Identification of Neuropsychiatric CNVs in a Health System Population:

High Prevalence, Penetrance, and Personal Utility

First Annual Conference on Precision Psychiatry
October 1, 2021

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Geisinger

Conflicts of Interest

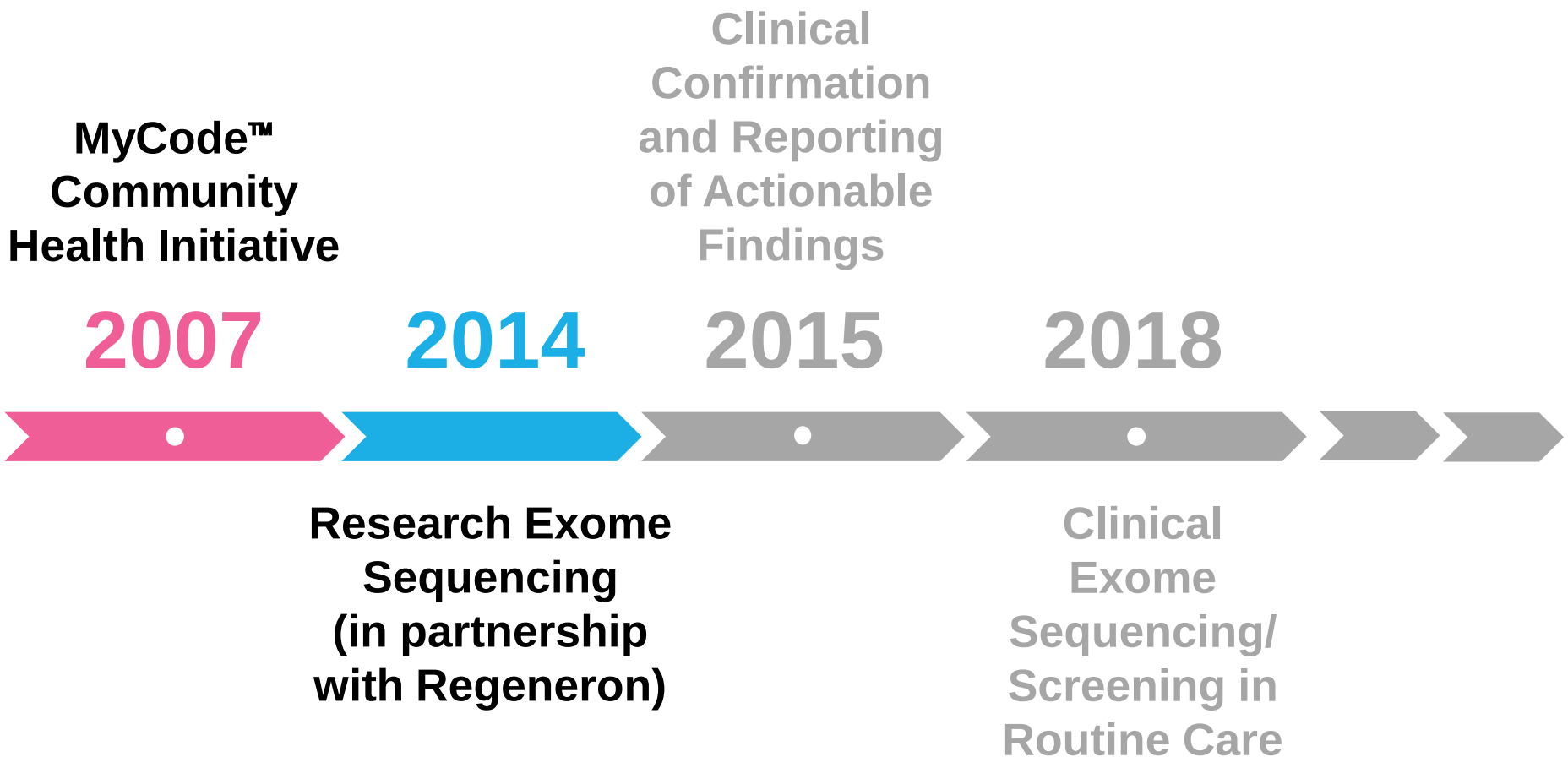
Christa Lese Martin – nothing to disclose

Life saving stories…

- 39-year old woman in MyCode research project found to have disease-causing change in the *KCNQ1* gene; her mother died suddenly in her sleep at age 26
- *KCNQ1* – potassium channel gene; causes form of arrhythmia called Long QT syndrome which can result in sudden death
- Familial testing revealed her two sons (ages 9 and 13) also carry the change in *KCNQ1;* both have prolonged QT intervals, consistent with Long QT syndrome
- Mother and boys prescribed beta-blockers --- and family has automatic external defibrillator which they take to all of the boys' sporting events

"I thank God for this program, that this [mutation] was found and I'm not burying one of my kids."

Using genomics to improve patient care: the evolution of Geisinger Genomic Precision Health



mycode Community Health Initiative

- High consent rate (~65-85%)
- Recruited throughout system - both in-person (in clinics) and online (MyGeisinger)
- Exome and genotype data linked to clinical information from EHR and claims data from Geisinger Health Plan
- Cohort Characteristics:
  - Most of European ancestry (~95%)
  - Median age of 54 years
  - Median 13.8 years of longitudinal EHR data
  - 46 clinical encounters; 426 lab test values; 717 vital measurements

Carey et al. (2016) GIM

Using genomics to improve patient care: the evolution of Geisinger Genomic Precision Health

2007 — **MyCode™ Community Health Initiative**

2014 — **Research Exome Sequencing (in partnership with Regeneron)**

2015 — **Clinical Confirmation and Reporting of Actionable Findings**

2018 — **Clinical Exome Sequencing/ Screening in Routine Care**

61 actionable genes (ACMG/CDC) – many cardiac disease and cancer; only reporting pathogenic and likely pathogenic variants

MyCode® Scorecard

Total consented participants	291,504
Samples provided	203,384
DNA sequences available for research	184,293
DNA sequences eligible and analyzed for clinical review	94,453
Participants with clinical result reported	2,733

Geisinger

As of September 1, 2021

Using genomics to improve patient care: the evolution of Geisinger Genomic Precision Health

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What about Precision Health for Brain Disorders?

Life *changing* stories…

- 48-year old man in MyCode project found to have pathogenic 22q11.2 deletion
- Lives with parents, single
- Graduated HS, certificate in welding, drives, independently manages appointments / finances
- Typical 22q11.2 deletion facial appearance; no history of chronic medical conditions or surgeries
- Psychotic episode at 35, required hospitalization; psychiatry attempted discontinuation of medication at age 40 with recurrence of psychosis
- 22q del diagnosis supported continued treatment; currently stable on low dose of antipsychotic medication

"It feels good to know that there's a medical name for my condition."

Neurodevelopmental/psychiatric Disorders (NPD)

- Characterized by impairments in cognition, communication, behavior, and/or motor functioning
- Impact about 14-18% of the nation's children and adults
- ~30% have genetic etiology
- Include disorders such as:

- autism spectrum disorder
- intellectual disability/ developmental delay
- epilepsy
- ADHD
- cerebral palsy
- bipolar disorder
- schizophrenia
- depression
- anxiety

Significant Genetic Heterogeneity and Variable Expressivity in NPD

CNV	Frequency in clinical cohorts*	Autism spectrum disorder	Intellectual disability or developmental delay	Schizophrenia	Epilepsy
22q11.2	1 in 167	√	√	√	√
16p11.2	1 in 241	√	√	..	√
1q21.1	1 in 309	√	√	√	√
15q13.2-q13.3	1 in 358	√	√	√	√
7q11.23	1 in 415	√	√	..	√
15q11.2-q13	1 in 553	√	√	√	√
17q21.31	1 in 700	√	√	..	√
16p13.11	1 in 788	√	√	√	√
17q12	1 in 985	√	√	√	√
17p11.2	1 in 985	√	√	..	√
8p23.1	1 in 1854	√	√	..	√
5q35	1 in 1970	√	√	..	√
3q29	1 in 2101	√	√	√	..

A. Moreno De Luca

S. Myers

Moreno De Luca et al.
Lancet Neurol 2013

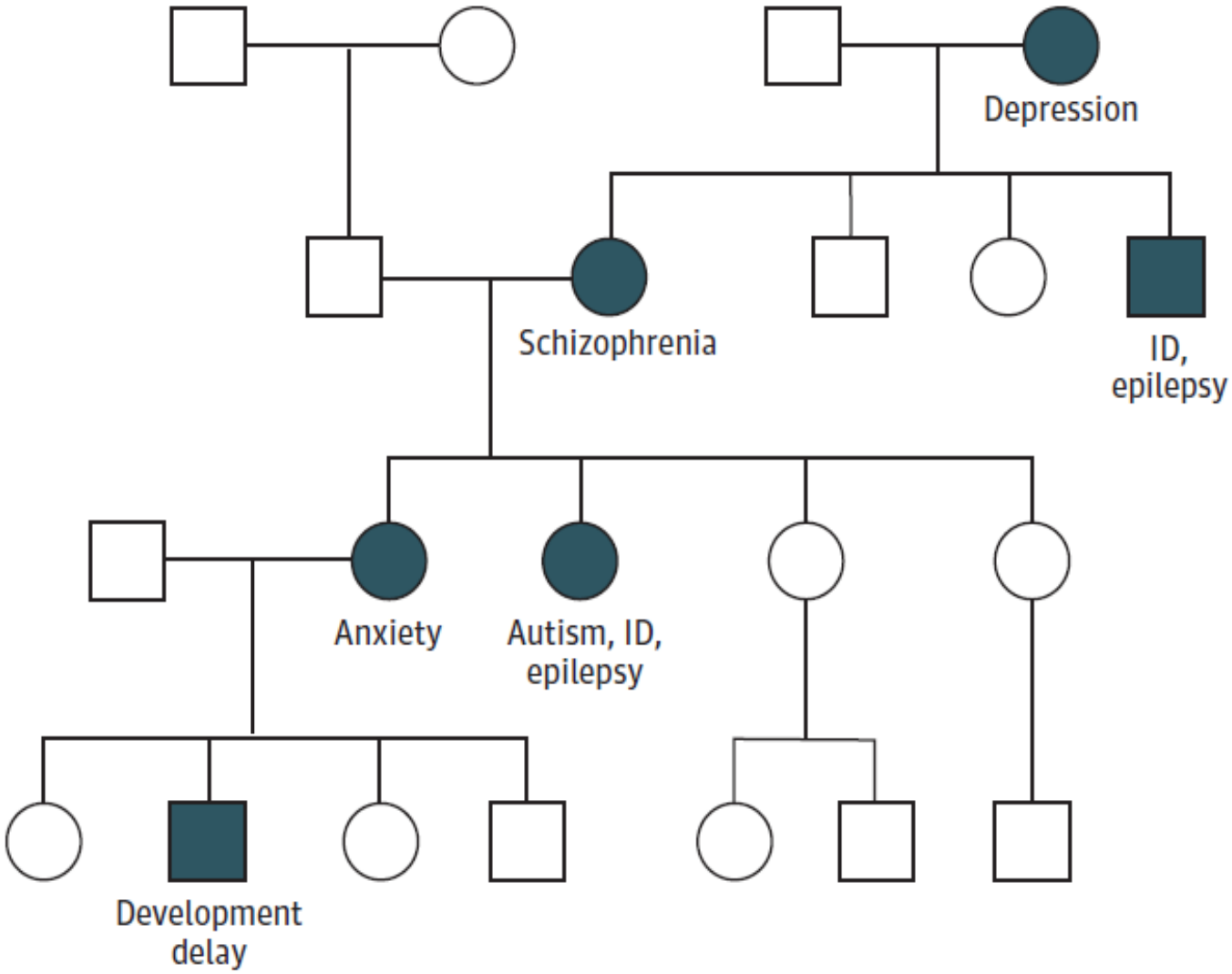
Significant Genetic Heterogeneity and Variable Expressivity in NPD

Gene	Autism spectrum disorder	Intellectual disability or developmental delay	Schizophrenia	Epilepsy
A2BP1	√	√	√	√
AUTS2	√	√	..	√
CACNA1C	√	√	√	√
CASK	..	√	√	√
CDKL5	√	√	..	√
CNTNAP2	√	√	√	√
DISC1	√	√	√	..
EHMT1	√	√	√	√
FMR1	√	√	√	√
FOXP1	√	√	..	√
FOXP2	√	√	√	..
GRIN2B	√	√	√	..
MBD5	√	√	..	√

Estimated at least 500 genes involved in NPD

Moreno De Luca et al. Lancet Neurol 2013

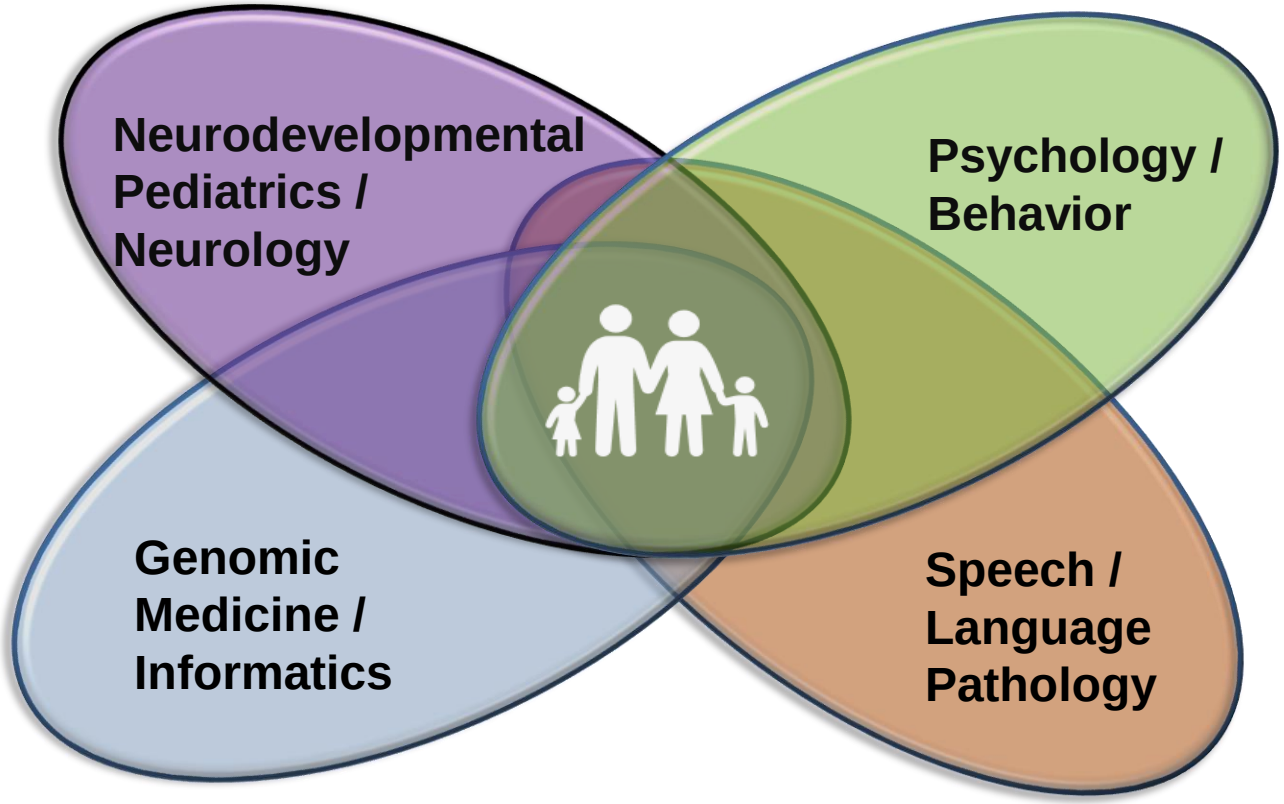
Variable Expressivity within Families



del 15q13.3

Martin, Wain et al., JAMA Psych 2020

Geisinger Autism & Developmental Medicine Institute
A Precision Health Approach to Clinical Care



Genetic testing ordered as part of routine clinical care:

- Fragile X
- Exome Sequencing with CNV Analysis
- Diagnostic Yield = 30-40%

>9,000 unique patients to date
~35 new referrals per week
~6,200 visits per year (new and returns)

Behavioral vs. Etiological Diagnosis

Autism

Autism

Autism

Behavioral vs. Etiological Diagnosis

Autism
Fragile X syndrome

Autism
22q11.2 del

Autism
Angelman syndrome

Behavioral vs. Etiological Diagnosis

Autism
Fragile X syndrome

Autism
22q11.2 del

Autism
Angelman syndrome

- Provides a diagnosis and accurate recurrence risk estimates
- Allows for targeted medical monitoring
- Enables development of etiology-specific interventions based on an individual's risk/resilience (primary etiology + polygenic risk scores)
- Genes to Mental Health (G2MH) Network (NIMH/NICHD)

Unbiased, Genotype First Ascertainment of NPD

- To date, most studies on the neurodevelopmental/psychiatric phenotypic effects of genomic variants have investigated clinical or research cohorts with ID, ASD, SCZ, and other disorders.
- These largely pediatric studies are biased towards ascertainment of the most severe phenotypic consequences of genomic variants.
- More data is needed on the clinical consequences of genomic variants in unselected populations to understand broader phenotypes.

Research

Published online July 22, 2020
PMID: 32697297

JAMA Psychiatry | Original Investigation

Identification of Neuropsychiatric Copy Number Variants in a Health Care System Population

Christa Lese Martin, PhD*; Karen E. Wain, MS*; Matthew T. Oetjens, PhD; Kasia Tolwinski, PhD; Emily Palen, MS; Abby Hare-Harris, PhD; Lukas Habegger, PhD, MS; Evan K. Maxwell, PhD, MS; Jeffrey G. Reid, PhD; Lauren Kasparson Walsh, MS; Scott M. Myers, MD; David H. Ledbetter, PhD

*Co-first authors

- Examined the ***prevalence***, ***penetrance***, and ***personal utility*** of neuropsychiatric copy number variants (CNVs)
- Are genetic causes of neuropsychiatric disorders ready for inclusion in population screening efforts?

Prevalence of Pathogenic CNVs

MyCode Cohort CNV Analysis

- 90,620 patient-participants with sequence data passing QC for exome-based CNV calling
- Determined frequency of 31 pathogenic recurrent NPD CNVs (>250kb; e.g., 22q11.2 deletion; ClinGen Dosage Score = 3)
  - CNVs called from exomes using CLAMMS algorithm (Packer et al., 2015); validated by Illumina SNP data with PennCNV
  - CLIA confirmation for clinical reporting to participants

M. Oetjens

A. Hare Harris

MyCode Cohort CNV Analysis - Prevalence

- 708/90,595 **(0.8%)** individuals have a pathogenic CNV
- *Only* 41/708 (5.8%) had a previously known genetic diagnosis in the EHR

  - Mean age = 20.33 yrs (compared to 50.04 yrs for all CNV+ individuals)

Multiple large, population-based studies estimate the prevalence of pathogenic CNVs to be ~1%

Table 1. Comparison of NPD-Associated CNV Prevalence in DiscovEHR, deCODE, EGCUT, and UK Biobank

CNV	Dosage	No. (%) DiscovEHR (n = 90 595)	deCODE[47] (n = 101 655)	EGCUT[46] (n = 7877)	UK Biobank[48] (n = 421 268)
Deletion	NA	354 (0.391)	226 (0.222)	16 (0.203)	745 (0.177)
Duplication	NA	356 (0.393)	276 (0.272)	28 (0.355)	879 (0.209)
CNV[e]	NA	708 (0.782)	502 (0.494)	44 (0.559)	1624 (0.386)

Broader Cumulative CNV list	0.8%	1.16%	0.7%	1.0%

Martin, Wain et al., JAMA Psych 2020

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DELETIONS					
1q21.1 (*GJA5*)[a,b]	del	59 (0.065)	35 (0.034)	3 (0.038)	113 (0.027)
3q29 (*DLG1*)	del	4 (0.004)	3 (0.003)	0	9 (0.002)
5q35 (*NSD1*)	del	0	NR	0	0
7q11.23 (*ELN*)[a]	del	4 (0.004)	4 (0.004)	1 (0.013)	1 (0)
8p23.1 (*GATA4*)	del	0	NR	1 (0.013)	4 (0.001)
10q23 (*BMPR1A*)	del	1 (0.001)	NR	NR	3 (0.001)
15q11.2q13.1 BP1-3 (*UBE3A*)	del	5 (0.006)	1 (0.001)	0	1 (0)
15q13.3 BP4-5 (*CHRNA7*)[a]	del	55 (0.061)	25 (0.025)	2 (0.025)	42 (0.010)
15q24 (*SIN3A*)[a]	del	2 (0.002)	NR	0	1 (0)
16p13.11 (*MYH11*)[a]	del	71 (0.078)	38 (0.037)	2 (0.025)	131 (0.031)
16p11.2 distal (*SH2B1*)	del	28 (0.031)	19 (0.019)	NR	58 (0.014)
16p11.2 (*TBX6*)[a]	del	59 (0.065)	43 (0.042)	4 (0.051)	110 (0.026)
17p12 (*PMP22*)	del	31 (0.034)	32 (0.031)	3 (0.038)	237 (0.056)
17p11.2 (*RAI1*)	del	4 (0.004)	NR	0	2 (0)
17q11.2 (*NF1*)[a]	del	3 (0.003)	NR	0	9 (0.002)
17q12 (*HNF1B*)[a]	del	4 (0.004)	7 (0.007)	0	9 (0.002)
17q21.31 (*KANSL1*)	del	0	NR	0	0
22q11.2 (*TBX1*)[a,c]	del	23 (0.025)	18 (0.018)	0	10 (0.002)
22q11.2 distal	del	1 (0.001)	1 (0.001)	0	5 (0.001)

Martin, Wain et al., JAMA Psych 2020

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DUPLICATIONS					
1q21.1 (*GJA5*)[b]	dup	90 (0.099)	60 (0.059)	6 (0.076)	177 (0.042)
5q35 (*NSD1*)	dup	0	NR	0	0
7q11.23 (*ELN*)	dup	8 (0.009)	1 (0.001)	1 (0.013)	14 (0.003)
8p23.1 (*GATA4*)	dup	0	NR	0	6 (0.001)
15q11.2q13.1 BP1-3 (*UBE3A*)	dup	3 (0.003)	13 (0.013)	0	19 (0.005)
16p11.2 (*TBX6*)	dup	63 (0.07)	51 (0.050)	7 (0.089)	138 (0.033)
17p12 (*PMP22*)	dup	38 (0.042)	28 (0.028)	2 (0.025)	124 (0.029)
17p11.2 (*RAI1*)	dup	0	NR	0	5 (0.001)
17q11.2 (*NF1*)	dup	4 (0.004)	NR	NR	2 (0)
17q12 (*HNF1B*)	dup	41 (0.045)	38 (0.037)	7 (0.089)	101 (0.024)
22q11.2 (*TBX1*)[d]	dup	108 (0.119)	85 (0.084)	5 (0.063)	280 (0.066)
22q11.2 distal	dup	1 (0.001)	NR	0	13 (0.003)
Deletion	NA	354 (0.391)	226 (0.222)	16 (0.203)	745 (0.177)
Duplication	NA	356 (0.393)	276 (0.272)	28 (0.355)	879 (0.209)
CNV[e]	NA	708 (0.782)	502 (0.494)	44 (0.559)	1624 (0.386)

Martin, Wain et al.,
JAMA Psych 2020

Point of comparison

0.8% NPD CNV prevalence

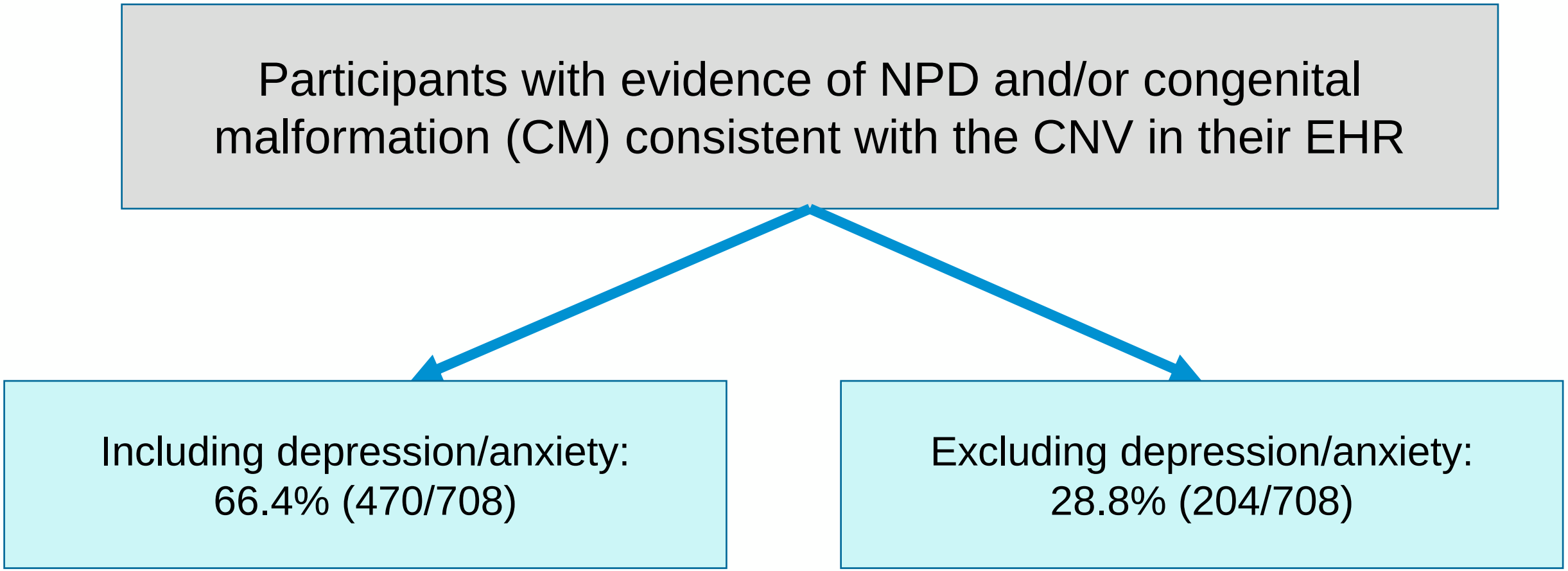
Familial hypercholesterolemia: 0.4-0.5%

Lynch syndrome: 0.2%

Hypertrophic cardiomyopathies: 0.2%

Penetrance Estimates for Pathogenic CNVs

MyCode Cohort CNV Analysis - Penetrance



MyCode Cohort CNV Analysis - Penetrance

- Congenital malformation codes (central nervous system, cardiac, kidney/urinary, genital, cleft lip/palate) were enriched in EHRs of CNV-positive individuals (OR 2.00; 95% CI 1.60-2.49; $p<0.001$)
- Cleft lip/palate was observed in CNV-positive group with 9.84 higher odds, and other CM groups were 2-3 fold higher in CNV-positive

Disclosing NPD CNVs to MyCode Participants

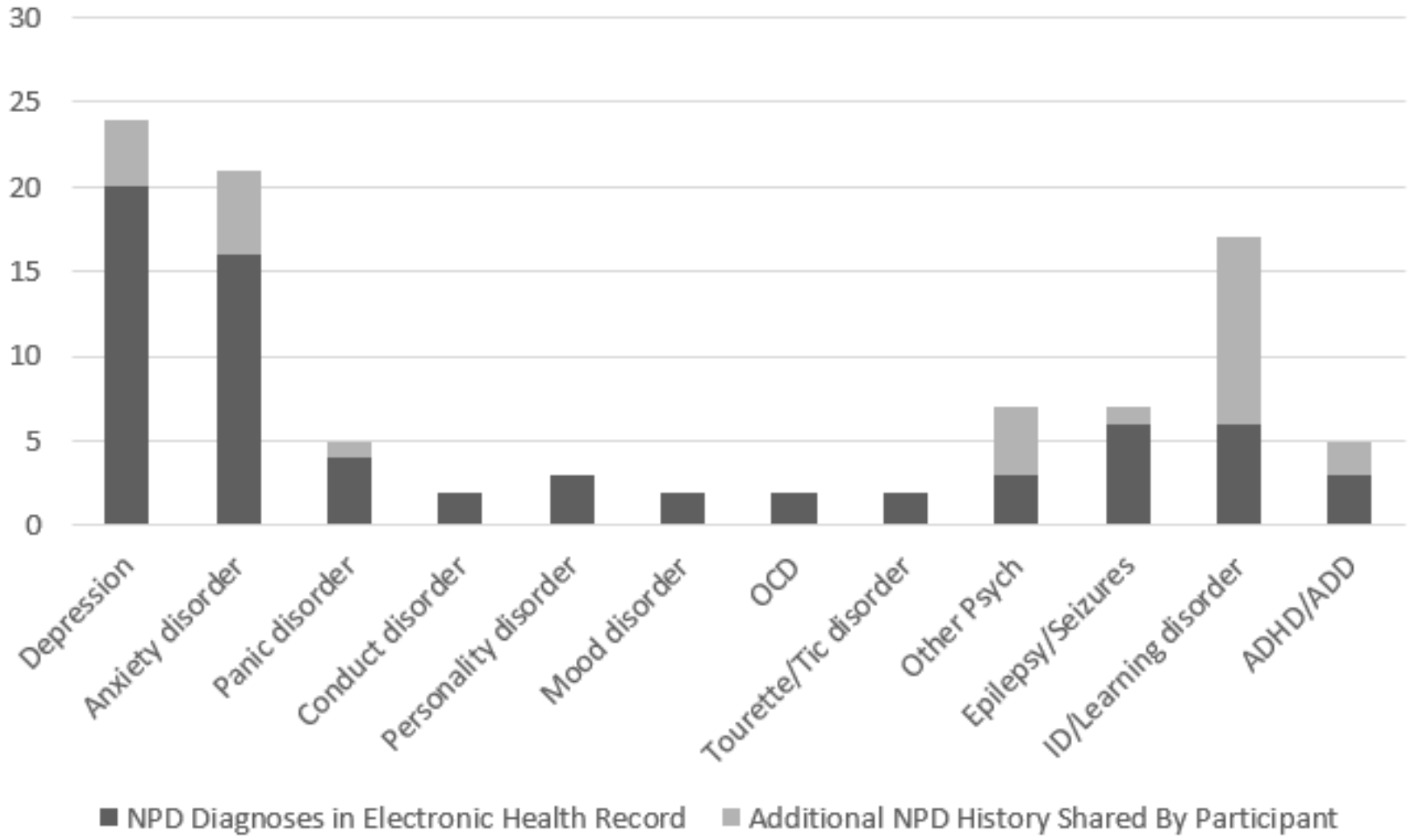
- **Select CNVs to be disclosed:**
  - Included recurrent, pathogenic CNVs mediated by segmental duplications
  - Clinical phenotypes that include NPD
  - Prioritized CNVs based on number and type of non-NPD medical implications
- **Requirements for returning results:**
  - Participant had NPD/CM documented in EHR
  - Age 18 years or older
  - Adequate consents on file and adequate sample available for clinical confirmation

9 CNVs
1q21.1 deletion
7q11.23 deletion
15q13.3 deletion
15q24 deletion
16p11.2 deletion
16p13.11 deletion
17q11.2 (NF1) deletion
17q12 deletion
22q11.2 deletion

K. Wain

Penetrance is underestimated by EHR alone

Participants receiving CNV results often shared NPD history not documented in EHR



Wain et al., J Pers Med (2021)

Point of comparison

35-70% NPD CNV penetrance estimate

Familial hypercholesterolemia:
30-50% risk for coronary event

Lynch syndrome:
52-82% lifetime colorectal cancer risk

BRCA1/2:
38-87% lifetime breast cancer risk

Personal Utility for Individuals with NPD CNVs

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17q11.2 (NF1) deletion
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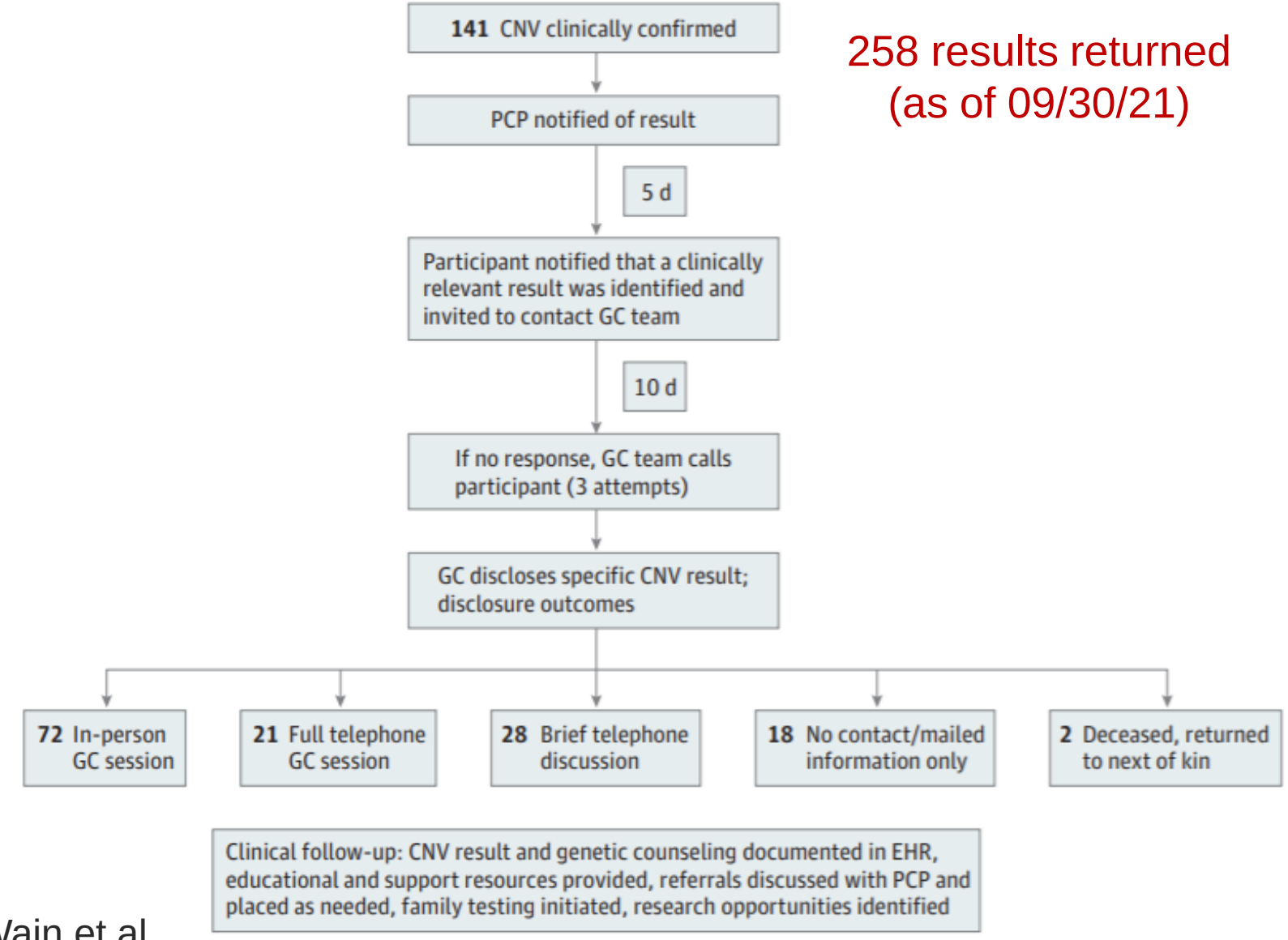
22q11.2 deletion

K. Wain

Figure 2. Genetic Screening and Counseling Disclosure Process and Outcomes From 141 CNV-Positive Participants



CNV indicates copy number variant; EHR, electronic health record; GC, genetic counselor; PCP, primary care professional.

258 results returned
(as of 09/30/21)

141 out of 280 total individuals were eligible to receive results

Gender

Female – 67%

Male – 33%

Age

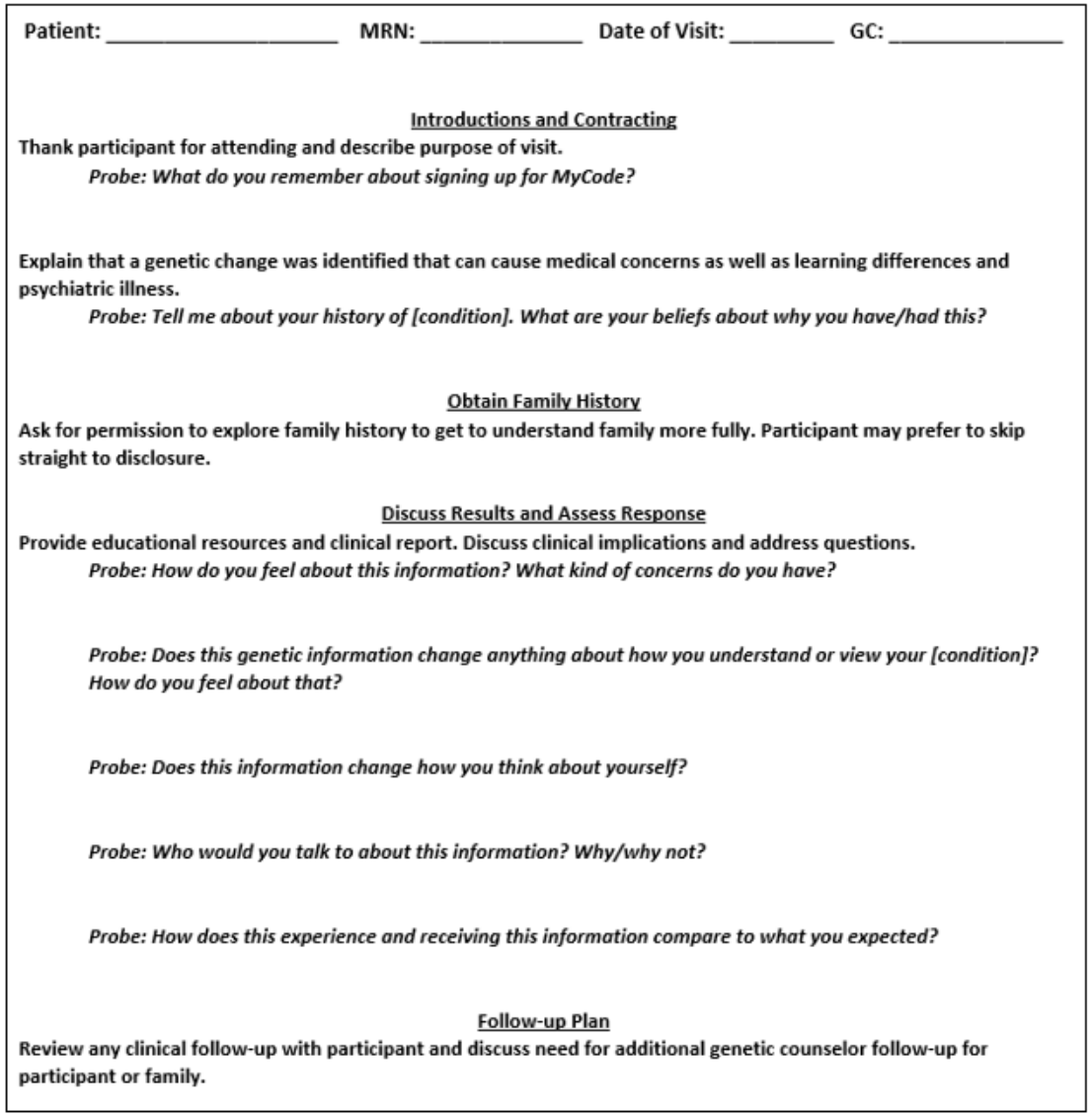
Average: 48.5 yrs

Range: 21-87 yrs

Martin, Wain et al., JAMA Psych 2020

Genetic Counseling Disclosure Session Outline

- Four domains of focus:
  - Consent experience
  - Immediate psychosocial reaction
  - Impact on medical beliefs and self-image
  - Communication about results
- Promoted consistency between GCs
- Used for post-session GC written notes about participant responses, quotes, etc.



Patient: _____ MRN: _____ Date of Visit: _____ GC: _____

Introductions and Contracting

Thank participant for attending and describe purpose of visit.

Probe: What do you remember about signing up for MyCode?

Explain that a genetic change was identified that can cause medical concerns as well as learning differences and psychiatric illness.

Probe: Tell me about your history of [condition]. What are your beliefs about why you have/had this?

Obtain Family History

Ask for permission to explore family history to get to understand family more fully. Participant may prefer to skip straight to disclosure.

Discuss Results and Assess Response

Provide educational resources and clinical report. Discuss clinical implications and address questions.

Probe: How do you feel about this information? What kind of concerns do you have?

Probe: Does this genetic information change anything about how you understand or view your [condition]? How do you feel about that?

Probe: Does this information change how you think about yourself?

Probe: Who would you talk to about this information? Why/why not?

Probe: How does this experience and receiving this information compare to what you expected?

Follow-up Plan

Review any clinical follow-up with participant and discuss need for additional genetic counselor follow-up for participant or family.

eFigure 1: A genetic counselor results disclosure session outline was used to guide sessions, promote consistency between genetic counselors, and to standardize participant response assessment for post-session data analysis.

Mixed Methods Assessment of Participant Experience

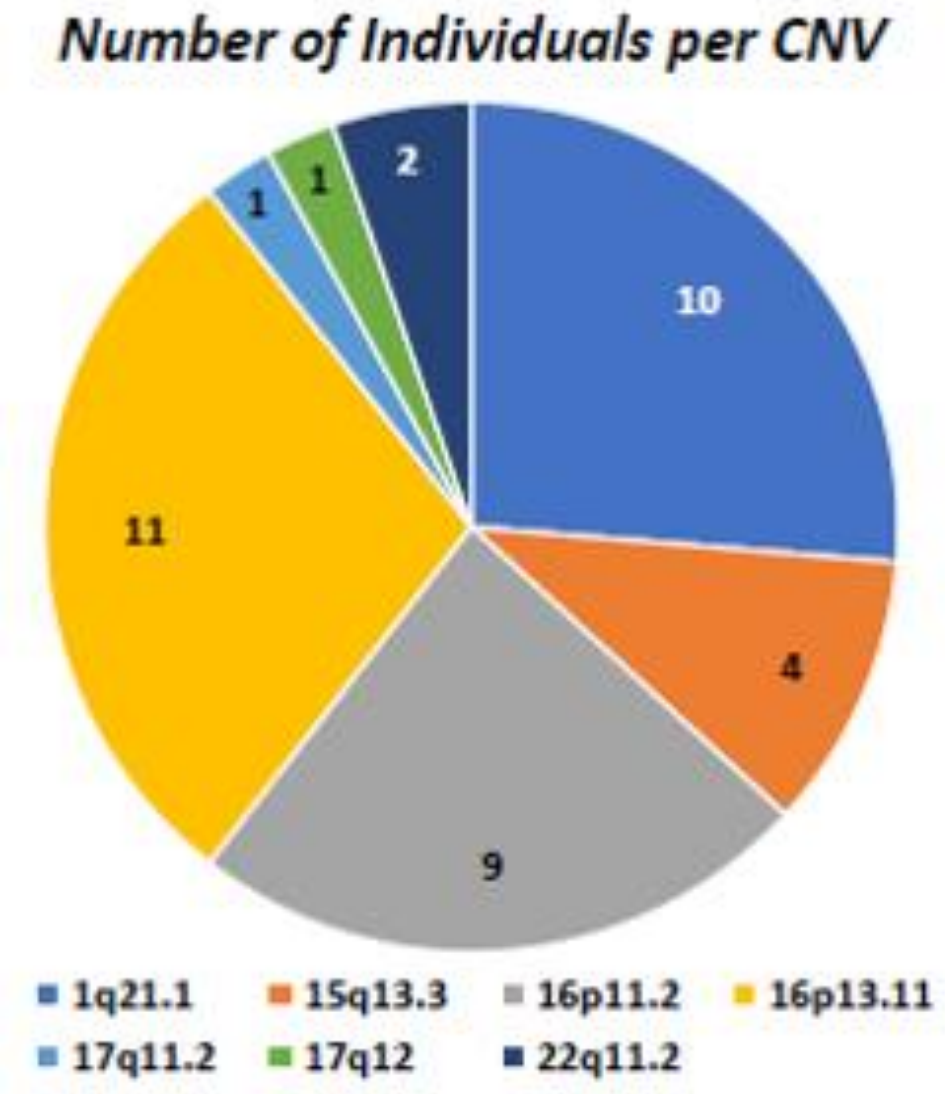
Qualitative data analysis of two data-sets

- GC written notes from GC session outline (n=38)
- Transcripts of audio-recorded sessions (n=14)
  - 13 additional transcripts (14 participants) with analysis now complete

Data-sets were assessed independently using a grounded theory approach

- Two independent coders generated codes for themes and subthemes
- Discussed to reach consensus and develop final codebooks
- For GC notes – coders discussed codebook with GC team as validation

Mixed Methods Assessment of Participant Experience

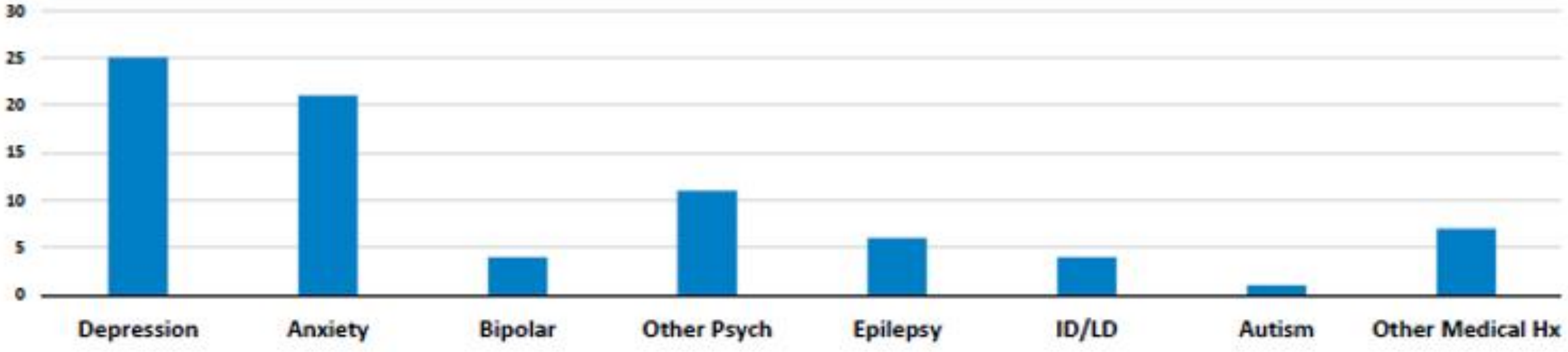


Mixed Methods Assessment of Participant Experience

Age and Sex Across Data-sets

	GC disclosure notes (n=38)	Transcribed audio-recordings (n=14)
Age (mean, range)	53.5 years, 21-87 years	49 years, 23-70 years
Sex	F = 24 (63%); M = 14 (37%)	F = 9 (64%); M = 5 (36%)

Number of Individuals per Clinical Diagnosis



Major Themes from CNV Disclosure Sessions Were Consistent Across Data-Sets

Discussed NPD history (e.g., learning/ interpersonal difficulties) that were not recorded in EHR	"I was a slow learner." (Female, 17q11.2) "I was left out… I was different from other kids." (Female, 1q21.1)
Had previously explained NPD as a result of social circumstances (trauma, family disruption)	"I do put a lot of [my learning disability on] what happened between mom and dad and the moving around." (Male, 16p11.2)
Expressed that CNV "fit" or "made sense" with lived experience	"I knew I had anxiety. I knew I had different things, but I didn't know where everything came from. This now brings everything around." (Female, 1q21.1)
Felt reassured that NPD was not their fault	"It was very helpful. It took a lot of guilt off." (Mother of Male, 22q11.2)
Reported that "sense of self" stayed the same or improved	"I think it does [change sense of self], because I realize there's a medical, that's something behind everything. It's not just all in your head." (Female, 1q21.1)
Positive and negative emotions were often expressed together	"I thought it was something bad, but it's bad and a good thing at the same time, that information that you gave me." (Female, 17q11.2)
Believed information to be valuable, for themselves and family members	"It feels good to know that there's a name for my condition." (Male, 22q11.2) "If this information is something that we can help [our son]… it's good to know that now and not more when he's… We can get a little bit more control of it now." (Wife of Male, 16p13.11)

Mixed Methods Assessment of Participant Experience

- Participants described CNV results as **personally valuable**.
- **Positive responses outweighed negative** responses. Negative emotions were related to recounting past experiences.
- **Results are actively incorporated** into personal narratives, their “sense of self” and their understanding of their medical and family histories.
- Learning and understanding the CNV information was often of a **“group” nature with their family.**
- Participants were **open to discussing their NPD history** with the GC and often **planned to share CNV result with family and healthcare providers**.

Point of comparison

Personal vs. clinical utility

"*Genomic information has personal utility if and only if it can reasonably be used for decisions, actions or self-understanding which are personal in nature*." – Bunnik et al. 2015

- Value of medical explanation for NPD
- Validation of experiences
- Enhanced understanding of self and family history

Inform educational and NPD support needs

Promote health decisions and treatment access to optimize NPD screening/care

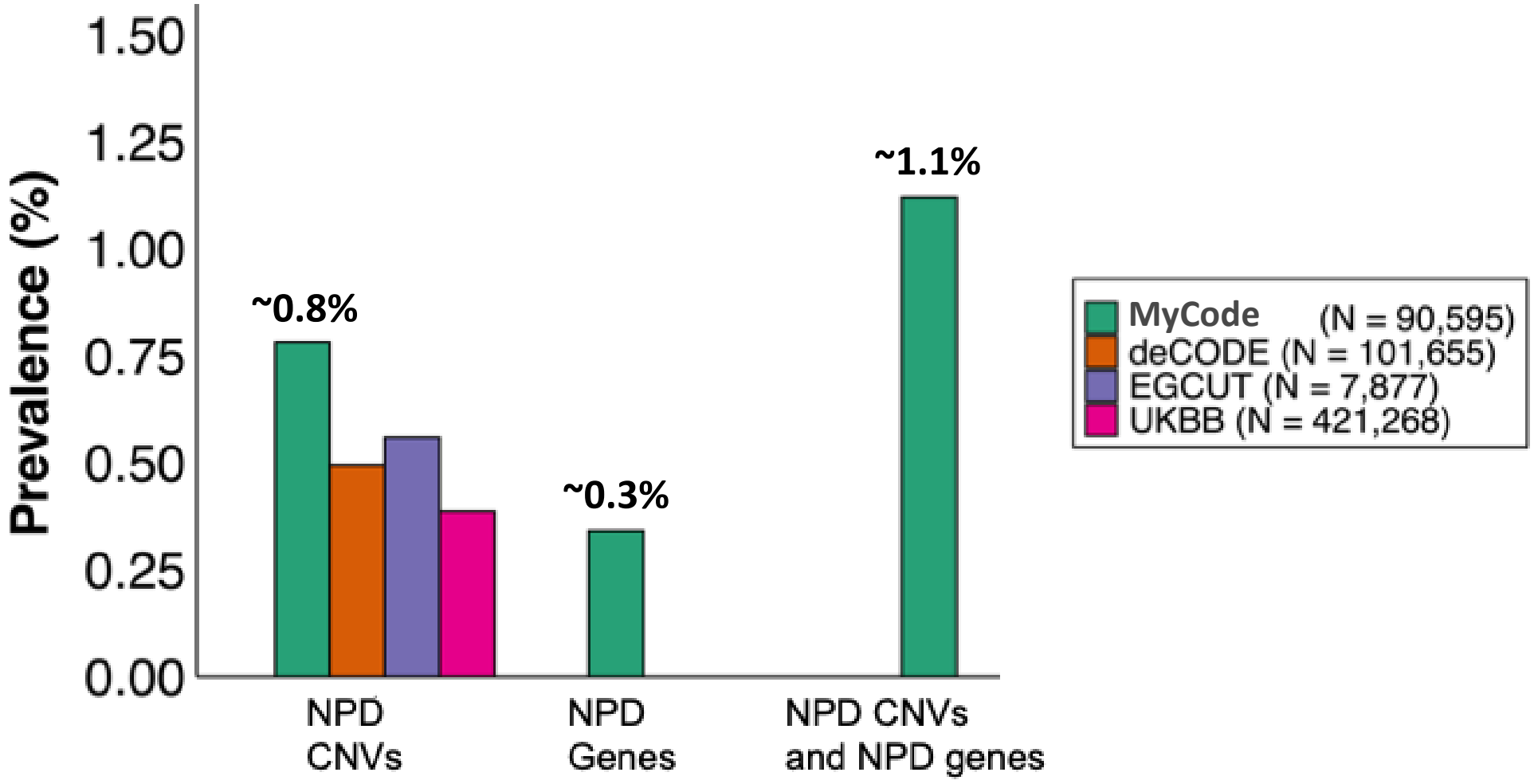
Screening for non-NPD health risks: hypocalcemia (22q11.2 del); renal cysts and diabetes (17q12 del)

Ability to test at risk family members (cascade testing)

NPD Precision Health: Key Take-Aways

- **Recurrent pathogenic NPD CNVs are prevalent (0.8%)**
  - The majority of adults with NPD CNVs have not received a genetic diagnosis
  - At least 1.1% when 94 single gene disorders are included

Prevalence of Pathogenic Variants in 31 NPD Recurrent CNVs and 94 NPD Genes in MyCode and Other Population-based cohorts



Shimelis and Oetjens et al., *in prep*

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- **NPD CNVs result in clinical symptoms (penetrance) at similar rates to other genomic disorders included in population health screening (35-70%)**

NPD Precision Health: Key Take-Aways

- **Recurrent pathogenic NPD CNVs are prevalent (0.8%)**
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  - At least 1.1% when single gene disorders are included

- **NPD CNVs result in clinical symptoms (penetrance) at similar rates to other genomic disorders included in population health screening (35-70%)**

- **Participants value receiving NPD CNV results and describe the experience as important and valuable**
  - Clinically and psychologically important – **"medicalizing" NPD**
  - May decrease stigma, increase self-advocacy, lead to closer engagement with healthcare providers, and improve outcomes

NPD Precision Health: Ongoing work…

- Now disclosing results to MyCode participants *without* NPD documentation in EHR – seeing similar trends
- Longitudinal follow up – 6-month surveys and interviews
- Exploring healthcare provider needs, patient support needs (navigator), and long-term impact on patient outcomes

Acknowledgements

Thank you to:

Our MyCode patient-participants, Geisinger Providers and Staff, and the MyCode Research Team

David Ledbetter, PhD and Karen Wain, MS, for co-leading these studies

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Funding from:
Geisinger Regeneron Pharmaceuticals
NIMH Martin/Ledbetter R01MH074090
NIMH/NICHD Martin/Ledbetter U01MH119705
NIMH Martin R01MH107431

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