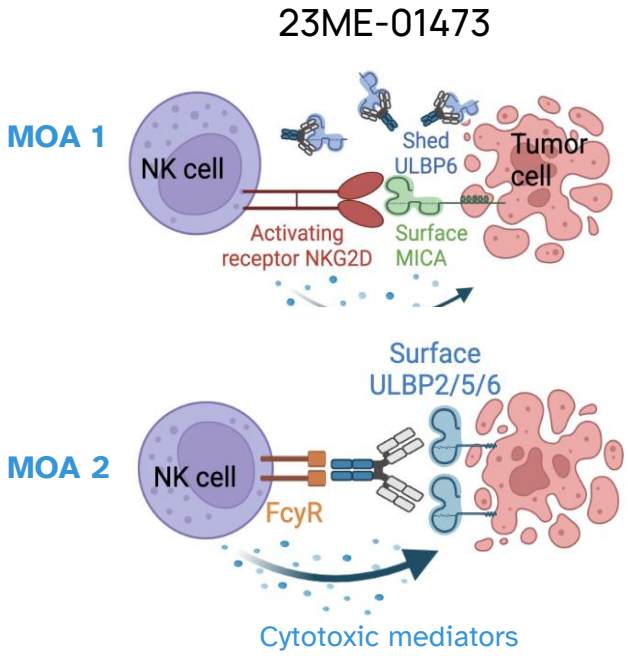
External clinical validation:
Monotherapy activity observed in NKG2D pathway activator (related mechanism) with complete and partial responses at a tolerable dose in early phase clinical trial[4]

23andMe '1473 targets the highest affinity NKG2D ligand with a tumor cell killing-enhanced antibody

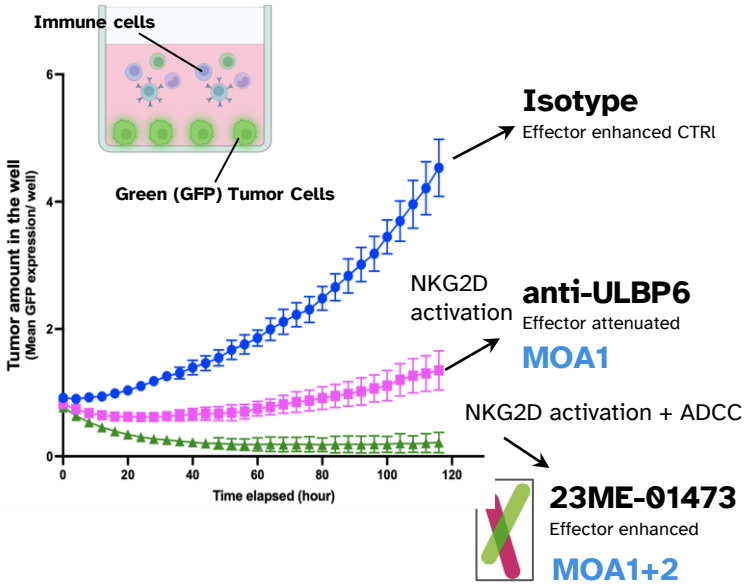
[4]Wang, et al., CLN-619 ASCO 2023

[2]HNSC, Head and Neck Squamous Cancer;
[3]CESC, Cervical Squamous Cell Cancer

'1473 Dual MOA: Effector Enhanced Antibody Binds to Tumor Cell Surface ULBP6/2/5 to Bolster NK Cell Antitumor Activity via ADCC



ADCC increases tumor cell killing



23andMe Therapeutics: Target Discovery

Experienced Discovery Leadership

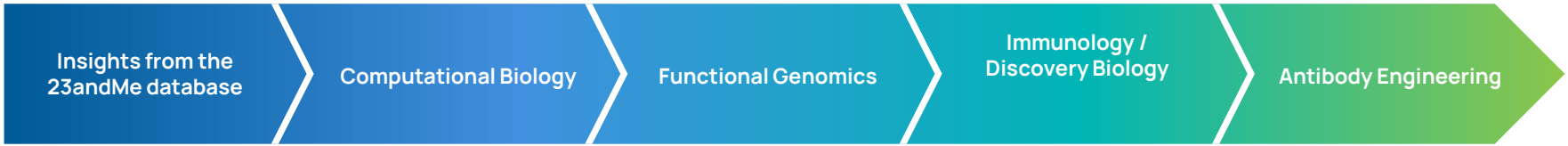
Bill Richards
Head of Therapeutics Discovery
AMGEN NURA BIO

Vladimir Vacic
Research Fellow, Computational Biology
NEW YORK GENOME CENTER

Patrick Collins
Director, Functional Genomics
AMGEN

Antony Symons
Senior Director Immunology & Inflammation
AMGEN

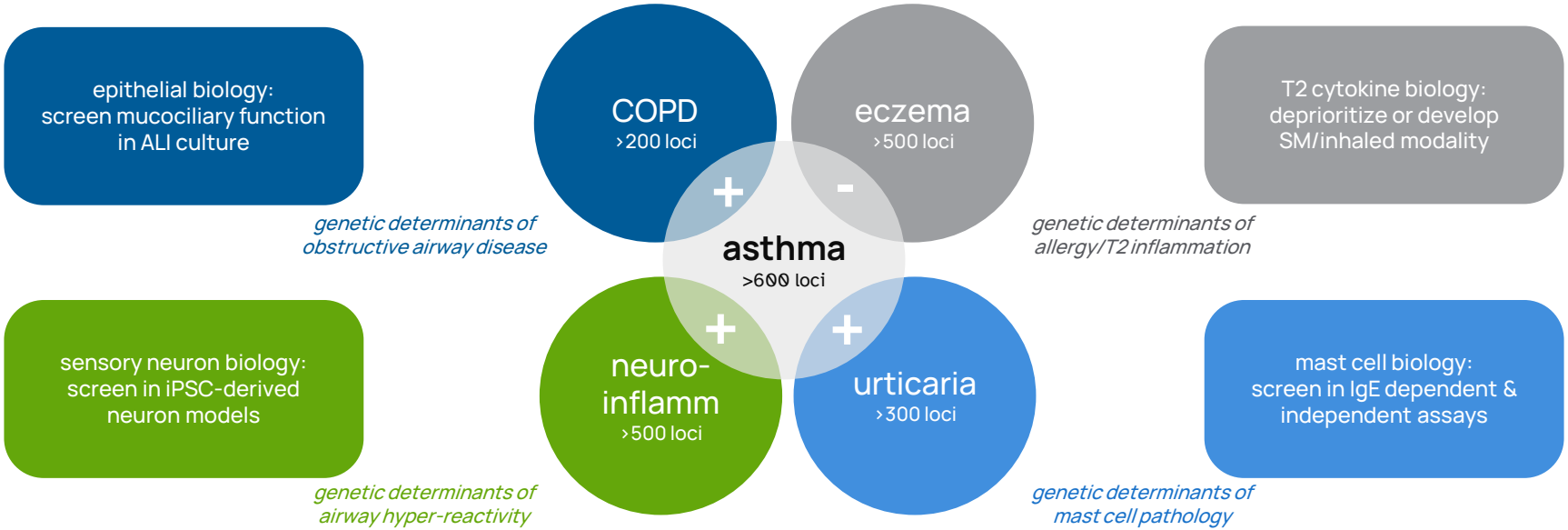
Germaine Fuh
Senior Director Antibody & Protein Engineering
Genentech



Experienced team that delivered genetics-based targets from discovery to the clinic

Leveraging Pleiotropy to Expand Airway Target Space

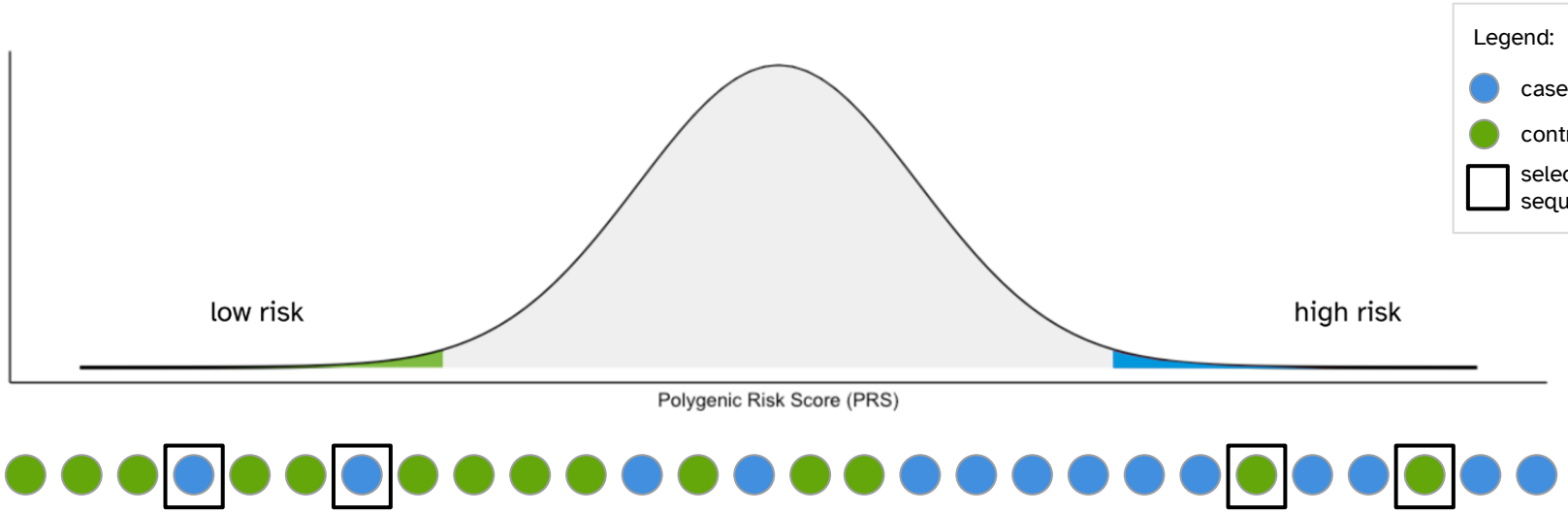
Hypothesis: loci associated with related phenotypes prioritize biologies not addressed by standard of care



Pleiotropy + functional genomics = best targets

Strategic Sequencing Based on Polygenic Risk Scores

Sequencing individuals from the tail ends of the polygenic risk score (PRS) distribution for whom the actual disease status does not match predictions



Discovery of genes harboring rare variants of large effect

FxG in Respiratory Disease & Beyond

	Cell type	Disease opportunities*
	Macrophage	Broad immune: skin, lung, GI
	Mast cells	Urticaria, allergy, RA, eczema
	Fibroblasts	Fibrosis, lung, skin, RA, IPF
	T cell	Broad immune: skin, lung, GI
	Sensory Neurons	Respiratory, IBD, eczema
	Endothelial cells	RA, sarcoidosis, IBD, PAH
	Airway Smooth Muscle	Asthma, COPD, PAH
	Dendritic cell	Broad autoimmune: T1D, Graves
	Keratinocytes	Skin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