Genetic Mosaicism and Clonal Hematopoiesis

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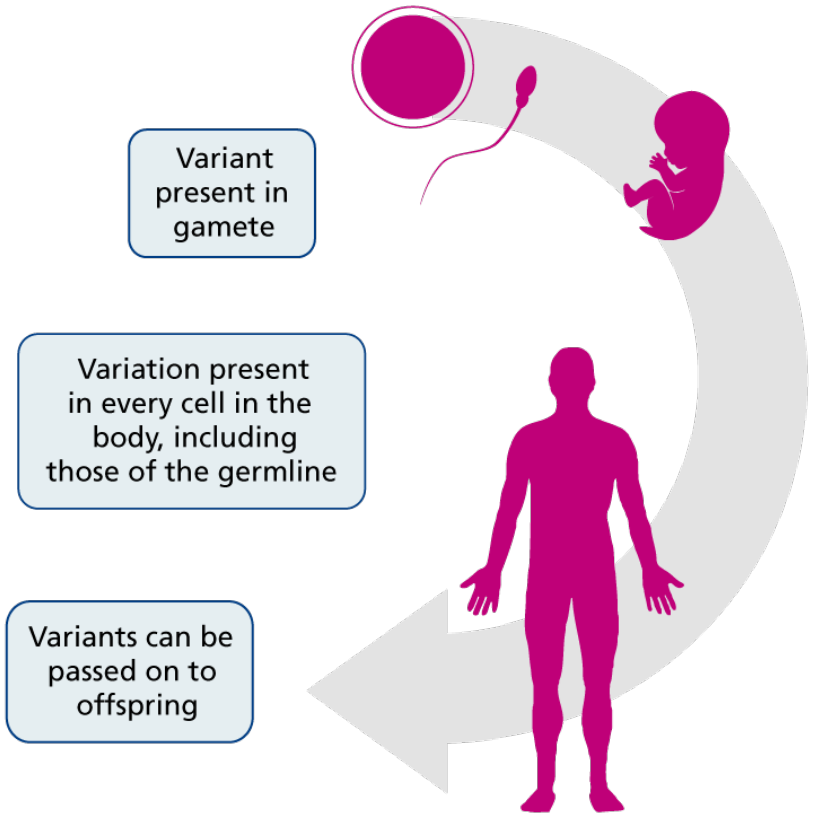
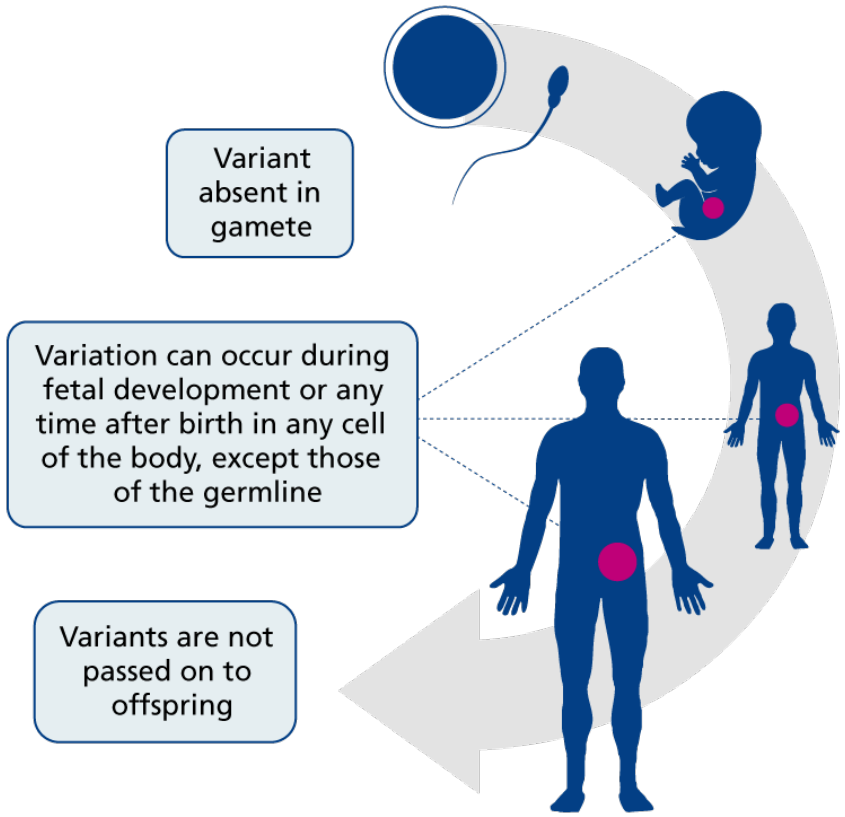
Types of Genetic Variation

Germline



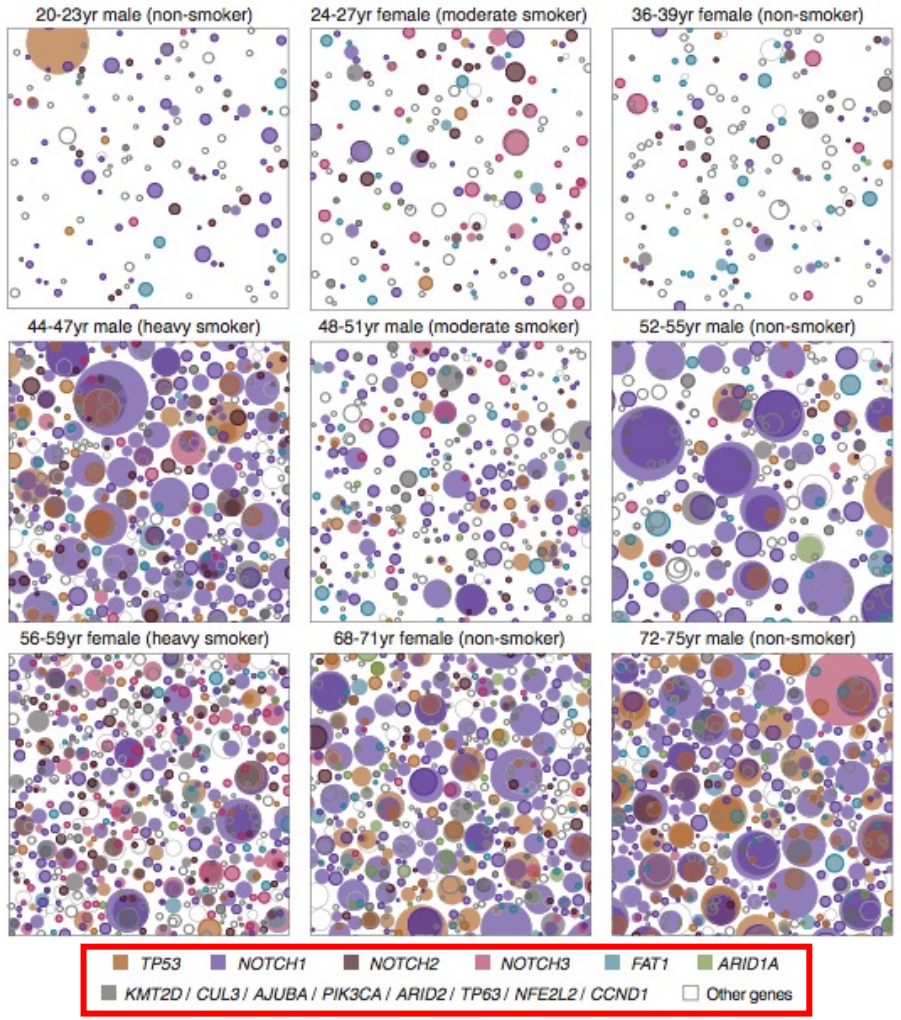
Somatic



Central Theme: Normal Tissues Harbor Mutations

- Improved sequencing depth/single cell technologies → better resolution for detection
- Mutations are abundant in normal tissues
  - most somatic cells harbor 100s of mutations
  - low frequency (0.0000001=1×10^{-7}) dependent on age, replication rate, environmental exposures
  - neurons lowest, sun exposed skin highest
- All mutations or clones do not progress to cancer (even mutations in cancer predisposition genes)
  - additional mutations/selective forces required for transformation
- Research Motivation: improve understanding of which clones confer elevated cancer risk and the factors promoting their clonal expansion

Normal Human Esophagus



Martincorena *Science* 2018

Clonal Hematopoiesis (CH)

- The acquisition of somatic mutation(s) in a clonal subset of hematopoietic progenitor cells that differs from the inherited germline genome

  Detectable CH requires two fundamental features:

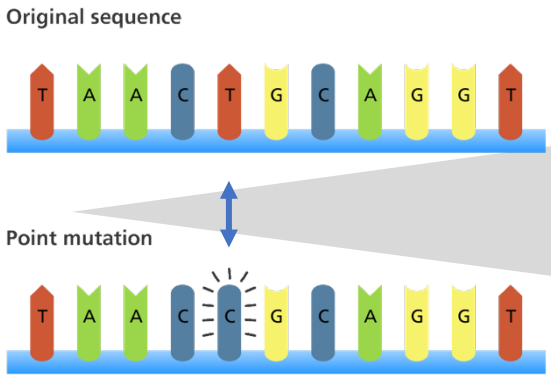
  1. the acquisition of a somatic mutation that goes unrepaired
  2. subsequent survival and clonal expansion transmitting acquired mutation on to daughter cells

➡➡ **Cancer**

Machiela *Curr Opin Genet Dev* 2017

Size Spectrum of Clonal Hematopoiesis (CH)

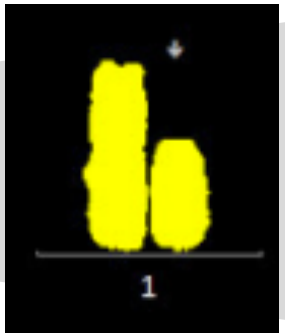
Clonal Hematopoiesis of Indeterminant Potential (CHIP)



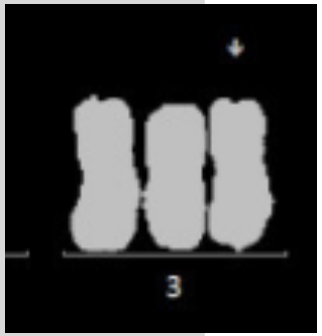
-Next generation sequencing (NGS)
-Deleterious impact on a single gene

Mosaic Chromosomal Alterations (mCAs)

Partial Chromosomal Events

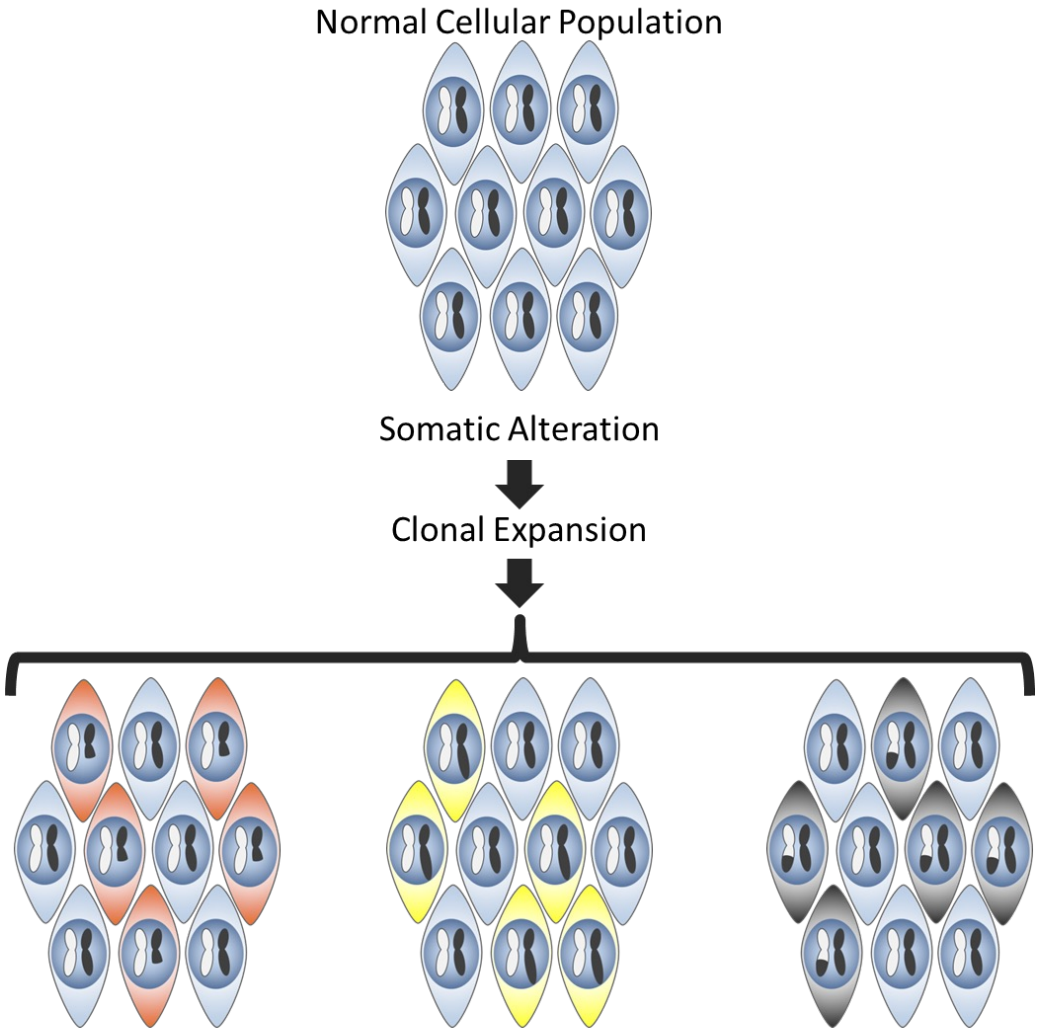


Whole Chromosome



-Commercially available genotyping arrays or NGS
-Can impact many genes simultaneously

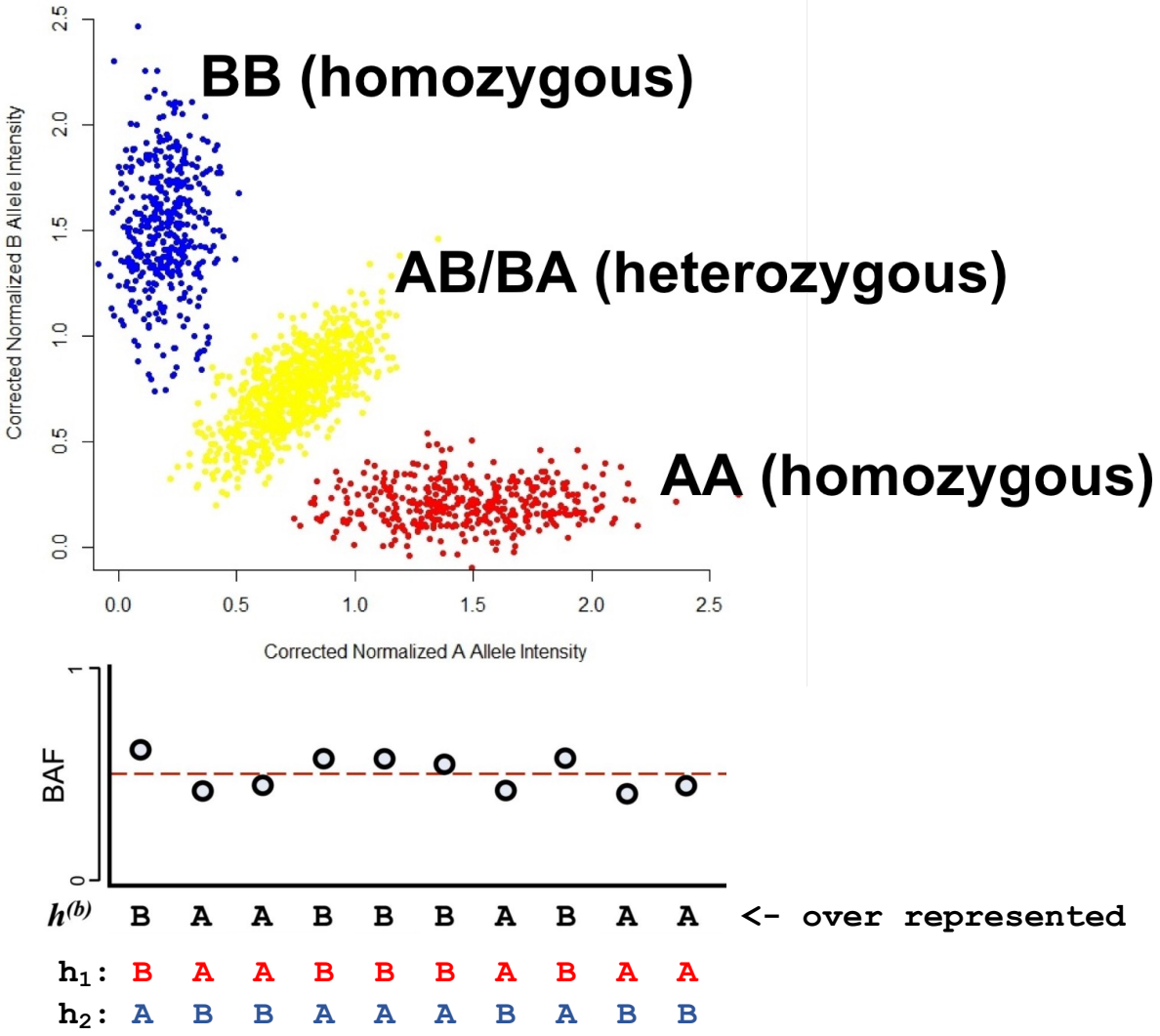
Types of Mosaic Chromosomal Alterations (mCAs)



Machiela *Semin Hematol* 2013

Detecting Mosaic Chromosomal Alterations (mCAs)





- B Allele Frequency (BAF)
  - allelic imbalances
- Log_2 R ratio (LRR)
  - abnormalities in intensity magnitude
- Haplotype phase
  - over-represented haplotypes

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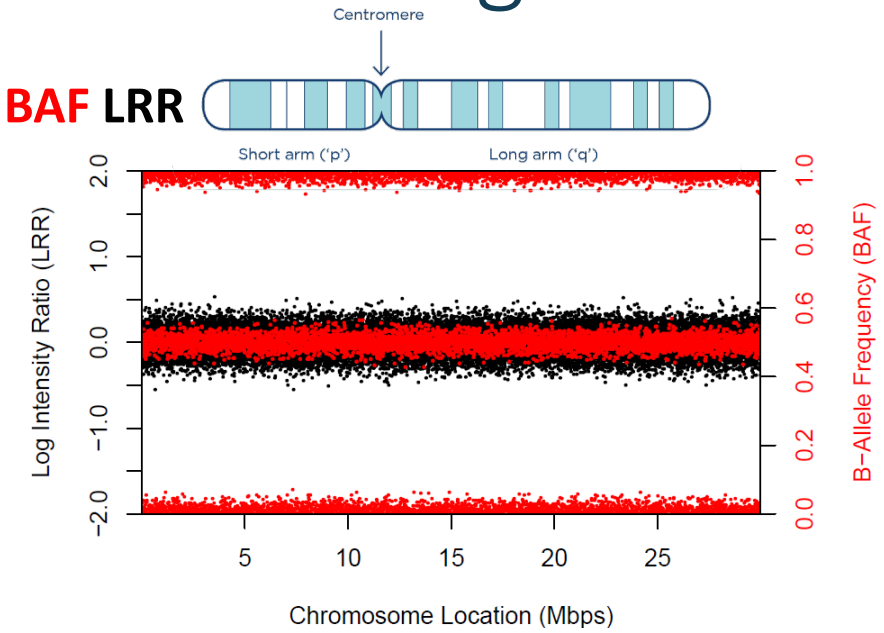
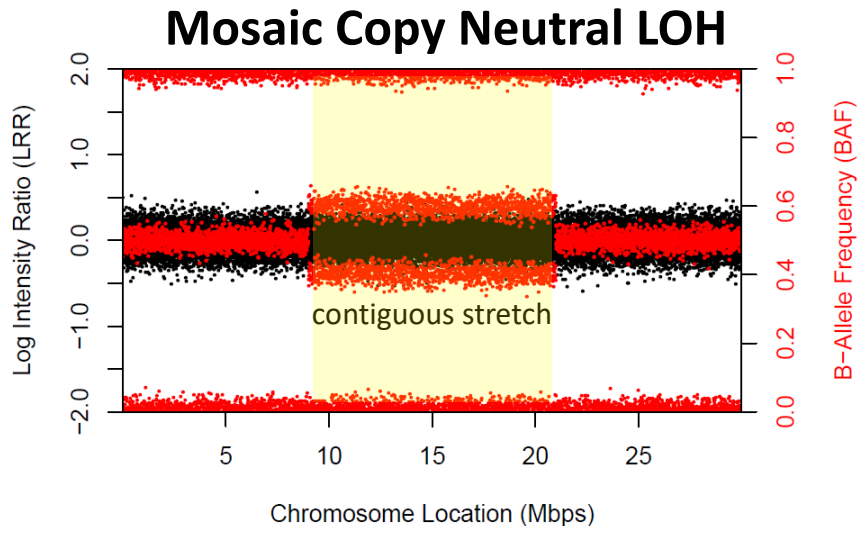
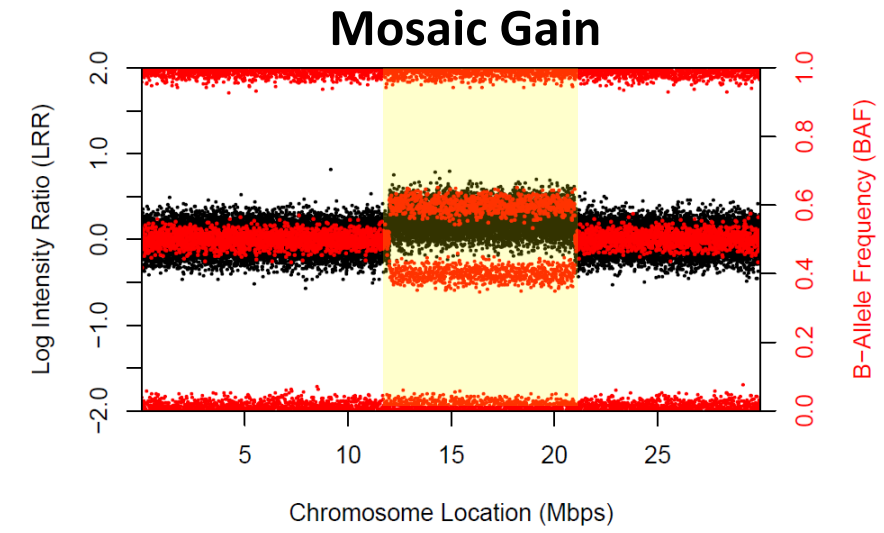
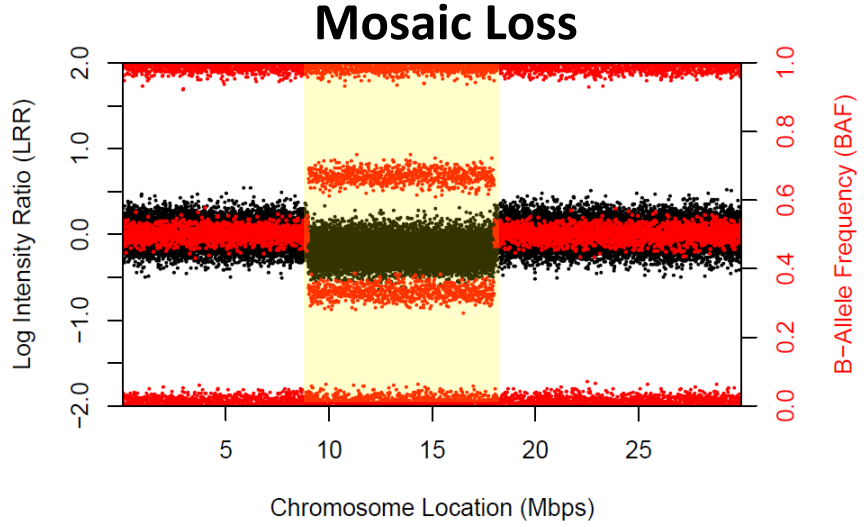
Rodríguez-Santiago *AJHG* 2010, Vattathil *AJHG* 2016, Loh *Nature 2019*

Detecting Mosaic Chromosomal Alterations (mCAs)

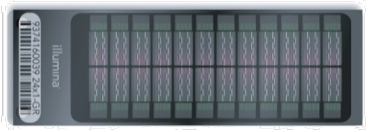






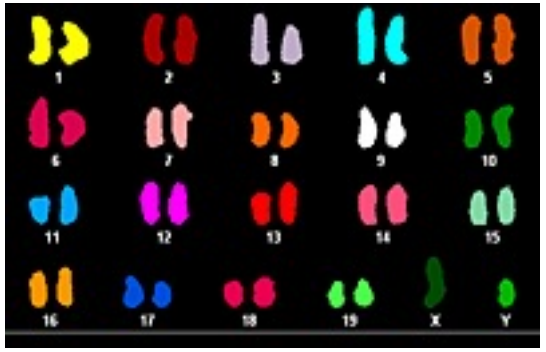


repurpose



MoChA

https://github.com/freeseek/mocha



Virtual Karyotypes

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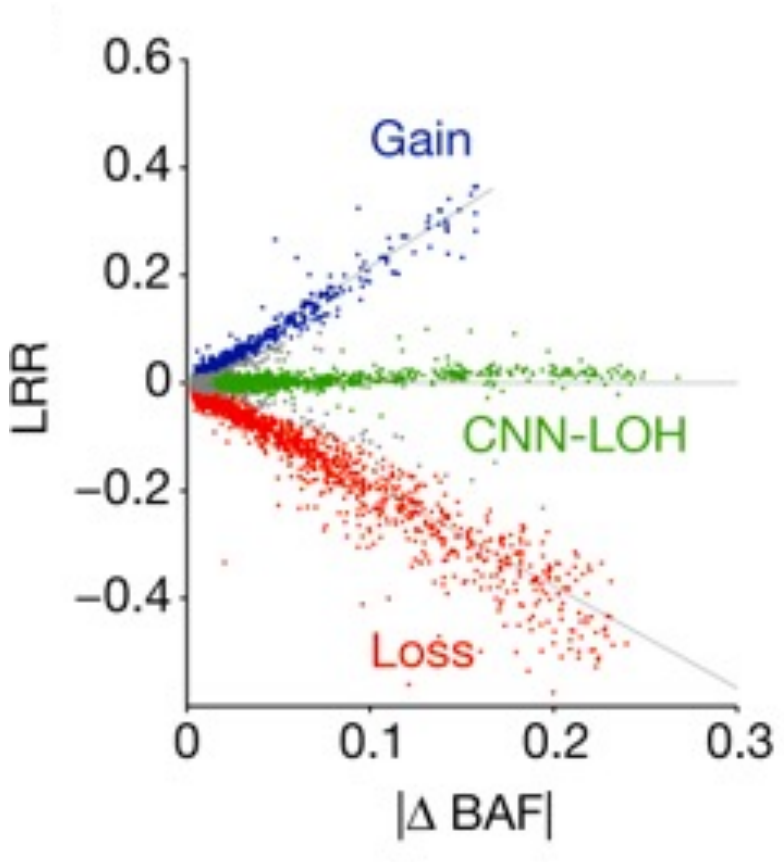
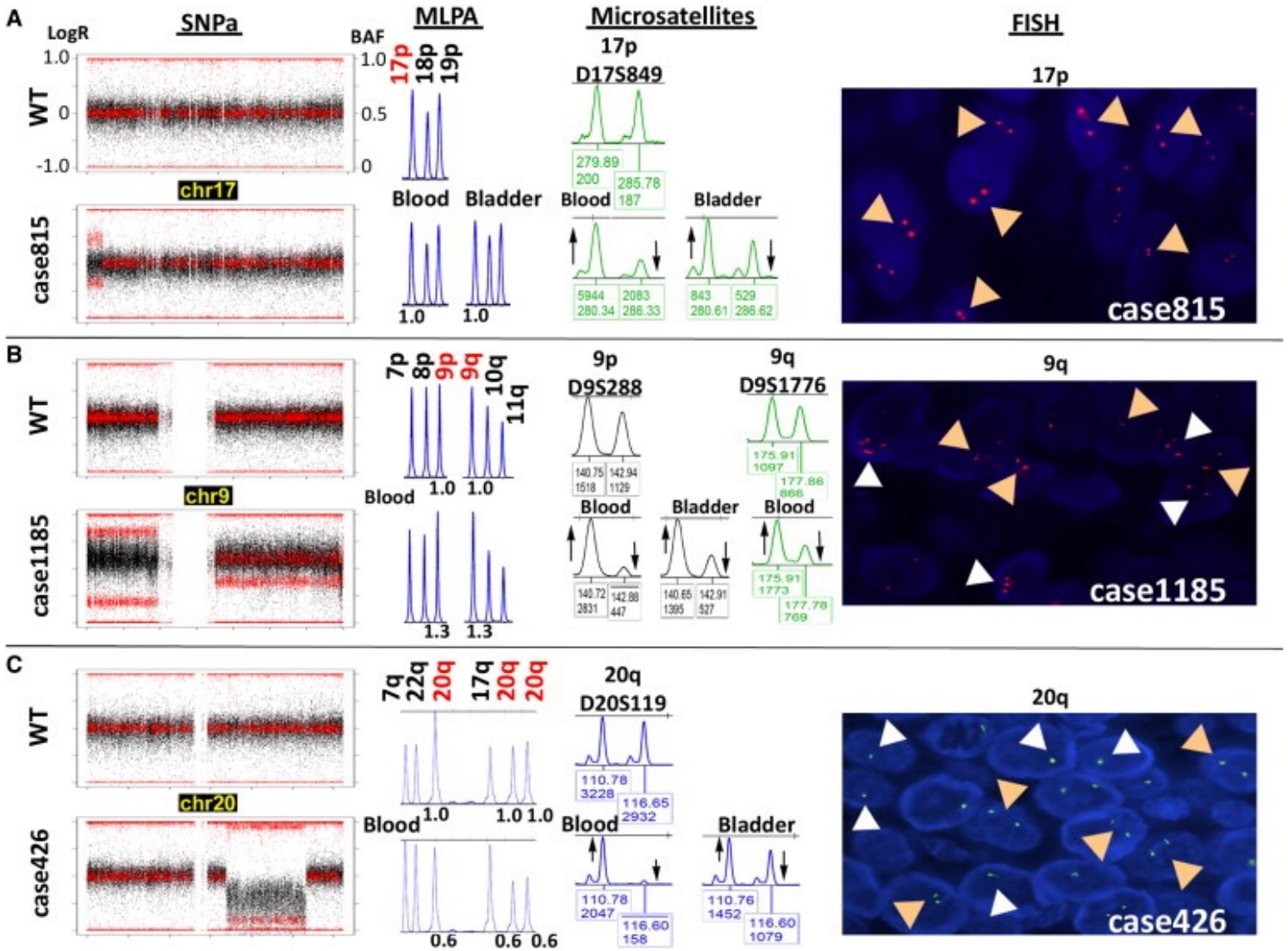
mCA Cell Fraction by Event Type



low **Percentage of cells affected** high

Loh *Nature 2019*

Experimental Validation



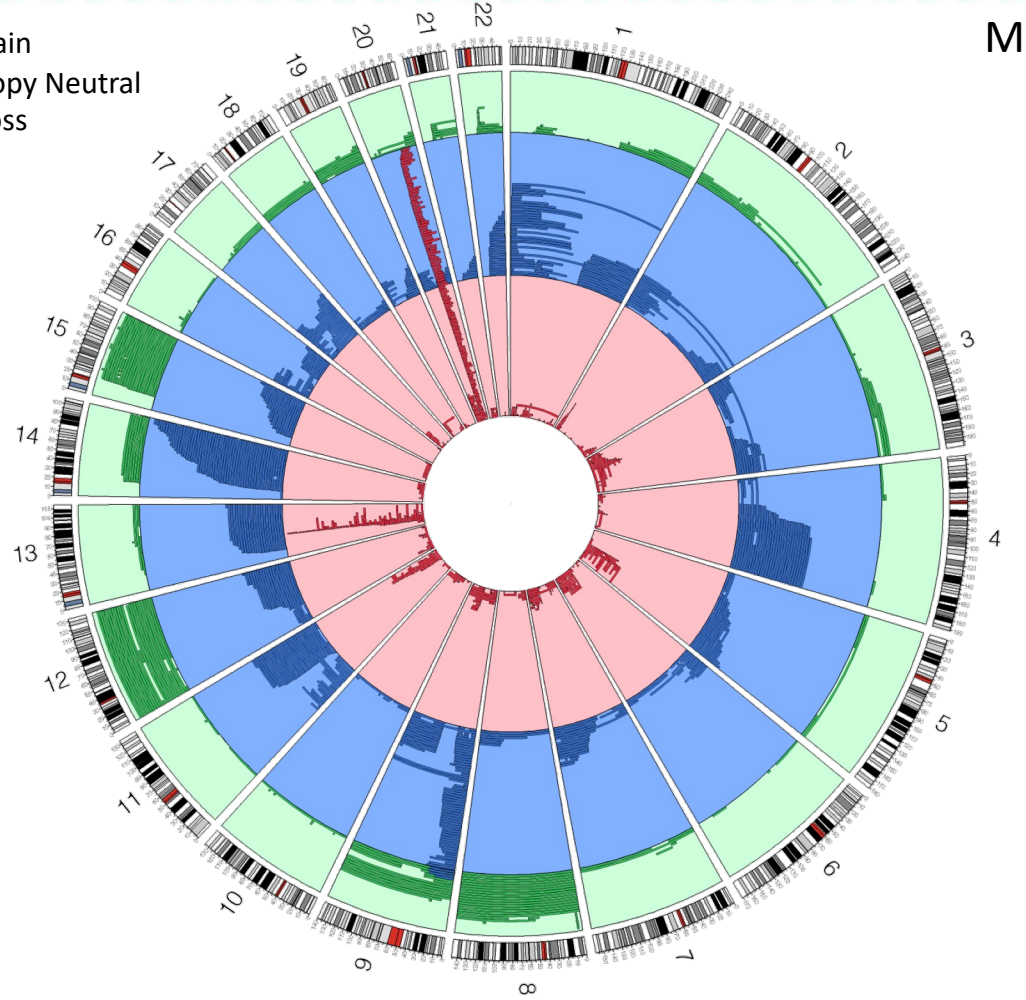
Rodríguez-Santiago *AJHG* 2010

Frequency and Distribution of mCAs

Autosomal mCAs (3-5%)



Machiela et al. *AJHG* 2015



Frequency and Distribution of mCAs

Autosomal mCAs (3-5%)



Machiela et al. *AJHG* 2015

Mosaic Y Loss (15%-20%+)



Zhou, et al. *Nat Genet* 2016

Female Mosaic X Loss (12%)



Liu et al. *Nature* 2024

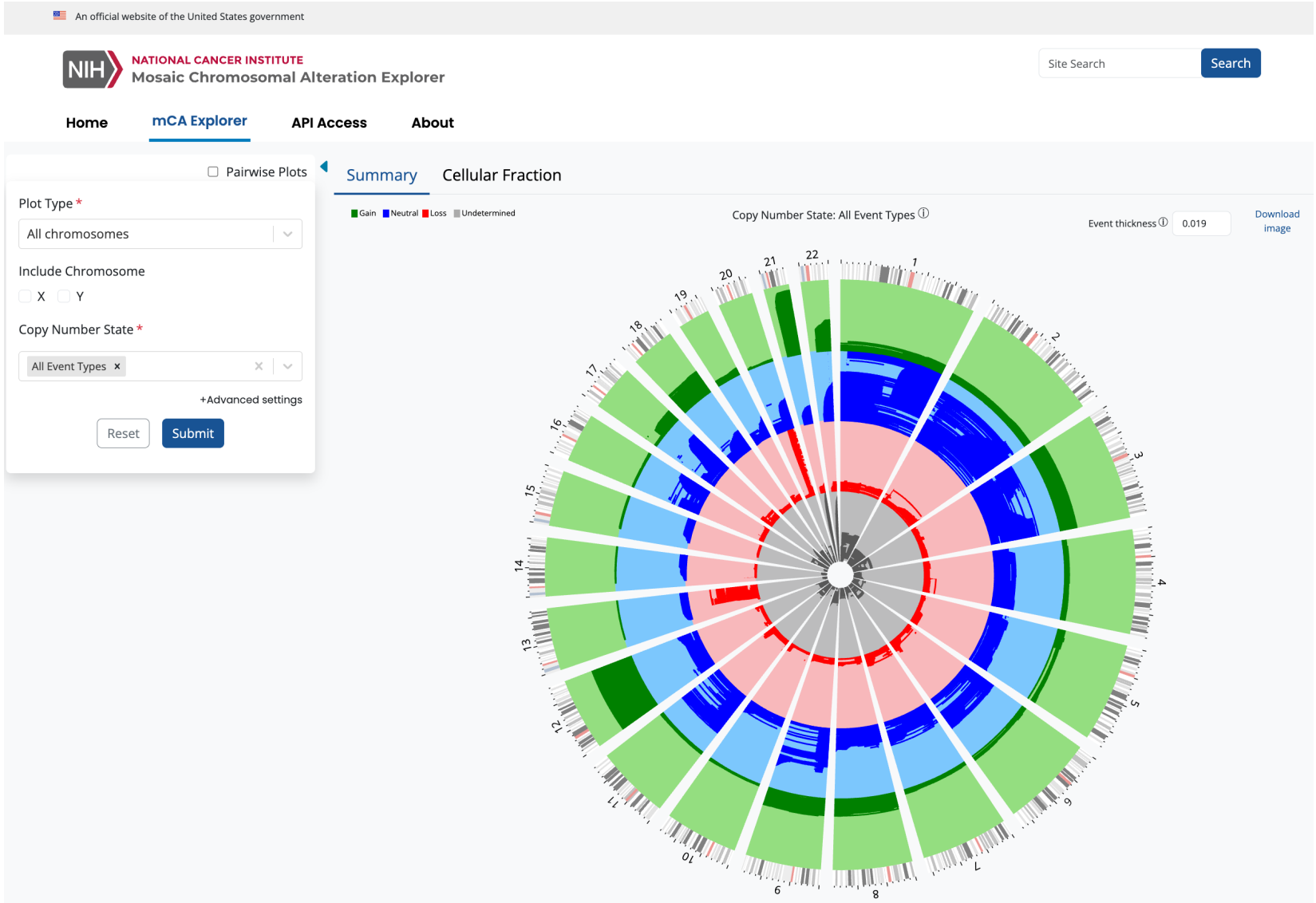
Male X Mosaicism (0.006%)



Zhou et al. *Sci Rep* 2021

mCAs are commonly detected in leukocytes of adult populations, but vary in frequency by the chromosome affected.

mCA Explorer: An interactive catalog of mCAs



A total of 1.1 M participants and 85,594 autosomal mCAs detected

https://mcaexplorer.cancer.gov/

Risk factors for mCAs

Ageing



Machiela et al. *Am J Hum Genet* 2015
Machiela et al. *Nat Commun* 2016
Loftfield et al. *Sci Rep* 2018

Ancestry



Machiela et al. *Am J Hum Genet* 2015
Jacubek et al. *Nat Genet* 2023

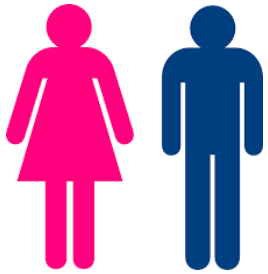
Smoking/Air Pollution





Zhou et al. *Nat Genet* 2016
Loftfield et al. *Sci Rep* 2018
Wong et al. *Environ Int* 2018

Sex



Machiela et al. *Am J Hum Genet* 2015
Zhou et al. *Nat Genet* 2016
Machiela et al. *Nat Commun* 2016

Geography



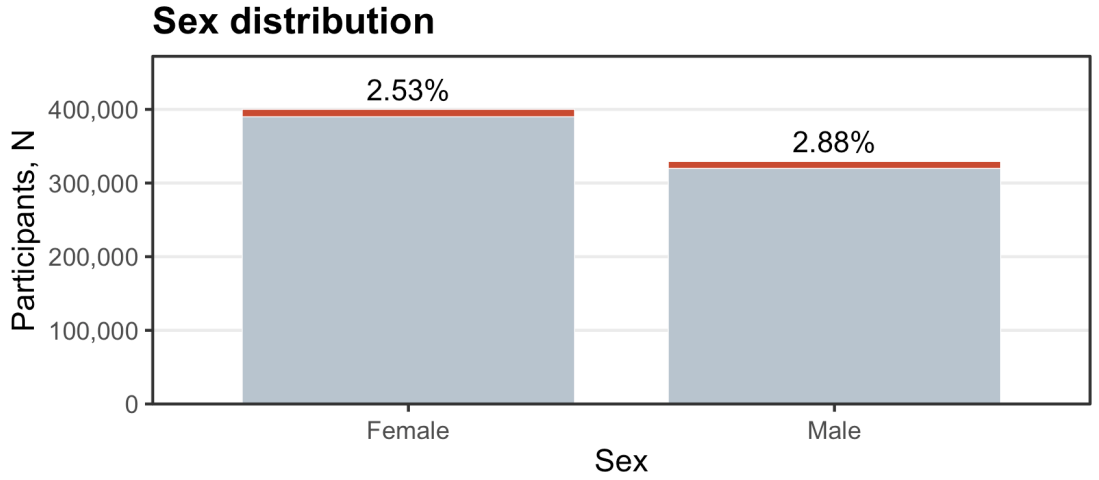
Zhou et al. *Nat Commun* 2023

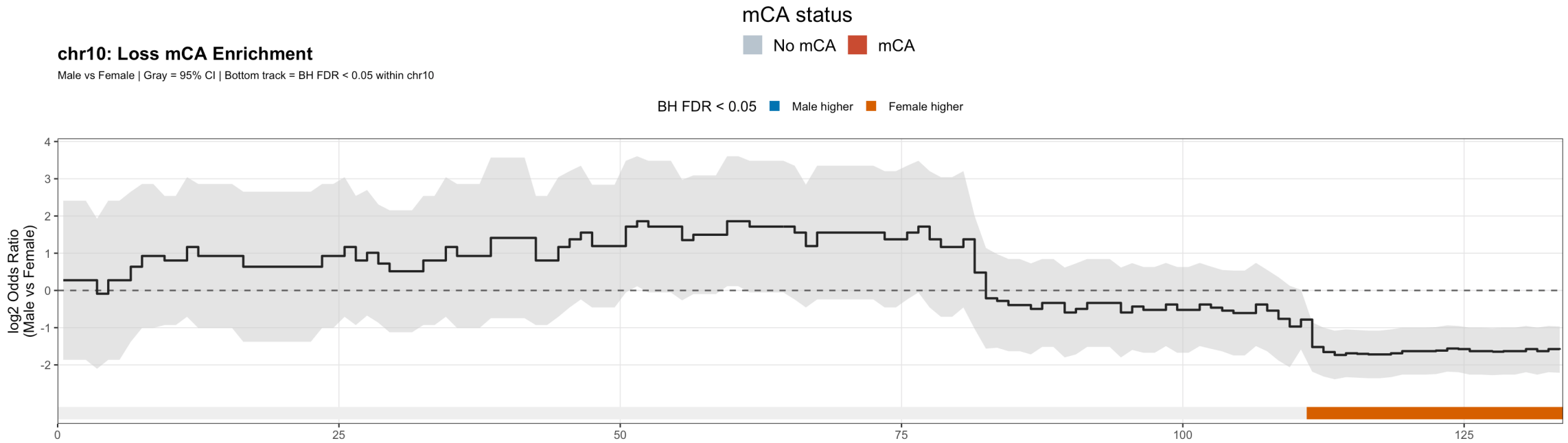
Glyphosate use



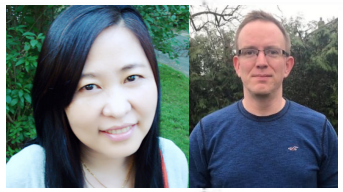
Chang et al. *EHP* 2023

Refining mCA Risk Factor Associations





Germline Variation and mLOY Susceptibility



UK Biobank, 23andMe, deCODE Genetics, Biobank Japan
($N_{discovery}$=205,011 , $N_{replication}$=757,114)

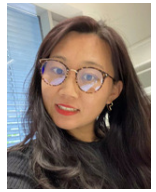
TCL1A (PLCO)

156 common susceptibility loci near genes relevant for cell-cycle regulation and cancer susceptibility

Inherited variation can predispose to the development of a somatic event

Zhou *Nat Genet* 2016, Wright *Nat Genet* 2017, Terao *Nat Commun* 2019, Thompson *Nature* 2019.

Germline Variation and mLOX Susceptibility

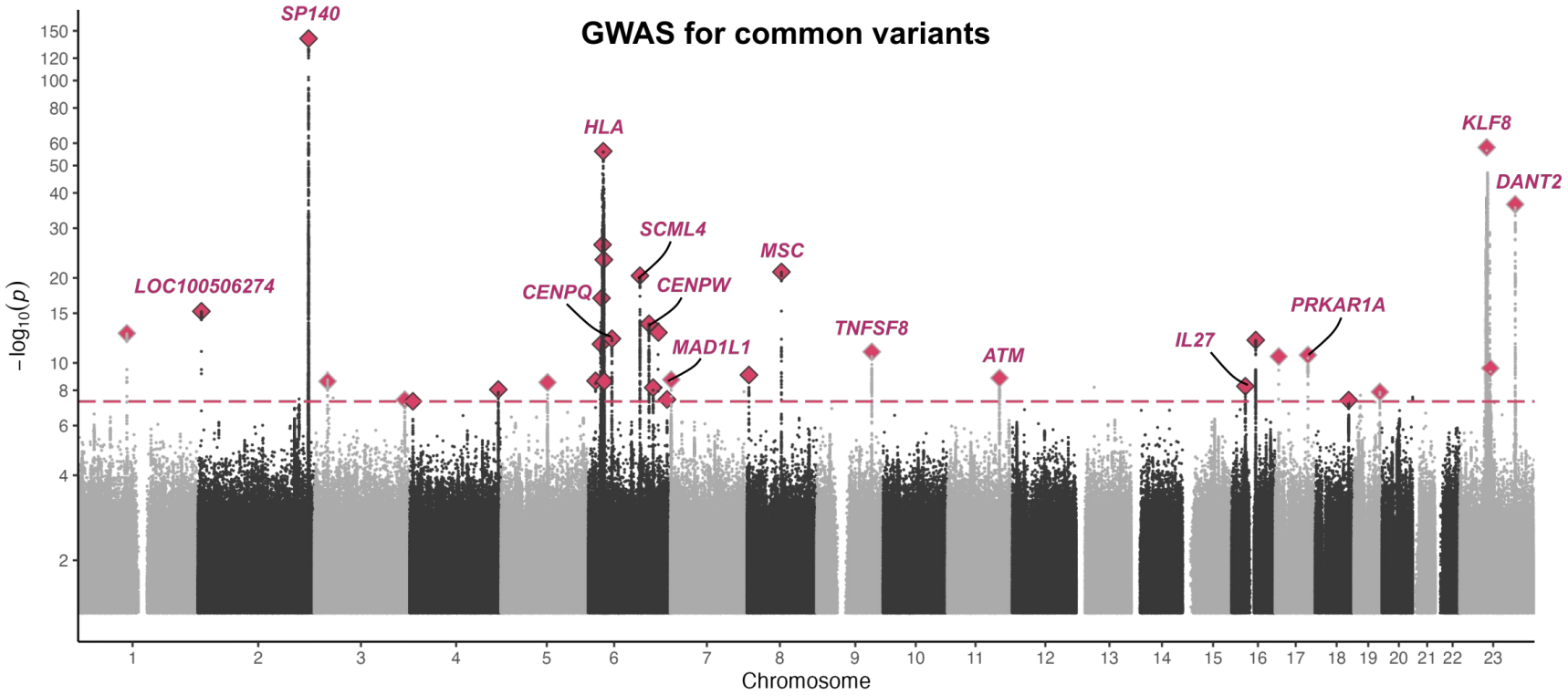


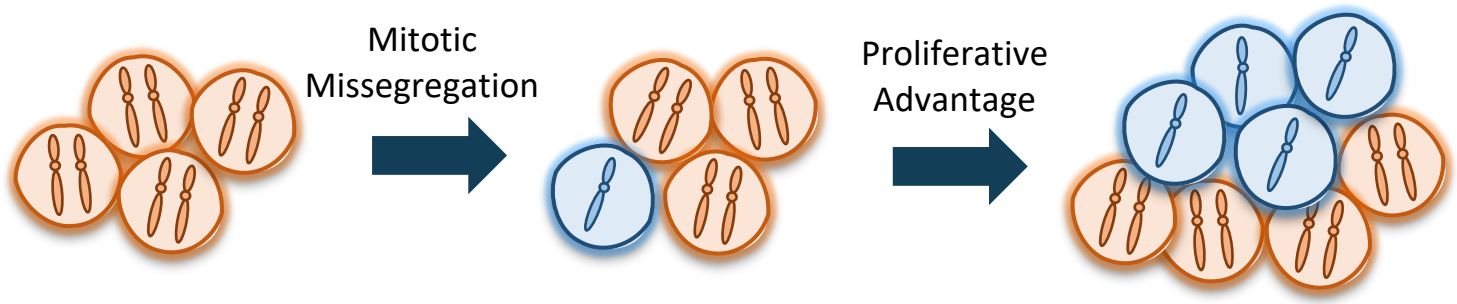
Biobank	Median age (SD)	mLOX Cases	Controls	Effective sample size	Continental ancestry groups
FinnGen	54 (18.2)	27,001	141,837	90,732	European, Finnish
Breast Cancer Association Consortium (BCAC)	57 (11.9)	21,966	155,356	76,980	European
Estonian Biobank (EBB)	44 (16.3)	20,232	110,547	68,408	European, Estonians
UK Biobank (UKBB)	57 (8.0)	16,214	244,931	60,829	European, British
Biobank Japan (BBJ)	65 (15.8)	13,597	63,720	44,823	East Asian, Japanese
Million Veteran Program (MVP)	54 (13.9)	1,496	33,192	5,726	European
Mass General Brigham Biobank (MGB)	54 (17.3)	2,108	11,527	7,128	European
Prostate, Lung, Colorectal and Ovarian Cancer Screening Trial (PLCO)	64 (5.4)	2,672	17,178	9,249	European

Blood-derived DNA from 883,574 females enrolled in eight biobanks worldwide

Liu *Nature* 2024

Germline Variation and mLOX Susceptibility





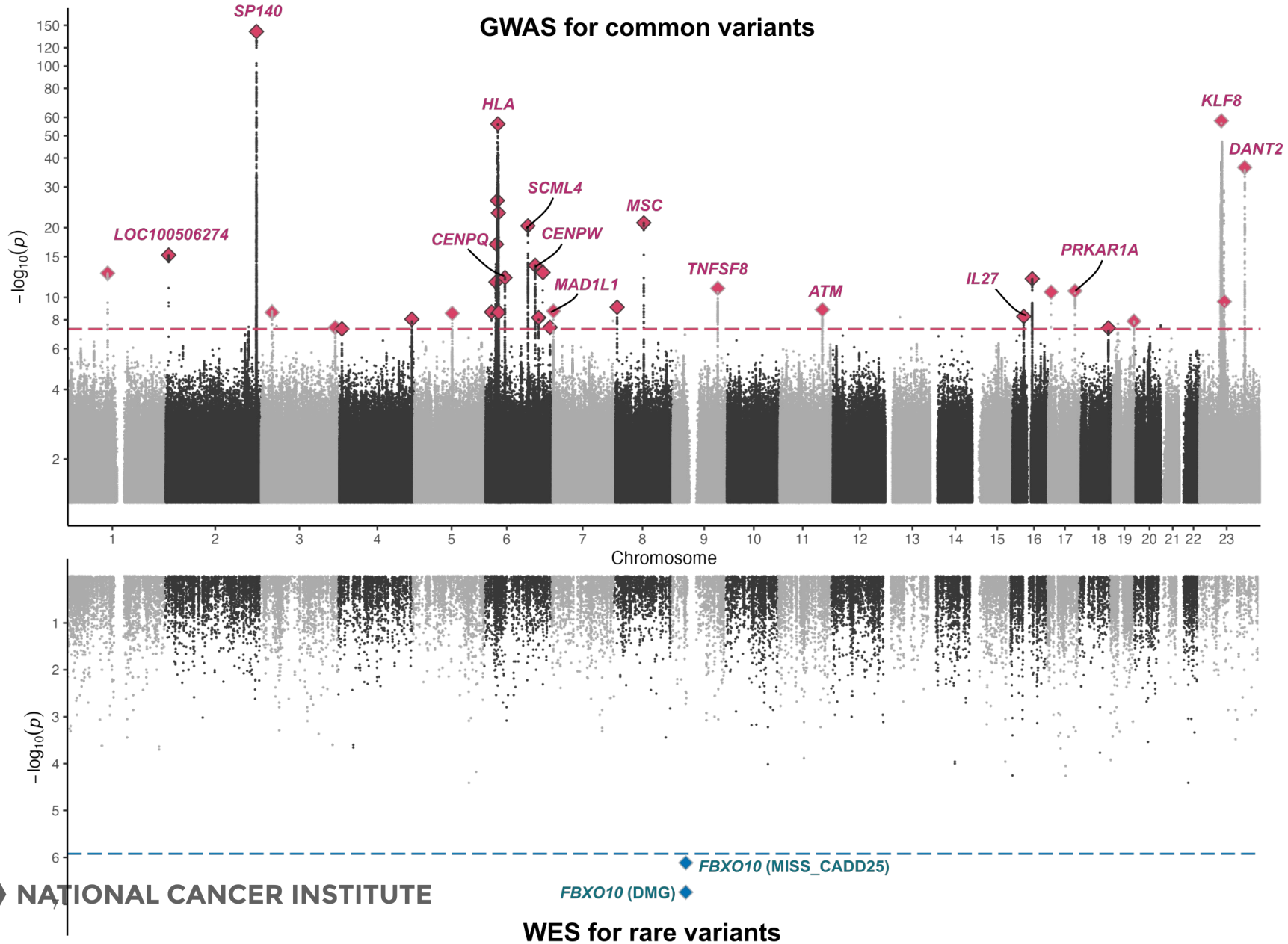
GWAS: 56 independent genome-wide significant variants ($P<5.0x10^{-8}$) across 42 loci

-strongest hit on chr 2 (*SP140*)
-clustering near HLA
-multiple hits on the X chromosome

<u>Nearby genes</u>:
-mitotic spindle assembly and kinetochore function
-blood cell traits
-immunity/autoimmunity
-cancer predisposition

Liu *Nature* 2024

Rare variation is associated with mLOX



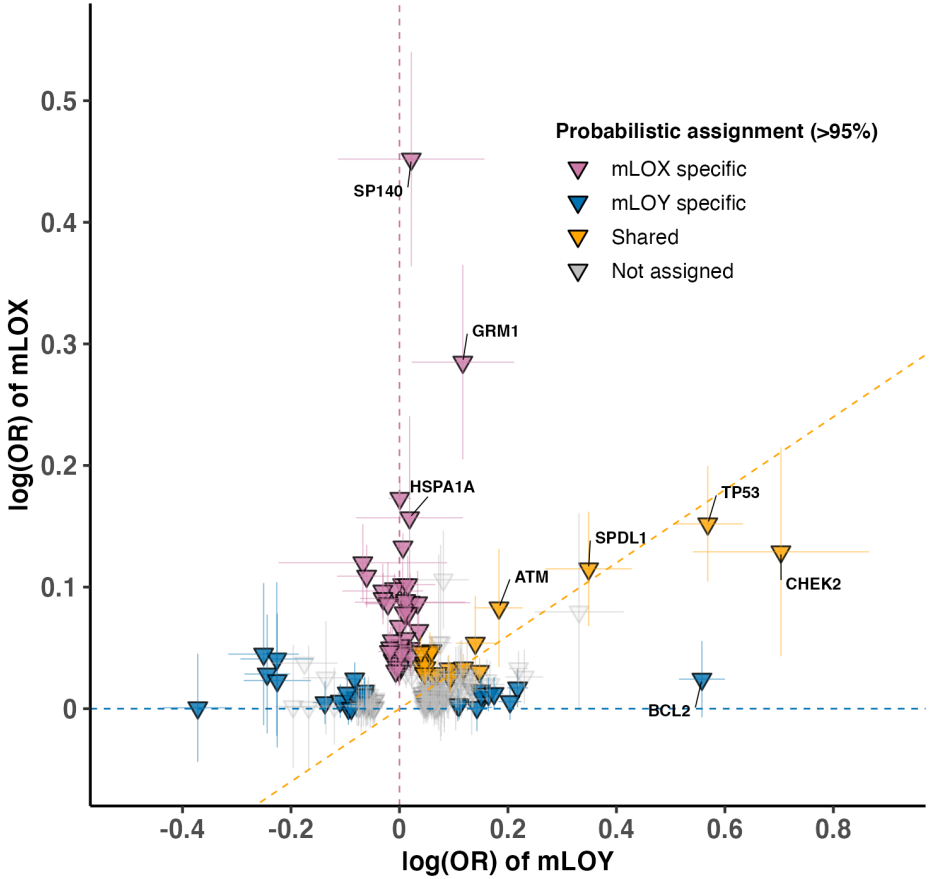
WES: rare missense variants of *FBXO10*
-ubiquitinates BCL2, apoptosis

Liu *Nature* 2024

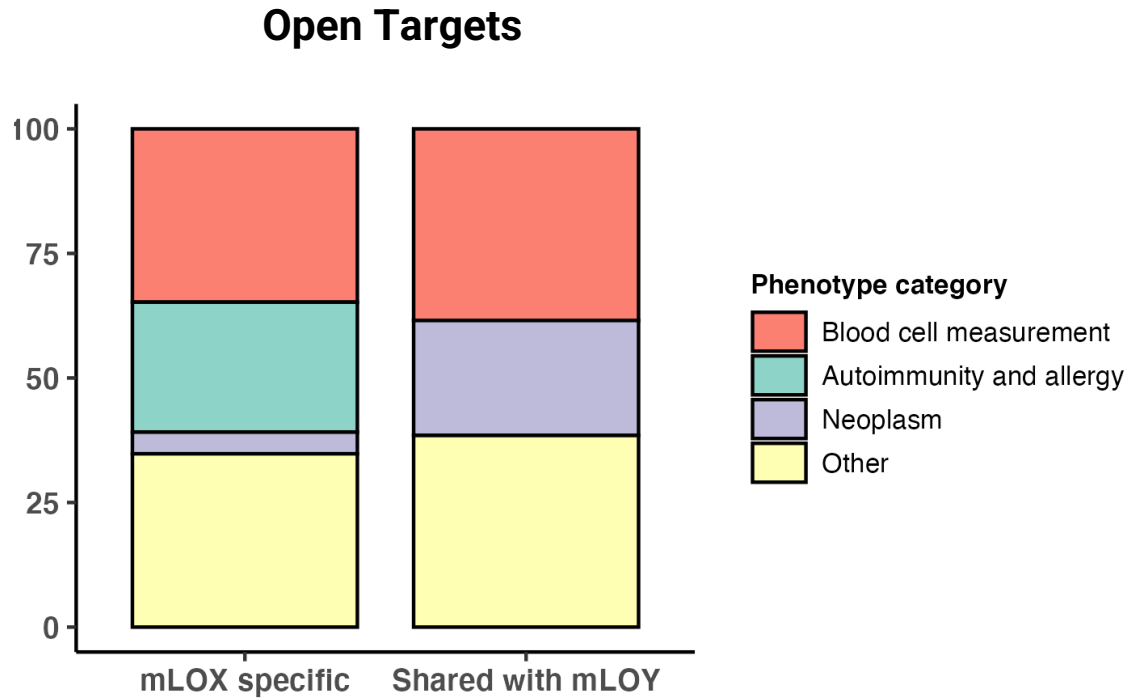
Comparison of mLOX genetic architecture with mLOY

➢ **Genetic correlation between mLOX and mLOY**

0.30 (LDSC, 95% CI=0.21-0.39, P=1.7x10^{-10})



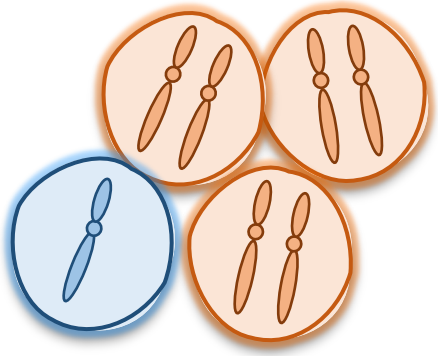
Out of 56 variants identified from mLOX GWAS, **34 variants were specific for mLOX**, **21 as shared with mLOY**, the remainder unassigned



- Both groups are associated with **blood cell phenotypes**
- For **autoimmunity**, associations were observed for mLOX specific variants but not for variants shared with mLOY
- For **neoplasm**, more associations were observed for variants shared with mLOY

Liu *Nature* 2024

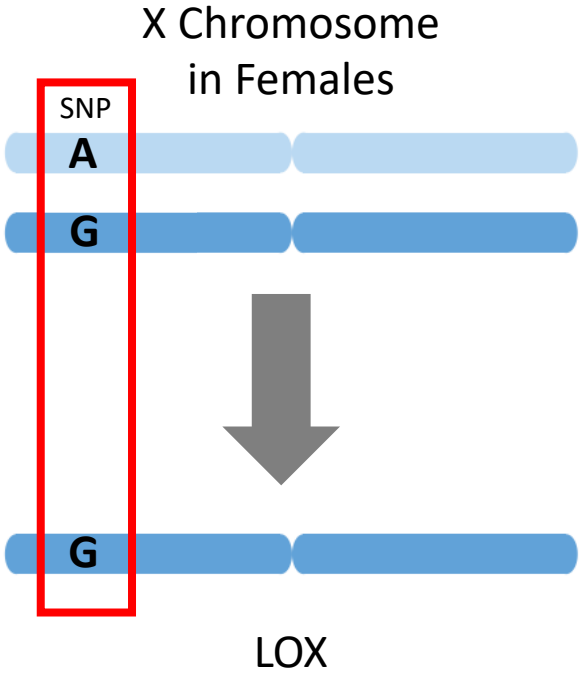
Utility of chr X germline variation to predict which X is lost/retained in mLOX



Allelic Shift Analysis

-similar to a transmission disequilibrium test (TDT) in family-based studies

-only heterozygous variants are informative



Chromosome X alleles nominate loci that could contribute to LOX selection

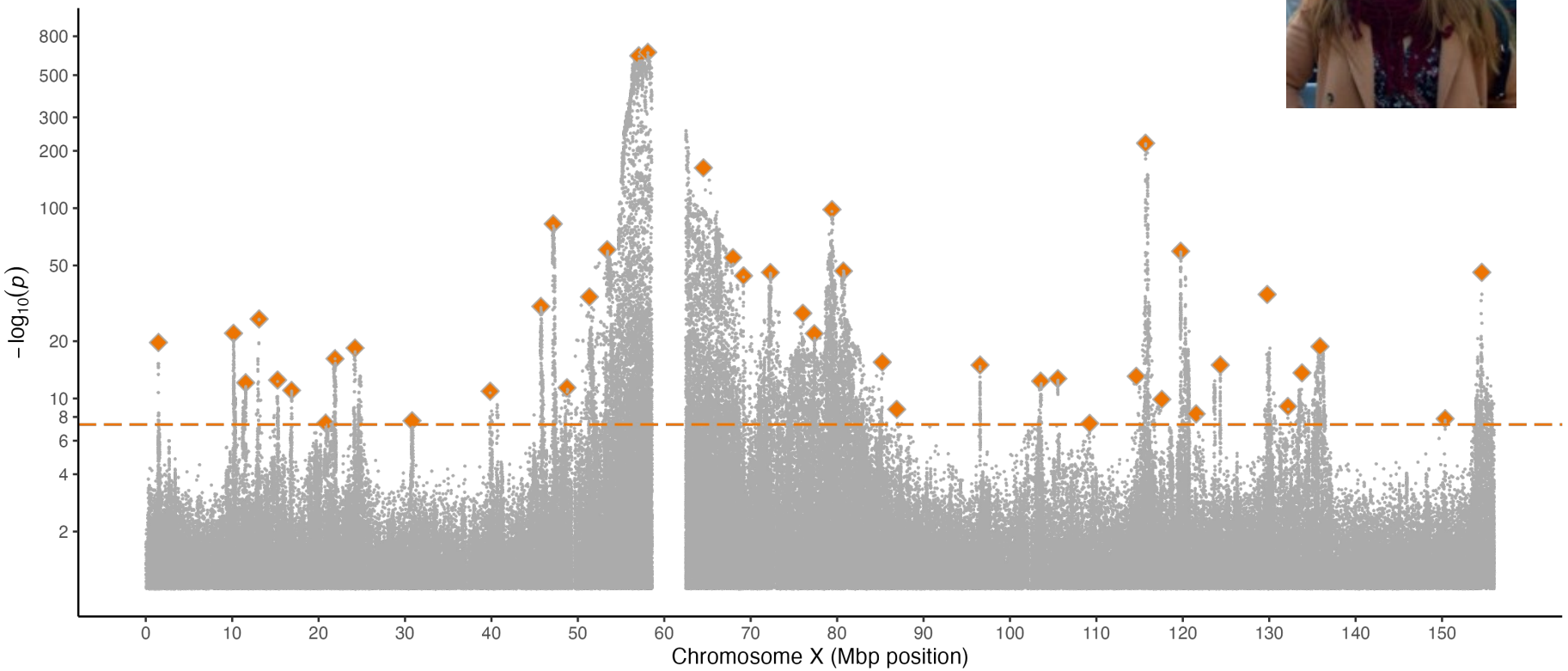
Allelic Shift:
-44 independent X chromosome loci

-loci located near genes relevant for **skewed X inactivation**, **cancer susceptibility** and **blood cell counts**

<u>Mechanism</u>?
(1) Centromere variation

(2) In the haploid state, these X loci could promote altered gene expression that confers a selective advantage

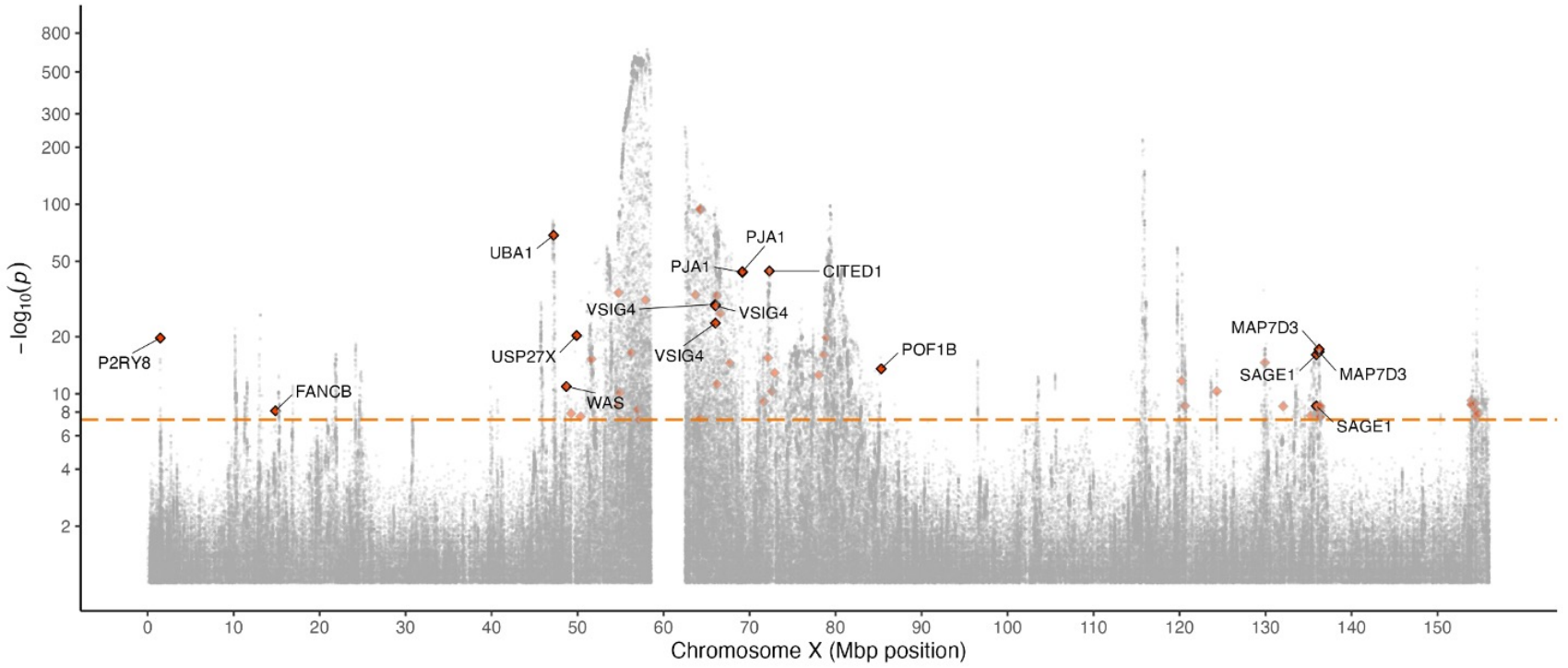




Liu *Nature* 2024

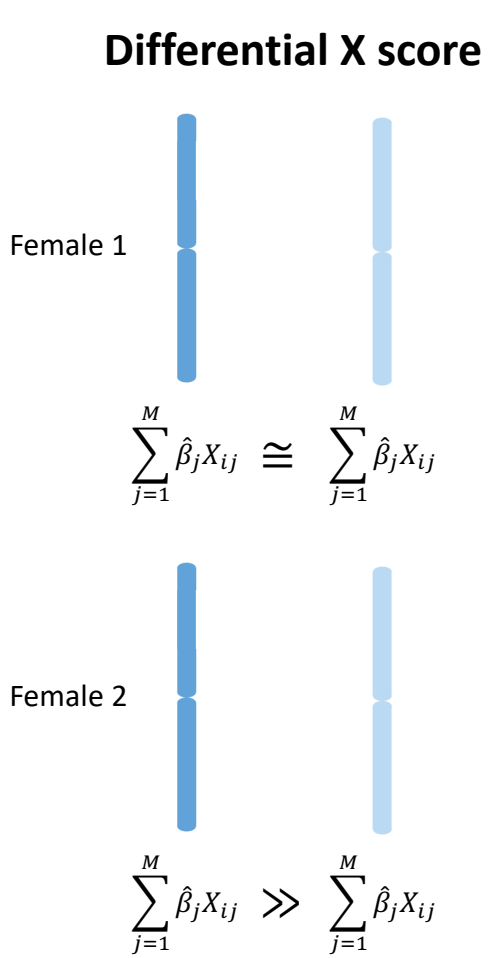
Several missense variants are near mLOX allelic shift loci

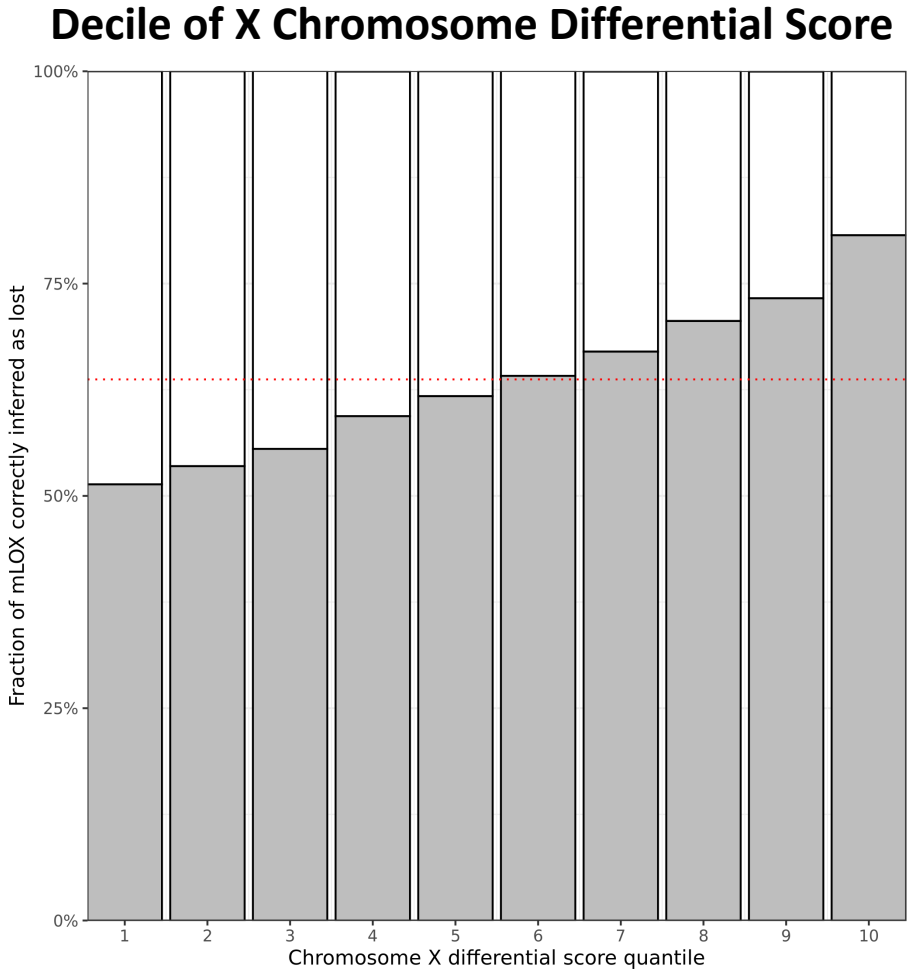
Mechanism?
(3) Chromosome X alleles might tag relevant missense variants that promote mLOX selection in the haploid state



Liu *Nature* 2024

Differential score of 44 allelic shift loci predict which X chromosome is retained in mLOX cases



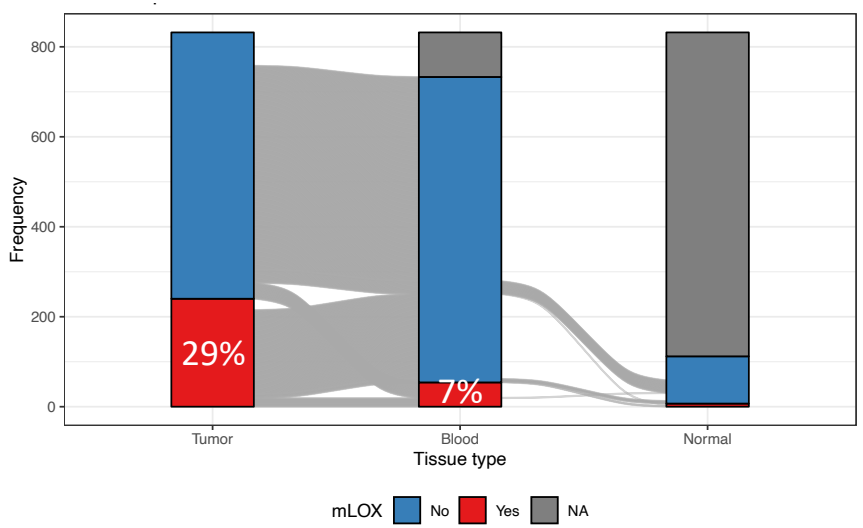


Liu *Nature* 2024

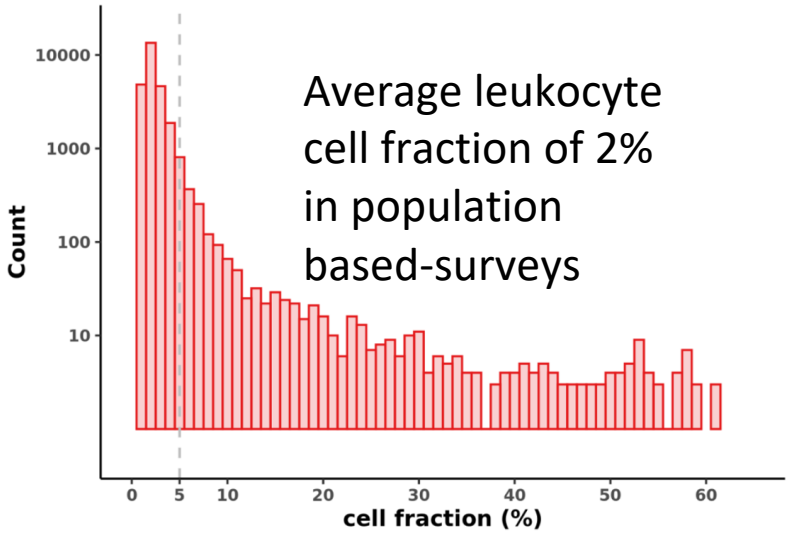
Utility of mLOX Susceptibility loci for LOX in Solid Tumors



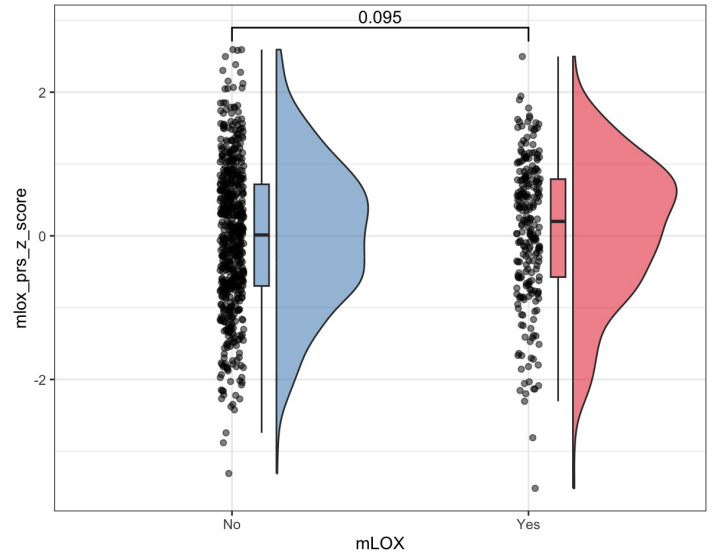
TCGA BRCA

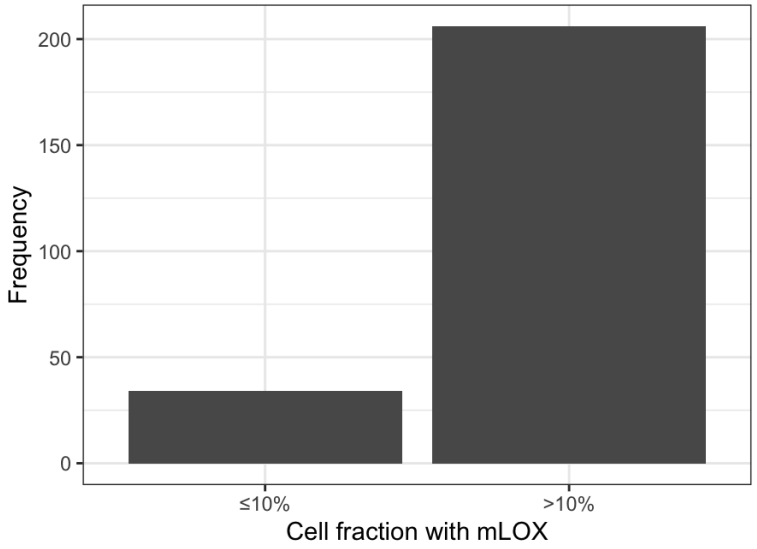


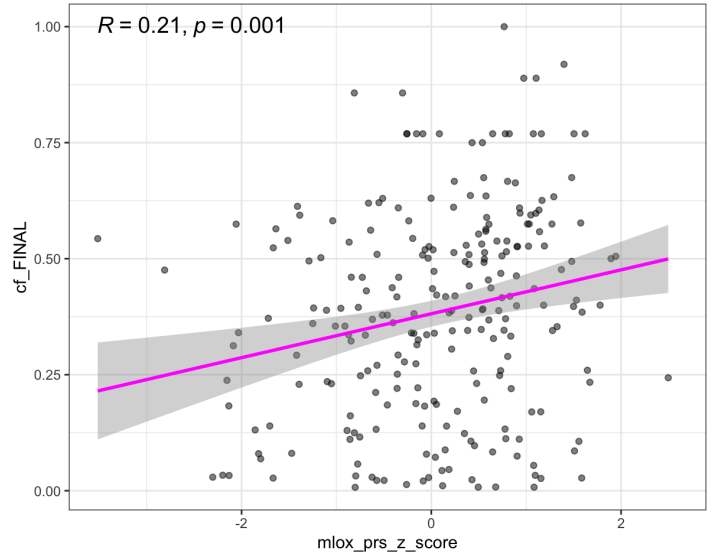
240 LOX cases of 832 BCa cases evaluated



Average leukocyte cell fraction of 2% in population based-surveys







Characterization of mCA clonal expansion

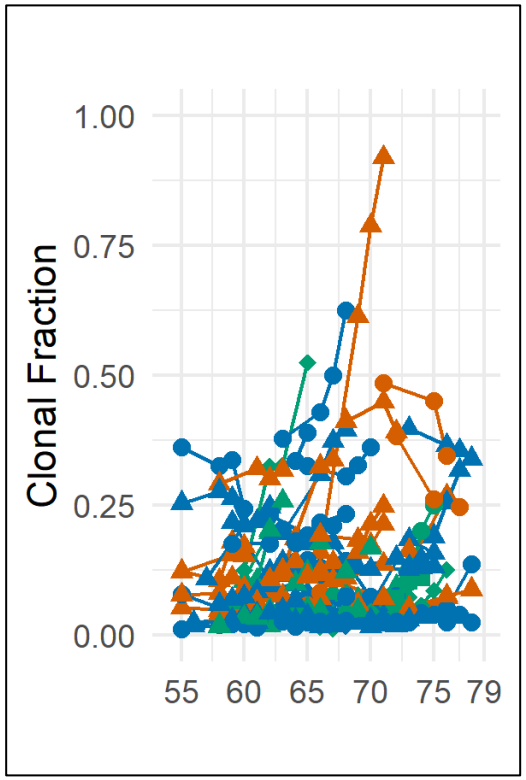


Characteristic	PLCO Individuals Scanned for mCAs (N = 56,324)	PLCO Individuals with Cross Sectional mCAs (N = 10,747)	PLCO Individuals with Longitudinal Assessment (N = 1,490)
Age at Genotyping (mean(SD))	65.30 (5.35)	67.17 (5.43)	64.3 (5.6)
Sex (column %)			
Female	28,042 (49.8)	4,512 (42.0)	469 (31.5)
Male	28,282 (50.2)	6,235 (58.0)	1,021 (68.5)
Smoking Status (column %)			
Never	26,476 (47.0)	4,722 (43.9)	605 (40.4)
Current	5,480 (9.7)	1,287 (12.0)	181 (12.1)
Former	24,122 (42.8)	4,702 (43.8)	712 (47.5)
Unknown	246 (0.4)	36 (0.3)	0 (0.0)
BMI (column %)			
<25	18,376 (32.6)	3,590 (33.4)	481 (32.1)
25-30	23,613 (41.9)	4,720 (43.9)	720 (48.1)
30-35	9,570 (17.0)	1,683 (15.7)	213 (14.2)
>35	4,752 (8.4)	753 (7.0)	73 (4.9)
Unknown	13 (0.0)	1 (0.0)	11 (0.7)
Individuals with mCA Type* (column %)			
Autosomal	-	2,179 (20.3)	671 (45.0)
mLOX	-	3,559 (33.1)	213 (14.3)
mLOY	-	5,355 (50.0)	834 (56.0)
X Chromosome mCA	-	131 (1.2)	42 (2.8)
Y Gain	-	32 (0.3)	10 (0.7)
Individuals with Multiple mCAs (column %)	-	676 (6.3)	467 (31.3)

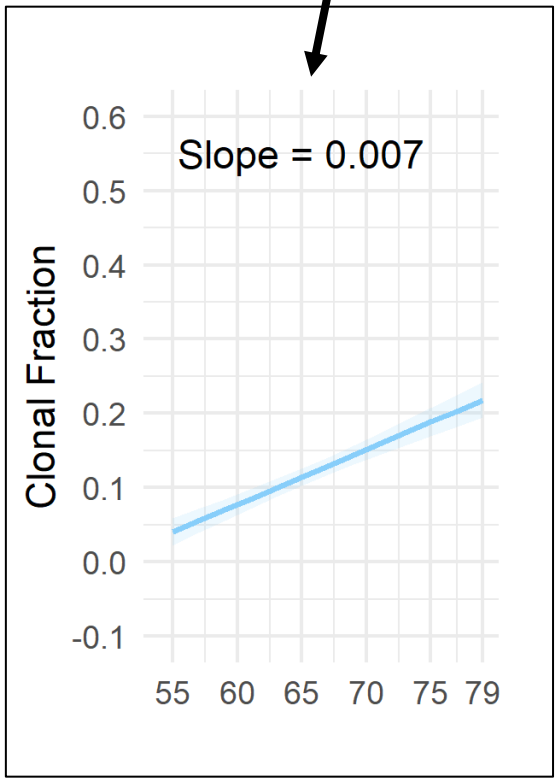
Kelly *Nat Commun* 2026

Clonal Expansion of mCAs in Longitudinal Samples

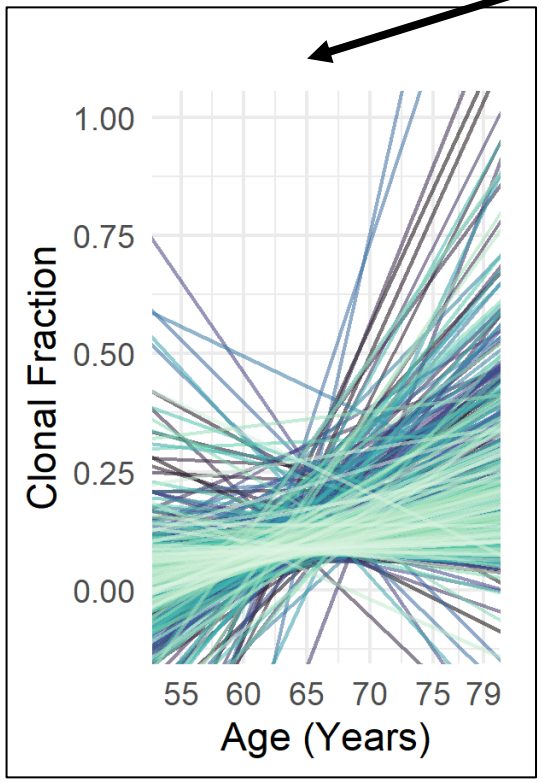
$$Y_{ijc} = \beta_0 + \beta_1 t_{ij} + \beta_2\ var\ (+ \beta_3 (t_{ij} * var) + b_{i0} + b_{i1} t_{ij} + b_{ci} + \varepsilon_{ijc}$$



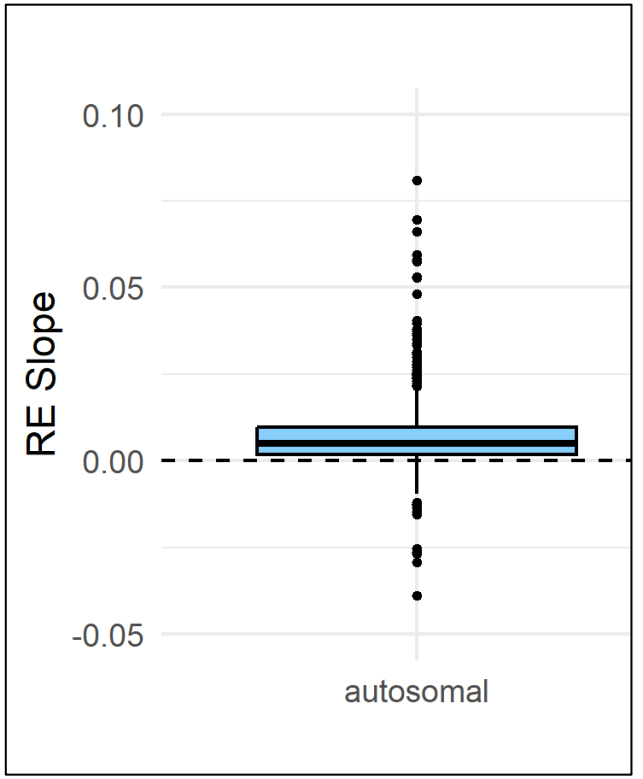
Raw Data



Population Average
(Fixed Effect)



Individual Profiles
(Random Effect)



Variation

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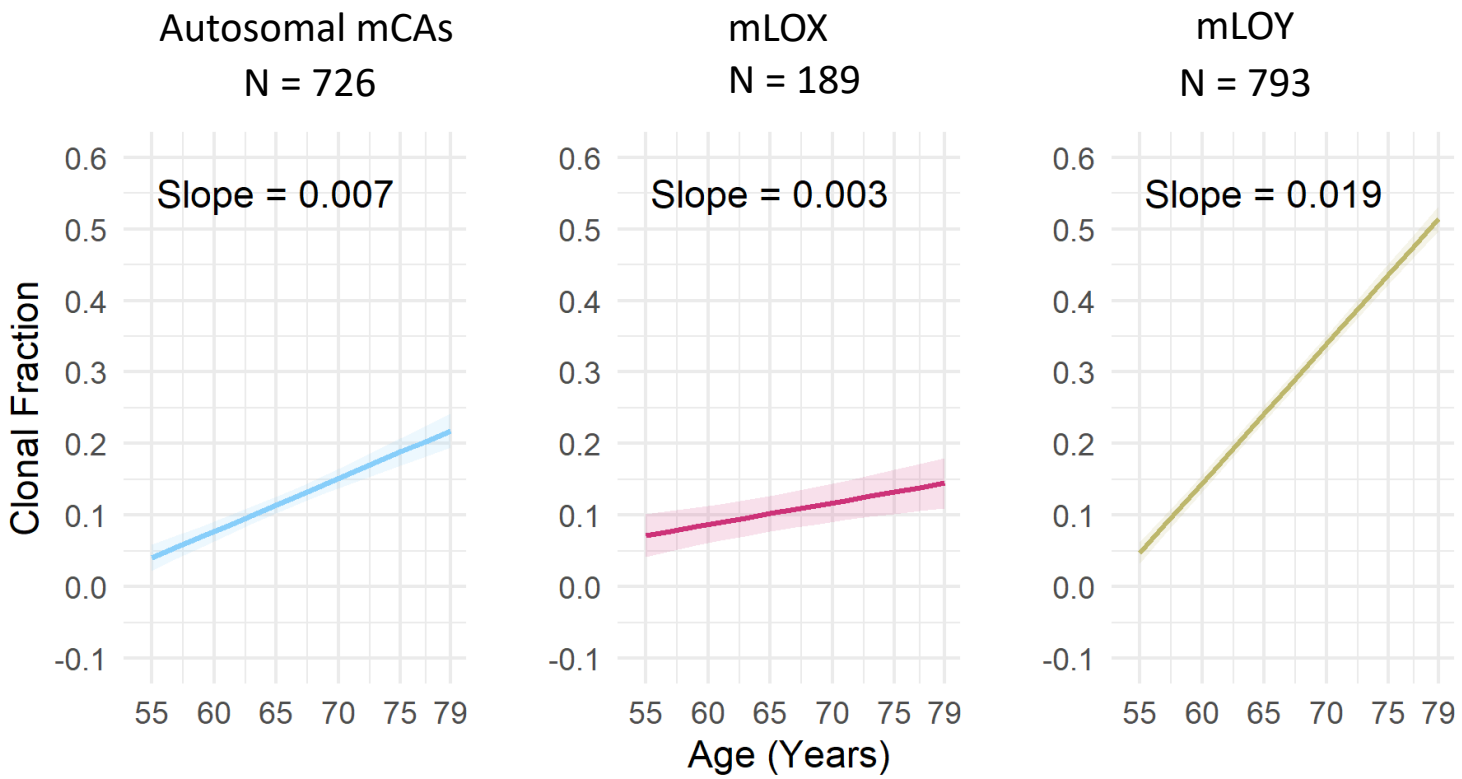
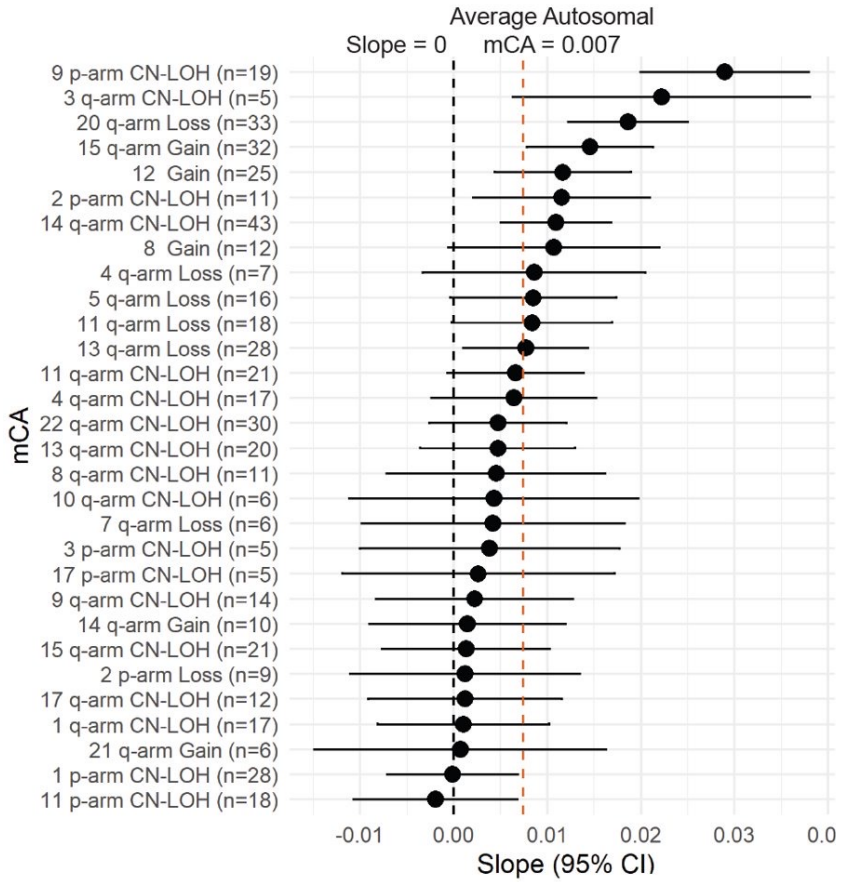
Clonal Expansion of mCAs in Longitudinal Samples





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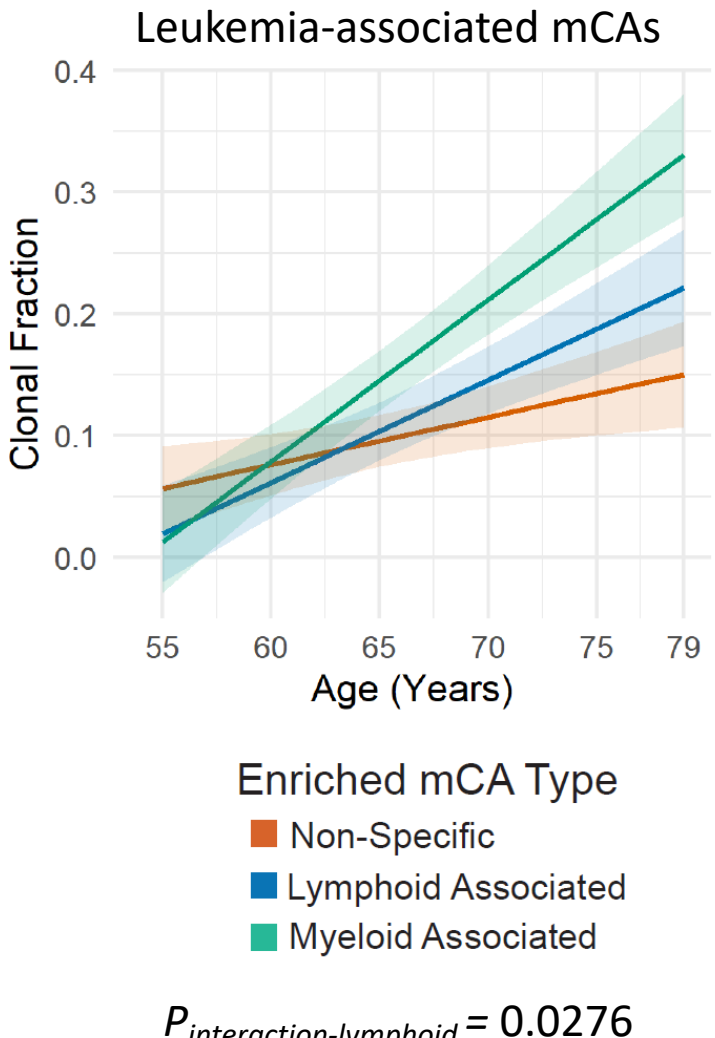
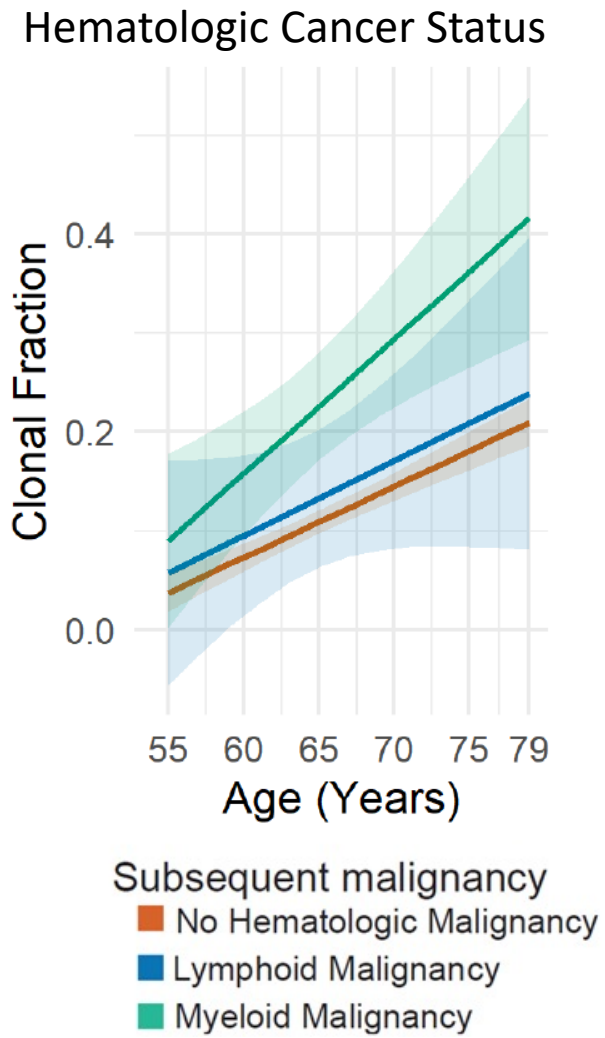
Higher Expansion Rate for Leukemia Associated mCAs





$P_{interaction\text{-}lymphoid} = 0.0276$

$P_{interaction\text{-}myeloid} = 1.48 \times 10^{-5}$

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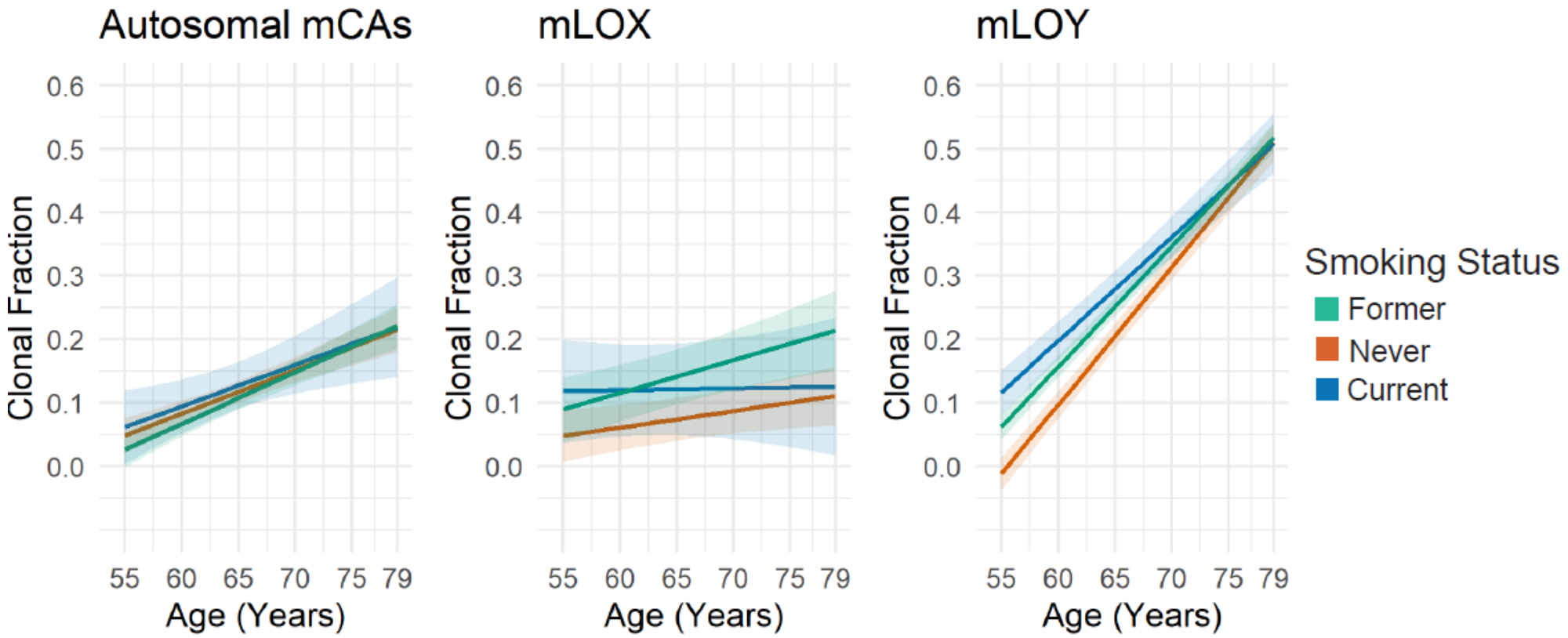
Smoking and Clonal Expansion



$P_{interaction\text{-}Current}$ = **0.0003**

$P_{interaction\text{-}Former}$ = **0.0091**

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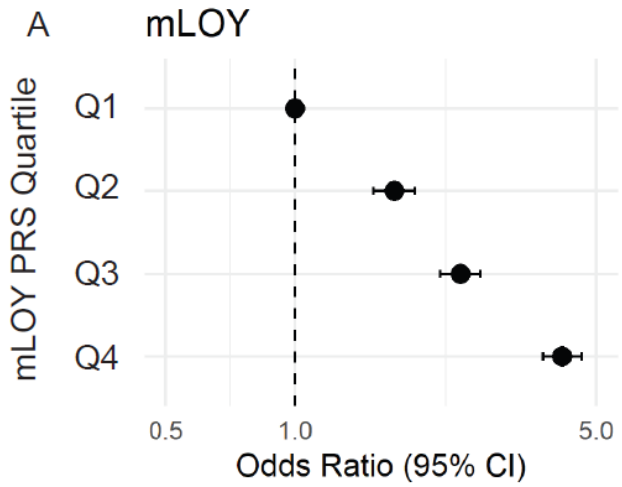
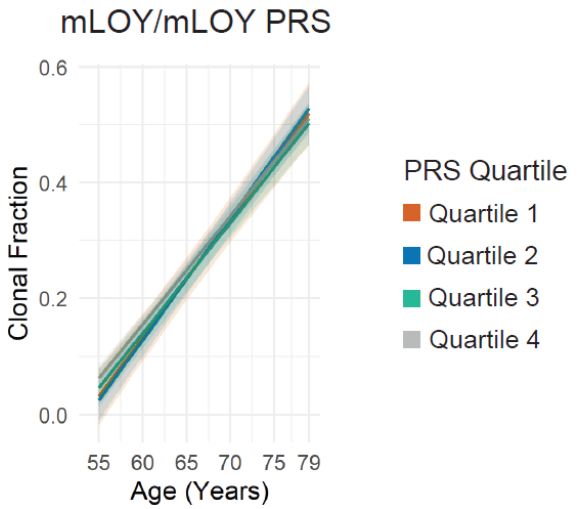
PRS for mLOY and mLOX and clonal expansion

Cross-sectional prevalence

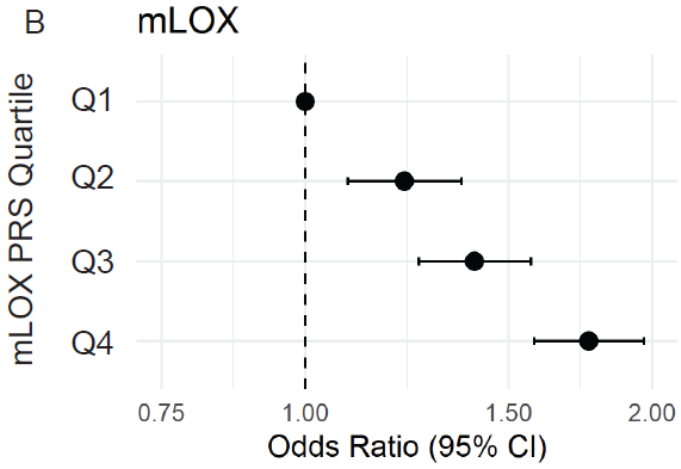
Longitudinal expansion

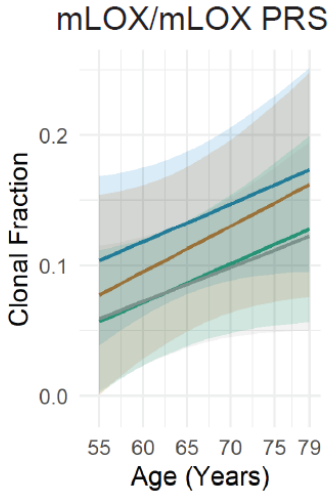
mLOY PRS





mLOX PRS





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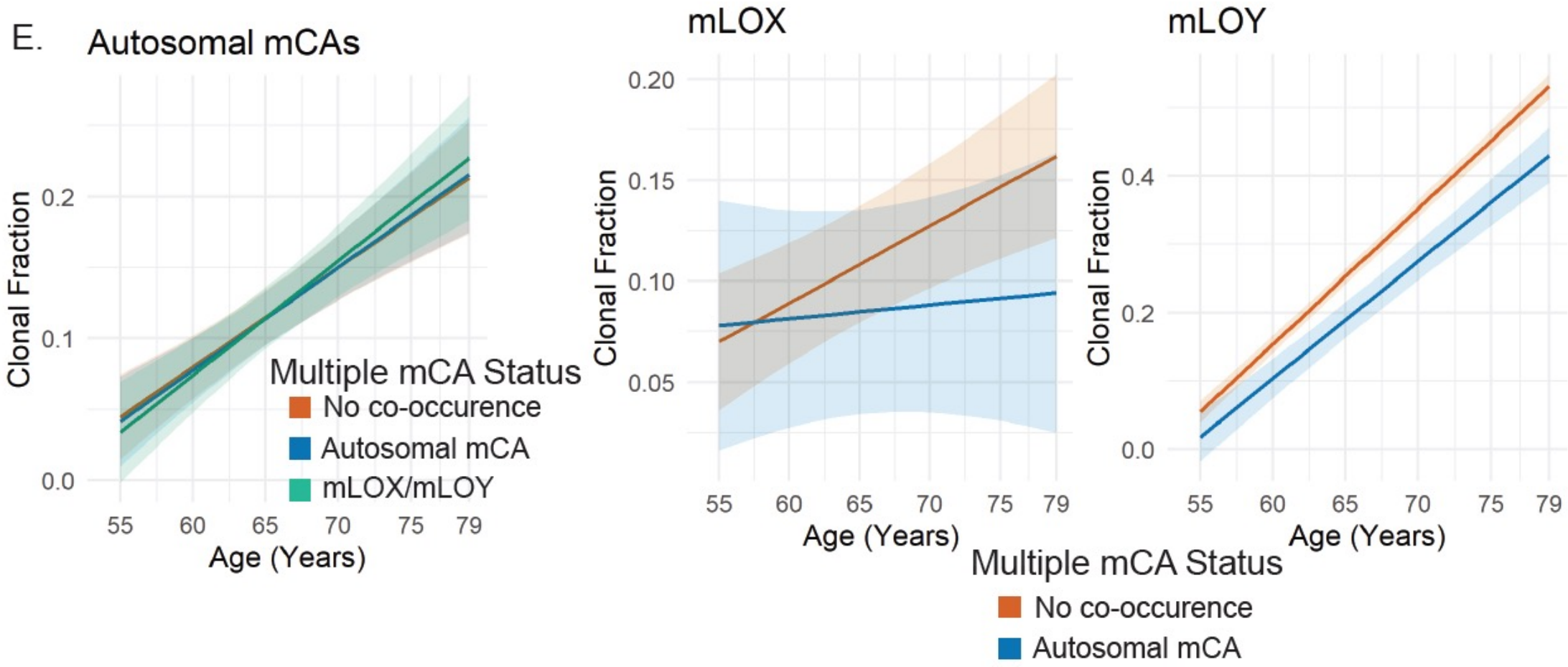
Multiple mCAs in an Individual and Clonal Expansion



$P_{interaction_autosomal}$ = **0.0390**

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Co-Occurring CH Exhibits Strong Selection



CHIP-CHIP

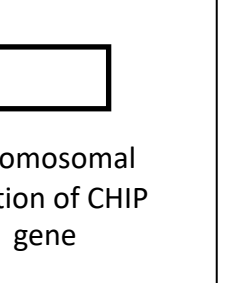
	DNMT3A	TET2	ASXL1	PPM1D	SRSF2	YLPM1	TP53	ZNF318	SRCAP	ZBTB33	SF3B1	JAK2	NF1	GNB1	ATM	CBL	KMT2C	MYD88	KMT2D	CHEK2	NFE2	IDH2
DNMT3A		4.2	0.6	2.1		3	1.9		2.6	4	3.4	2.9		2.8	4.9	2.3				3.7		
TET2	4.2		4.8	2.1	28.8	3.3	2.7	4.5	3.8		8.1	8.3	3.7						6.2			
ASXL1	0.6	4.8			10.3							8.5										
PPM1D	2.1	2																				
SRSF2		28.7	10.4																			783.1
YLPM1	3	3.3																				
TP53	1.9	2.7																				
ZNF318		4.4																				
SRCAP	2.5	3.8																				
ZBTB33	4																					
SF3B1	3.3	8																				
JAK2	2.9	8.3	8.7																		182.5	
NF1		3.7																				
GNB1	2.8																					
ATM	4.8																					
CBL	2.3																					
KMT2C																						
MYD88																						
KMT2D		6.2																				
CHEK2	3.6																					
NFE2												182.7										
IDH2					804.1																	

mCA-mCA

	Chr1	Chr2	Chr3	Chr4	Chr5	Chr6	Chr7	Chr8	Chr9	Chr10	Chr11	Chr12	Chr13	Chr14	Chr15	Chr16	Chr17	Chr18	Chr19	Chr20	Chr21	Chr22	mLOX	mLOY
Chr1		9.3	12.3	7.2	10.3	15	12.1	13.3	4.4	9.2	3.9	7.5	8.4	5.9	2.2	7	6.7	12.4	4.4				1.4	
Chr2	9.2		12	19.5	16.1	18.6	8.9	16.8	7.2	15.5	8.2	11.6	8.4	6.2	4.7	9.7	12		9.4			5.6	1.6	
Chr3	12.3	11.9		14.7	13.7	35.6	47.6	35.5	13.3	19.4	7.5	48.8	13.8	6.2	4	6.9	15.6	119.4	10.2		13.9	4.2	2	
Chr4	7.2	19.4	14.5		15.2	12.8	11	21.7	6.2	13.3	7	16.5	12.6	8.8			8.9	28.8	8.1			7.4	2.3	
Chr5	10.3	16.1	13.7	15.3		22.1	21.1	19.3	6.9	15.4	5.2	14	9.7	6.1		9.4	12.6	18.1					2.1	
Chr6	15	18.6	35.6	12.8	22.1		28.5	48.2	8.9	23.1	9.2	11.1	14.1	6		10.8	20.9	27.8		7			1.7	
Chr7	12.1	8.9	47.7	11	21.1	28.5		32.2	6.8	13.3	6.1	22.7	16.5	13.5			14.1	20.3	18.4			5.9		
Chr8	13.3	16.8	35.6	21.9	19.2	48.2	32.4		16	30.2	5.4	24.8	12.6	13.1		9.7	28.7	50.5			11.4	10	1.7	
Chr9	4.4	7.2	13.3	6.2	6.9	8.9	6.8	16			3	6.6	7.7	9	2.7		6.2	13	4.9	12.7		5.8		1.3
Chr10	9.2	15.5	19.3	13.2	15.4	23	13.3	30.2			4.3	9	7.8	5.3		10.3	12.1	16.2	9.8					
Chr11	3.8	8.2	7.6	7	5.2	9.1	6.1	5.5	3	4.4		3.6	6.4	4.6		6	4.2	4.9				3.1	1.6	
Chr12	7.5	11.6	49	16.5	14	11.1	22.5	24.8	6.6	9	3.6		19.4	7.1	2.7	5.3	7.7	94.4	79.2		9	11.4	2.3	
Chr13	8.4	8.4	13.9	12.7	9.7	14.1	16.4	12.5	7.8	7.8	6.4	19.5		15.7	3	8.9	11.9	19.5	8.3	5	6.8	25.3	2.6	0.8
Chr14	5.9	6.2	6.2	8.9	6.1	6	13.4	13.1	9	5.3	4.5	7.1	15.6		2.8	3.6	3.1	5.6	3.7	5.3	4.8	6.1	1.8	1.9
Chr15		4.7	3.9						2.7			2.7	2.9	2.9			4.3	5.2				2.9		2
Chr16	7	9.7	6.9		9.3	10.7		9.7		10.3	6	5.3	8.9	3.5			8.5	9.1		7.8	9.1	4		
Chr17	6.7	12	15.6	8.9	12.6	20.9	14.1	28.7	6.2	12.1	4.2	7.7	11.9	3.1	4.3	8.5		6.6	8.5		43.5	3.5	1.4	
Chr18	12.4		119.7	28.9	18.1	27.8	20.2	50.3	13.1	16.3	4.9	94.5	19.5	5.6	5.2	9.2	6.6		102.5	9	16.4	21.1	2.5	
Chr19	4.4	9.4	10.2	8.1			18.4		4.9	9.9		79.2	8.3	3.7			8.5	102.8				7.3	2	
Chr20						6.9			12.8				4.9	5.3		7.8		8.9						
Chr21			13.9				11.4					9	6.8	4.8		9.1	43.7	16.3				6.7	2.5	
Chr22		5.6	4.2	7.4			5.9	9.9	5.8		3.1	11.4	25.3	6.1	2.9	4	3.5	21.1	7.3		6.7		3	
mLOX	1.4	1.6	1.9	2.2	2.1	1.7		1.7			1.6	2.3	2.6	1.7				2.4	2		2.4	2.9		
mLOY									1.4				0.7	1.9	2.1									

CHIP-mCA

	Chr1	Chr2	Chr3	Chr4	Chr5	Chr6	Chr7	Chr8	Chr9	Chr10	Chr11	Chr12	Chr13	Chr14	Chr15	Chr16	Chr17	Chr18	Chr19	Chr20	Chr21	Chr22	mLOX	mLOY
DNMT3A		2.6		2.4					1.4			1.5				1.5						1.5		0.7
TET2	1.8		2.4	26.2					2.6		2.6	1.8		3.3				2.1	2.8			3.1	1.3	0.7
ASXL1	3			6.2					2.8		2.4			3.5			2.5							
PPM1D																								
SRSF2				18										6										0.5
YLPM1														14.6										
TP53													12.5				26.7							
ZNF318																								
SRCAP																								
ZBTB33																								
SF3B1																								
JAK2	8.8							29.4	481.4					22.4						31.2				0.4
NF1																								
GNB1																								0.4
ATM											27.4													
CBL																								
KMT2C																								
MYD88			133.6	42.2								29.3	105.6					165.6				27.5	3.1	
KMT2D												29												
CHEK2																								
NFE2									67.5											64.6				
IDH2																							4.5	



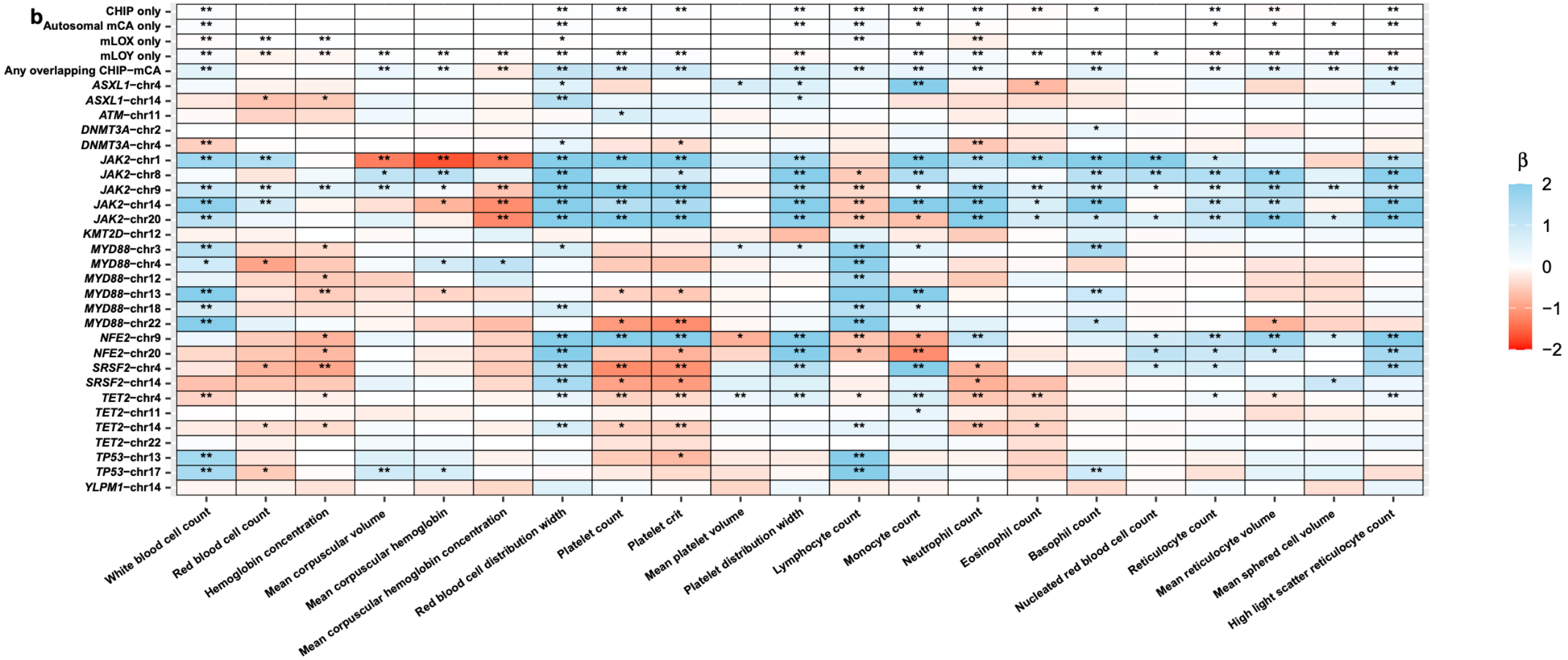
Note: Model covariates include age, age^2, sex, smoking status, and genetic similarity to reference genomes

1.6% of individuals have CH co-occurrences

- Co-occurrences are highly enriched
- Some potential clonal competition
- CHIP CF > mCA CF

Barnao *Nat Commun* 2026

Co-Occurring CH Alters Blood Cell Counts



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Co-Occurring CH Confers High Leukemia Risk

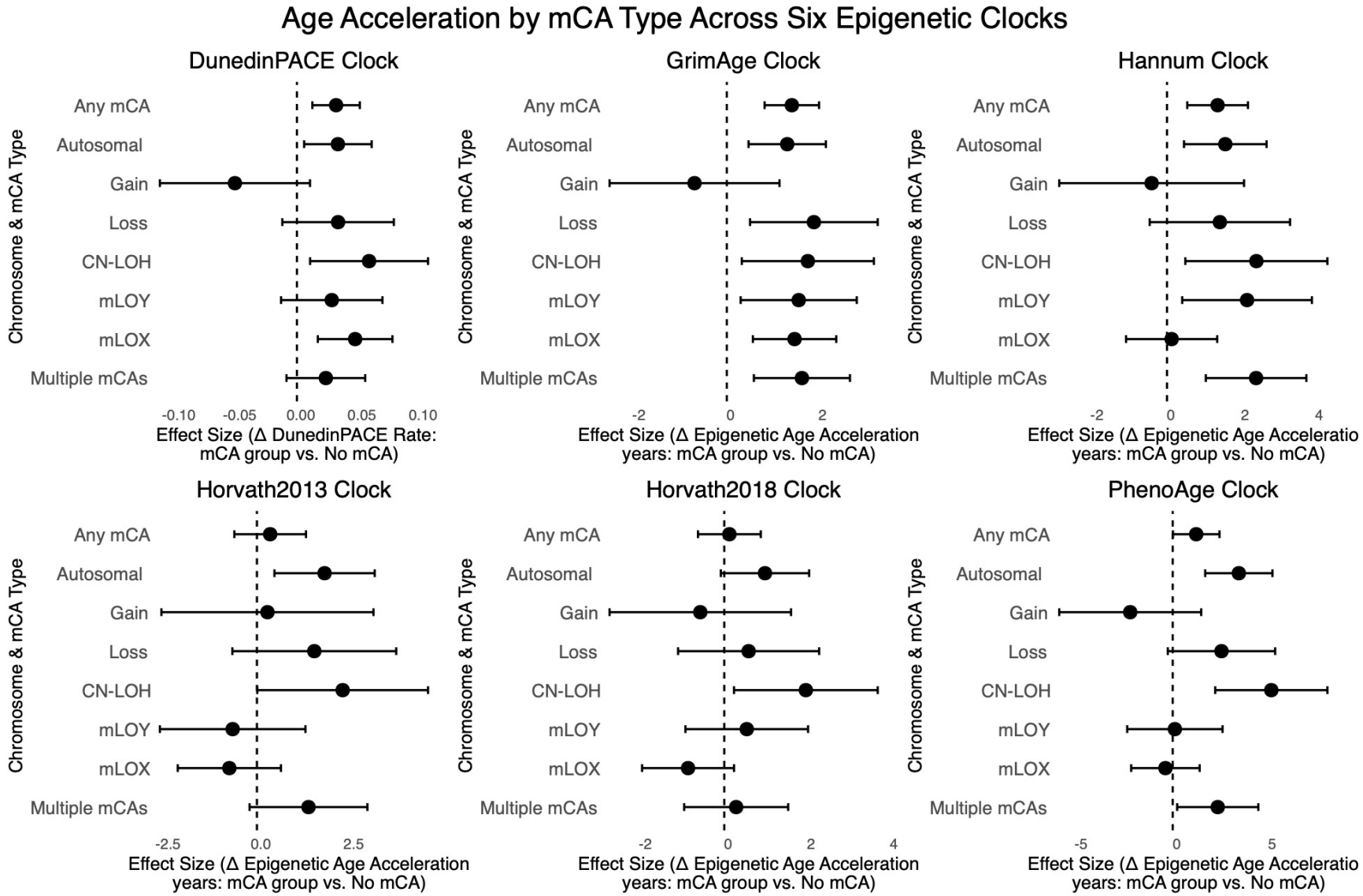
Co-occurrence	Malignancy type	Cases	Hazards Ratio	95% CI	*p* value
CHIP only	Myeloid	201	5.5	5.8 - 6.4	6.58E-113
Autosomal mCA only	Myeloid	66	3	2.3 - 3.8	2.24E-18
Overlapping CHIP-mCA	Myeloid	56	80.6	61.7 - 105.4	9.78E-226
NFE2 -chr9	Myeloid	6	649	290.4 - 1450.4	4.09E-56
JAK2 -chr14	Myeloid	10	573	306.3 - 1071.8	6.51E-88
JAK2 -chr20	Myeloid	8	548.4	272.7 - 1092.8	3.98E-24
JAK2 -chr9	Myeloid	40	233.6	170.3 - 320.4	5.95E-251
ASXL1 -chr4	Myeloid	5	183	75.9 - 441.4	4.31E-31
TET2- chr4	Myeloid	11	44.9	24.8 - 81.3	4.02E-36

Barnao *Nat Commun* 2026

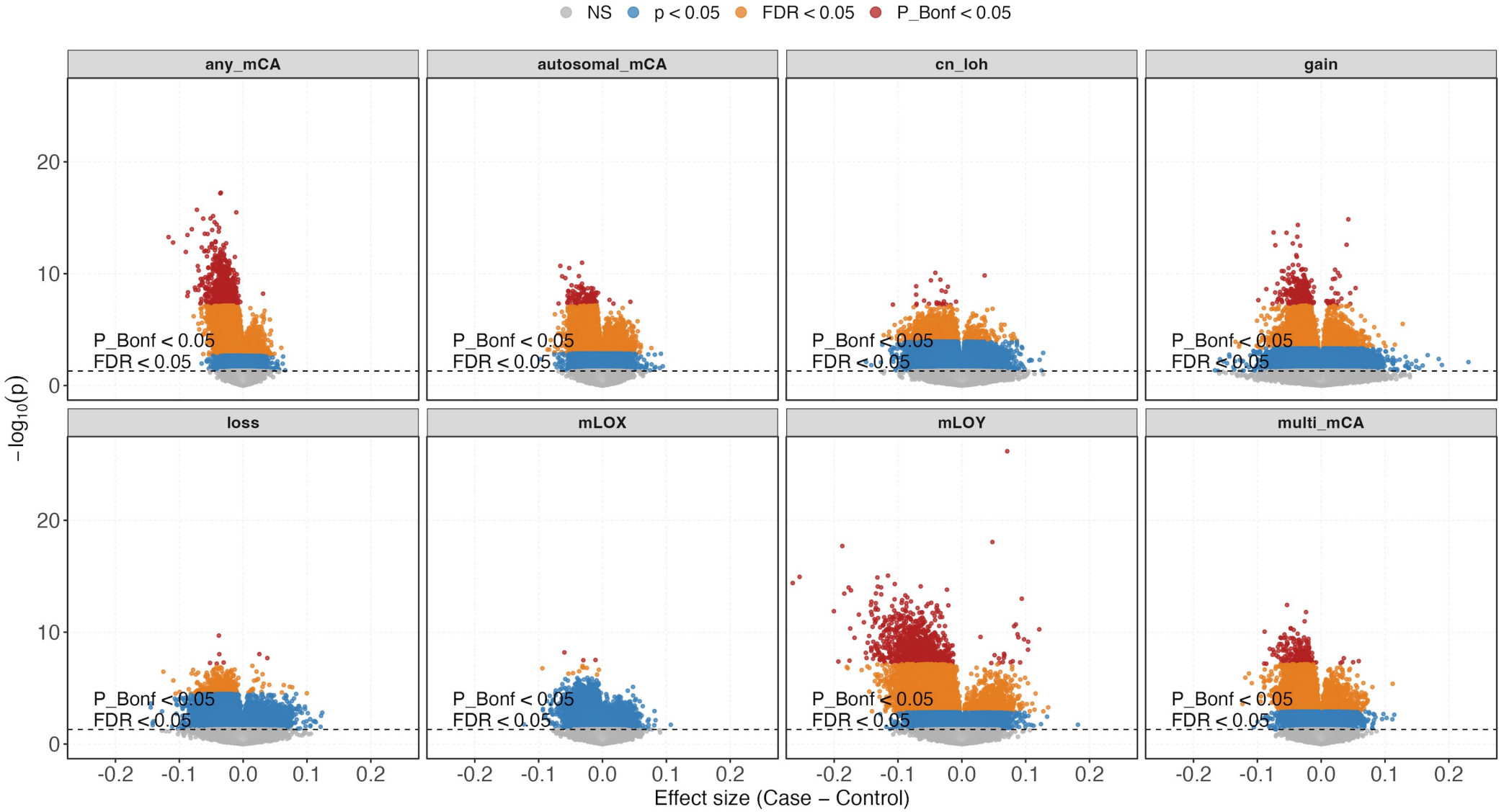
mCAs and Methylation Clocks

482 PLCO participants with EPIC methylation array

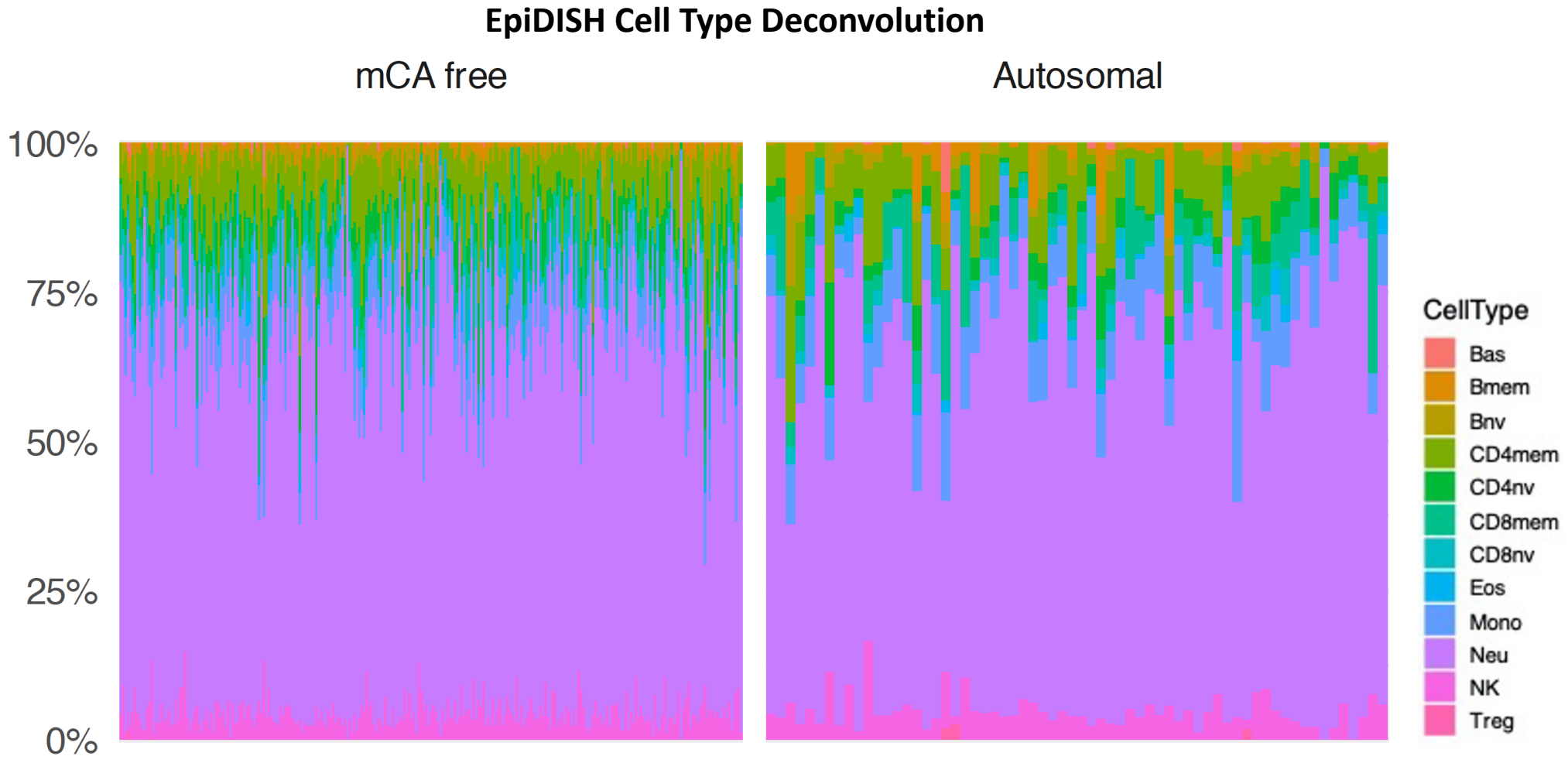




EWAS to Nominate Differential Methylated Regions



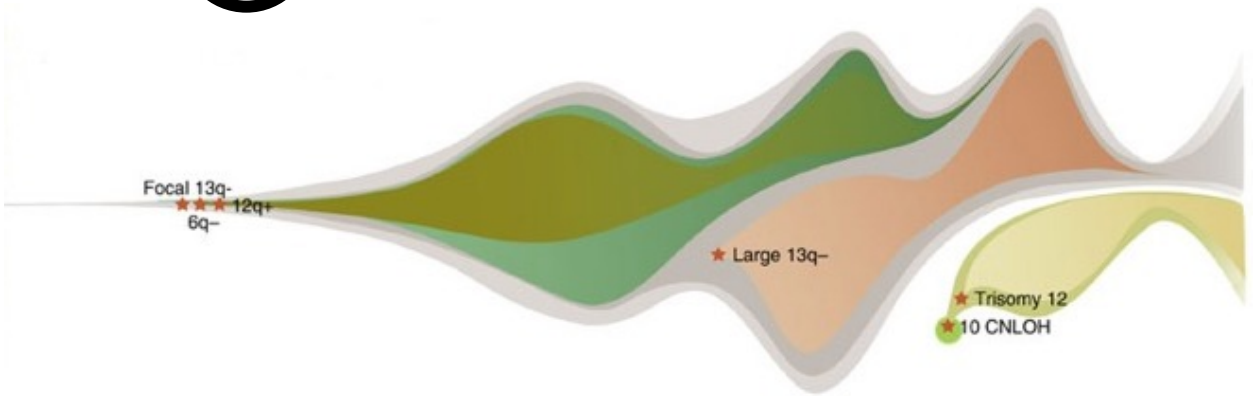
mCA Methylation Immune Cell Profiles



Higher B cell fractions in individuals with autosomal mCAs (β=0.011, p-value=2×10^{-8})

What do mCAs tell us about health/disease status?

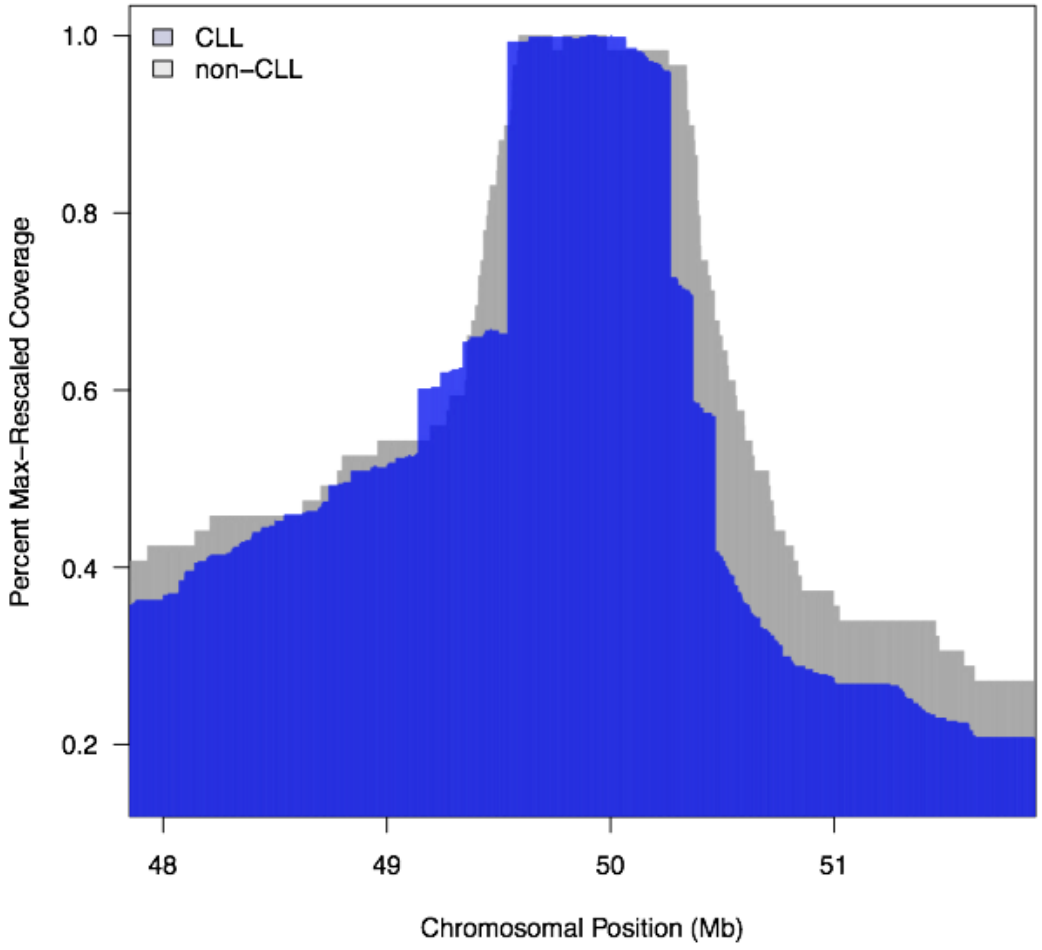
1 Early Pre-dysplastic Clones



Clones with one of many early events with potential predisposition for future hematologic cancer risk

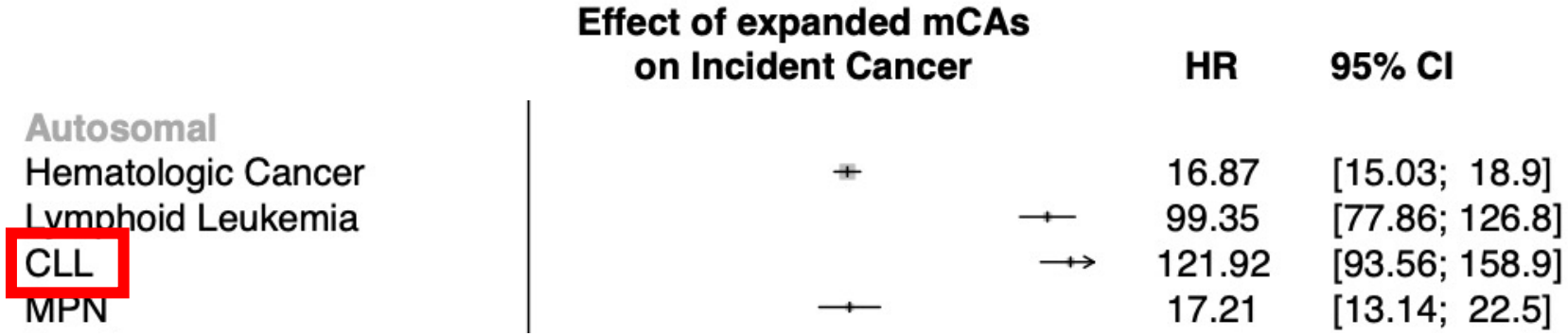
Early Pre-Leukemic Clones

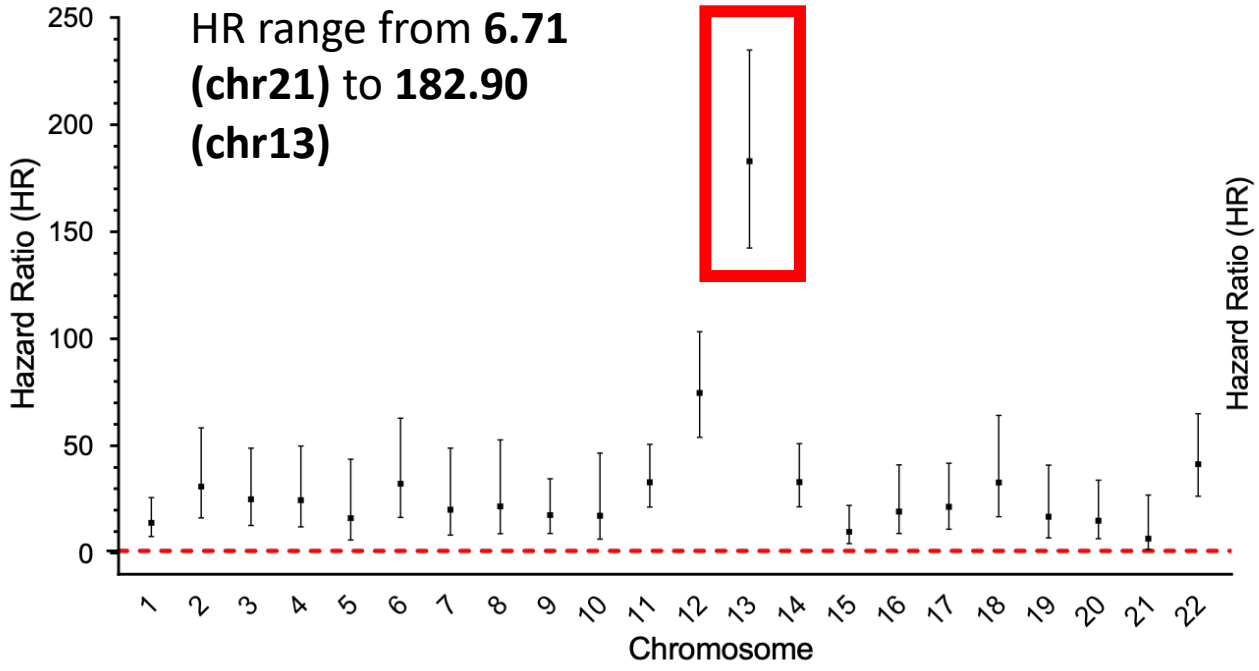
- Mosaic 20q and 13q14 deletions span the same genomic region as in myeloid and lymphoid malignancies
  - Observed up to 15 years prior to hematologic malignancy diagnosis
- Frequencies of 20q and 13q14 deletions are higher than population rates of the respective disease
  - Not all individuals with mCAs progress to disease, motivating further investigation

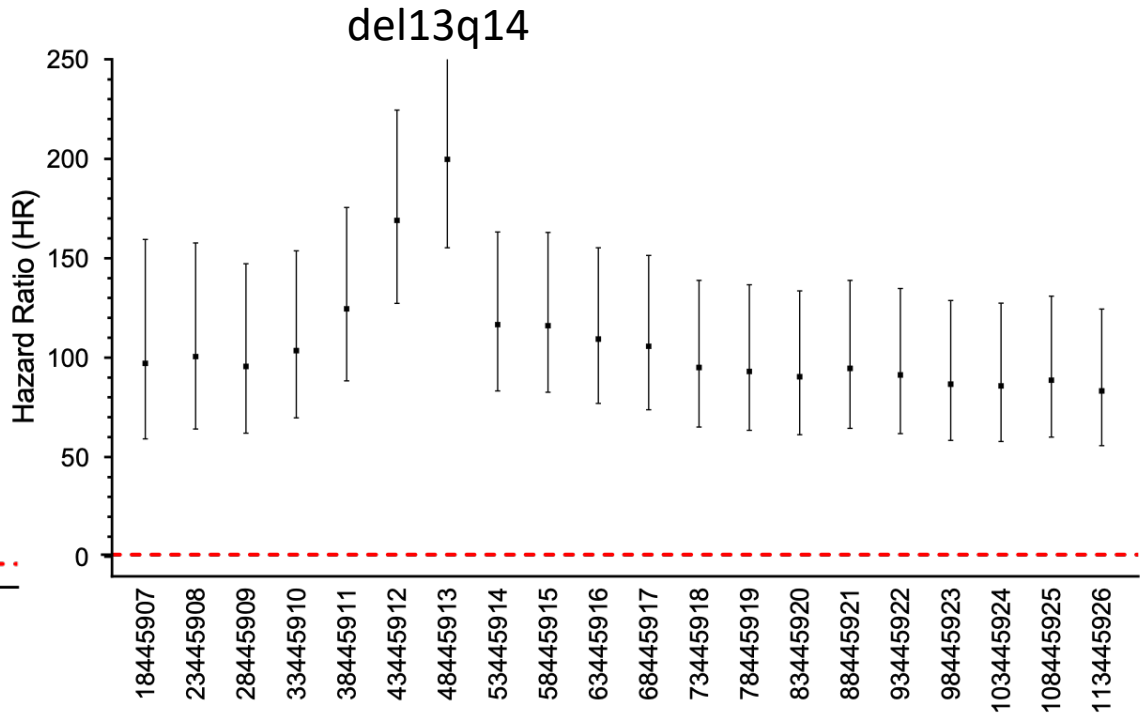


Machiela *J Hum Genet* 2016, Machiela *Blood Adv* 2017

Consequences of mCAs – Elevated Leukemia Risk







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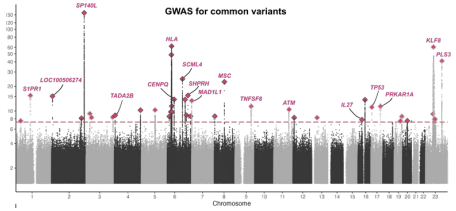
Zekavat *Nat Med* 2021, Hubbard *Cancer Res* 2025

CH and Utility for CLL Risk Stratification

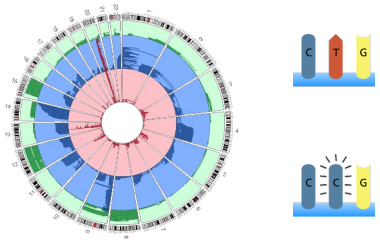
Classical Risk Factors



Germline Genetic Risk – Polygenic Scores (PGS)



Somatic Risk – mCAs and CHIP



Model	Test Set AUC (5-year risk*)
Model 0: Age, age-squared, sex, genetic ancestry, smoking + blood cell count	0.8372
Model 1: Model 0 + CLL PGS	0.8617
Model 2: Model 1 + any autosomal mCA	0.8824
Model 3: Model 1 + any CHIP mutation	0.8770
Model 4: Model 1 + any autosomal mCA + any CHIP mutation	0.8864

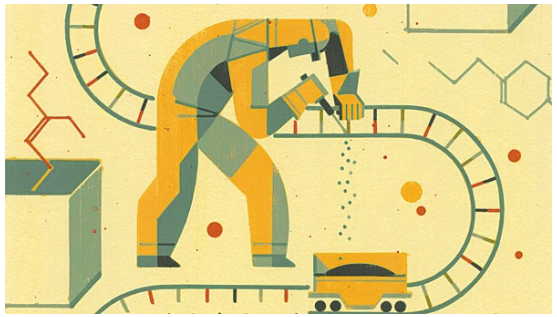
* AUC estimates for 10-year risk are slightly attenuated suggesting repeat measures could be necessary to maintain high predictive utility

Hubbard *Cancer Res* 2025

What do mCAs tell us about health/disease status?

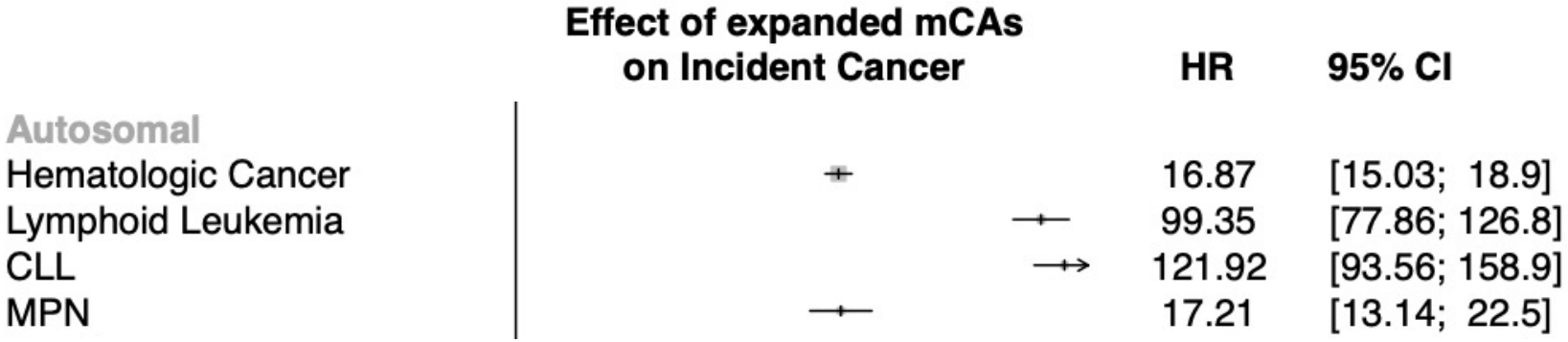
2 Marker of Genomic Integrity

Molecular barometer measuring interaction of endogenous/exogenous processes and a tissue's capacity to maintain/repair damage



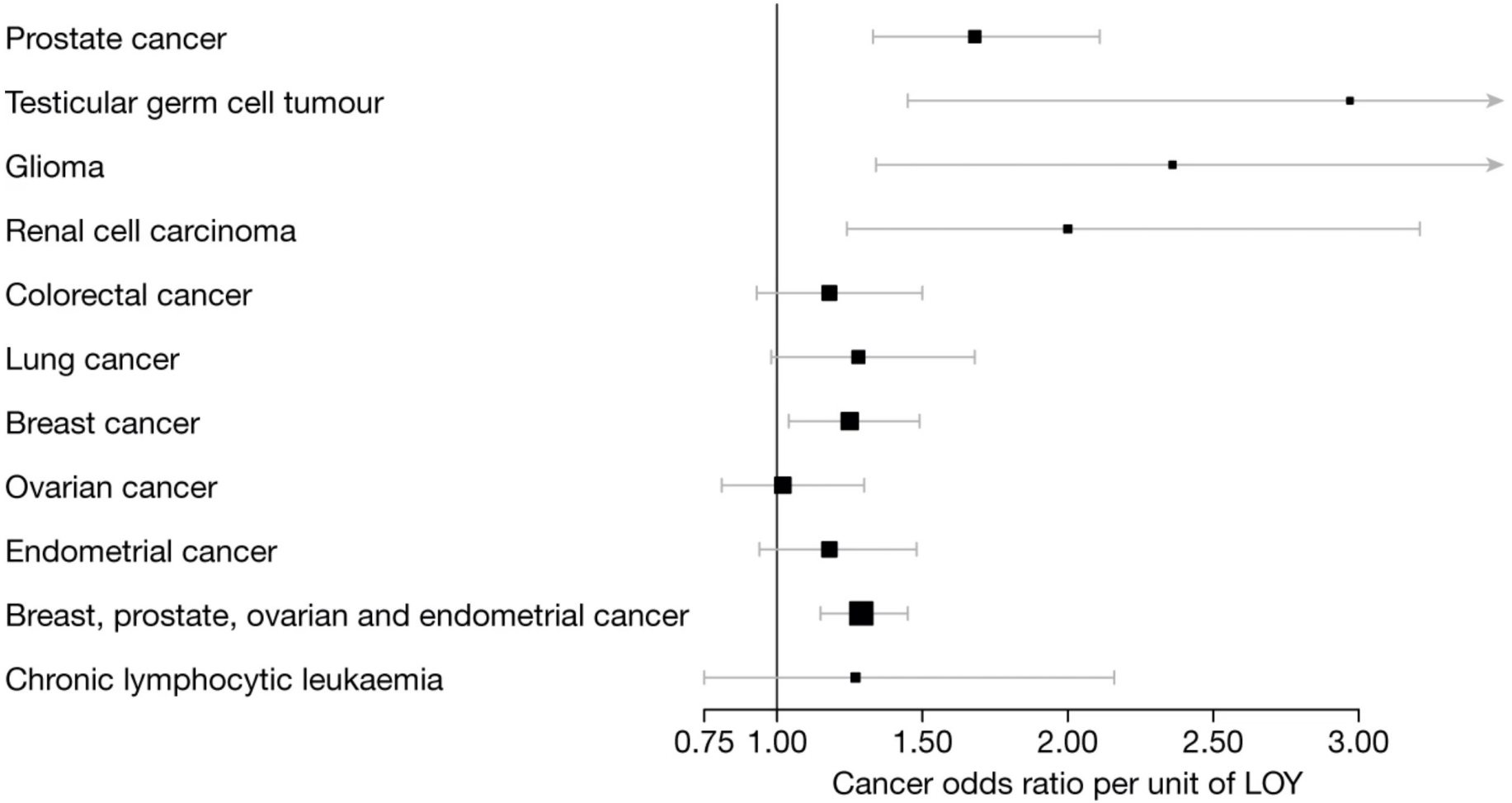


Consequences of mCAs – Solid Tumor Risk



Loftfield et al. *Can Res* 2019, Thompson et al. *Nature* 2019, Zekavat et al. *Nat Med* 2021

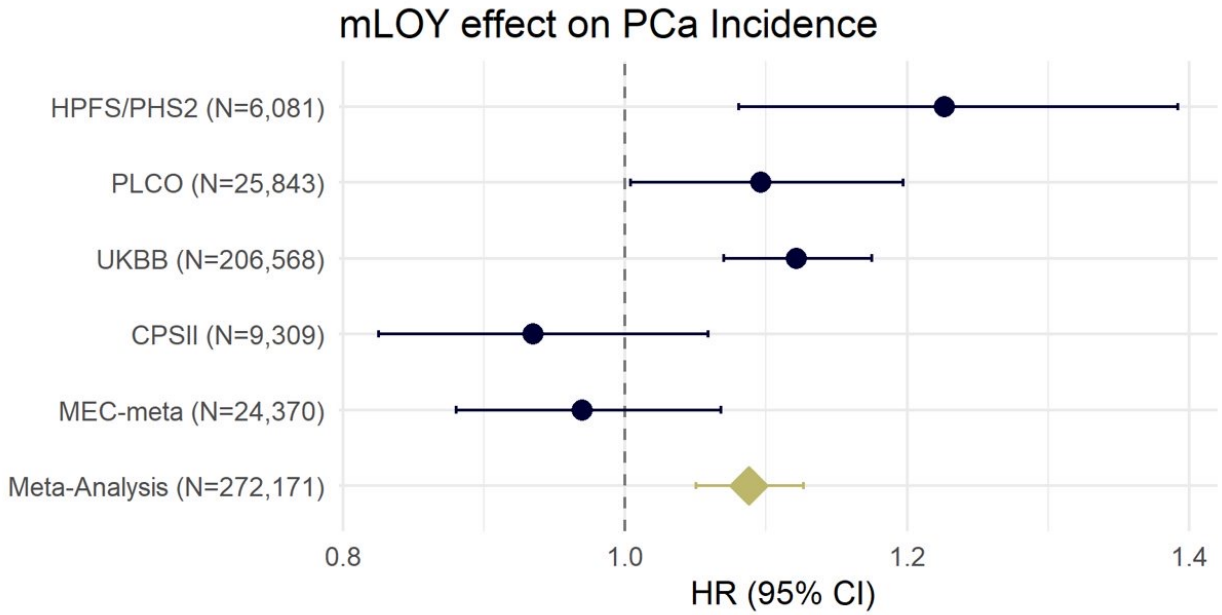
mLOY Susceptibility Variants are Associated with Solid Tumor Risk



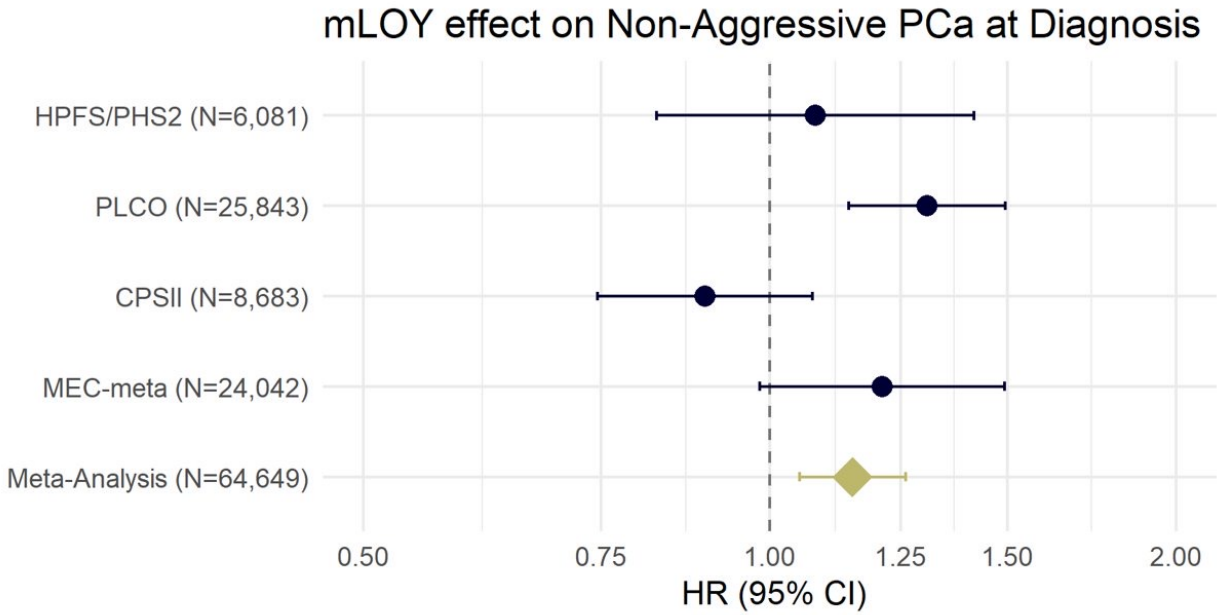
Thompson *Nature* 2019

mLOY is Associated with Prostate Cancer Risk





HR = 1.09 [1.05, 1.13]
P-Value = 2.25×10^{-6}

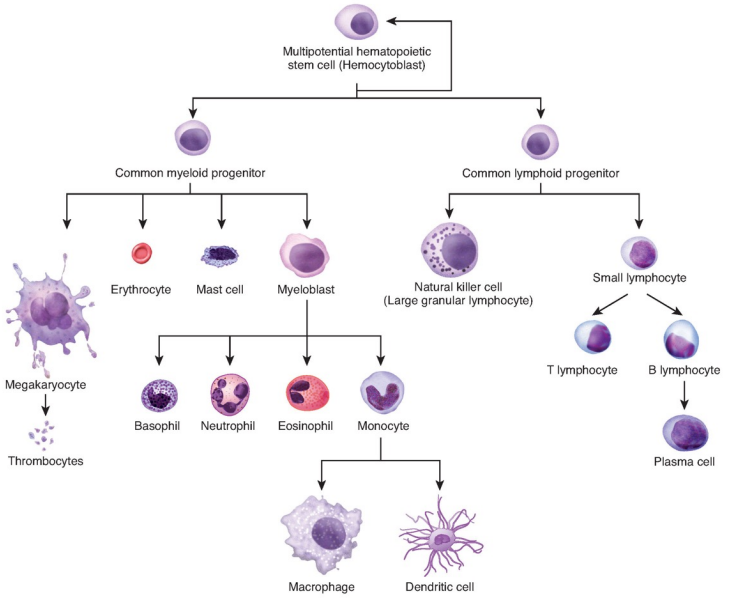


HR = 1.15 [1.05, 1.26]
P-Value = 0.0022

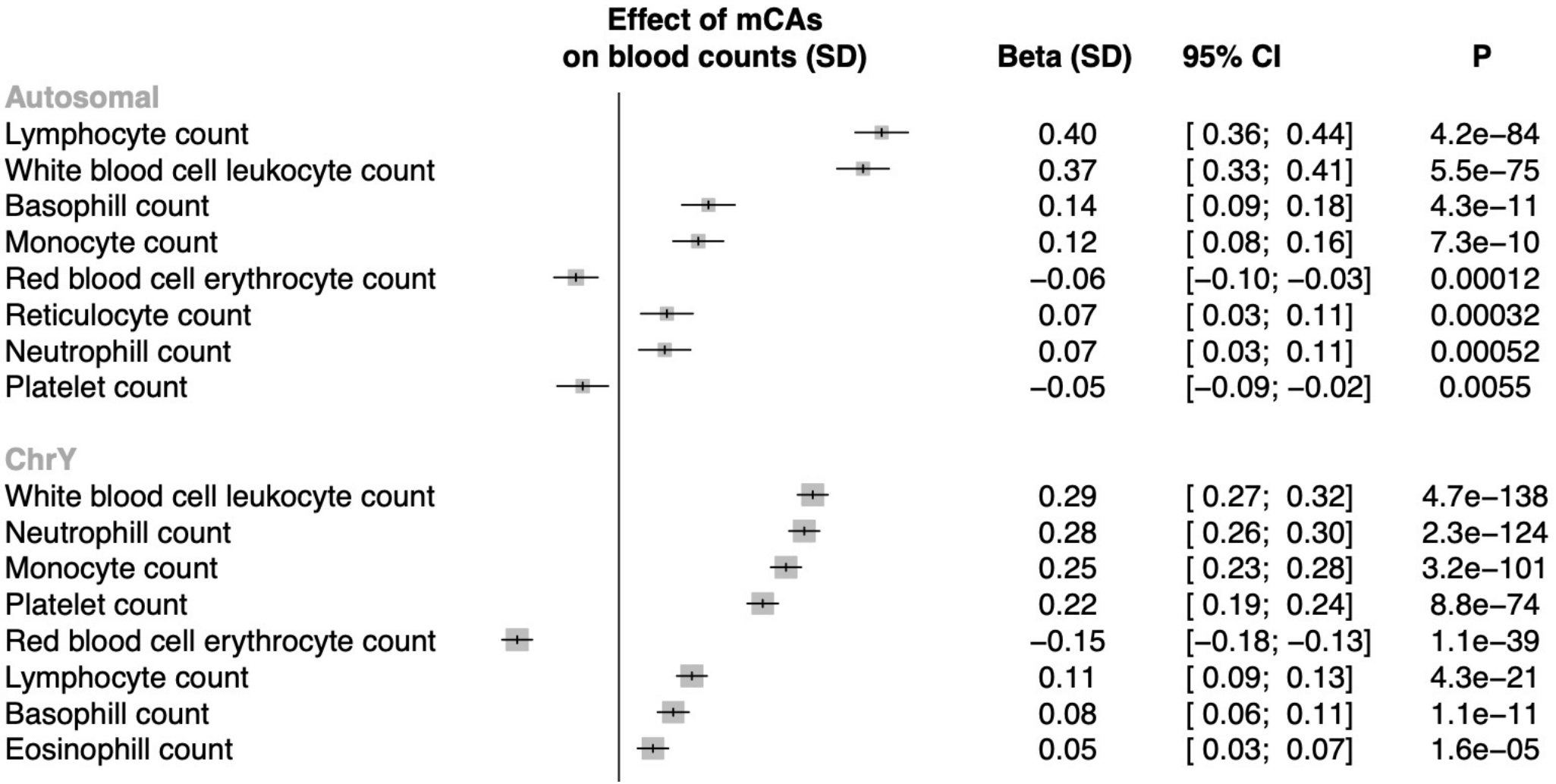
What do mCAs tell us about health/disease status?

3 Marker of Immune System Function

Hematopoietic stem cell production and differentiation



Consequences of mCAs – Altered Blood Cell Counts



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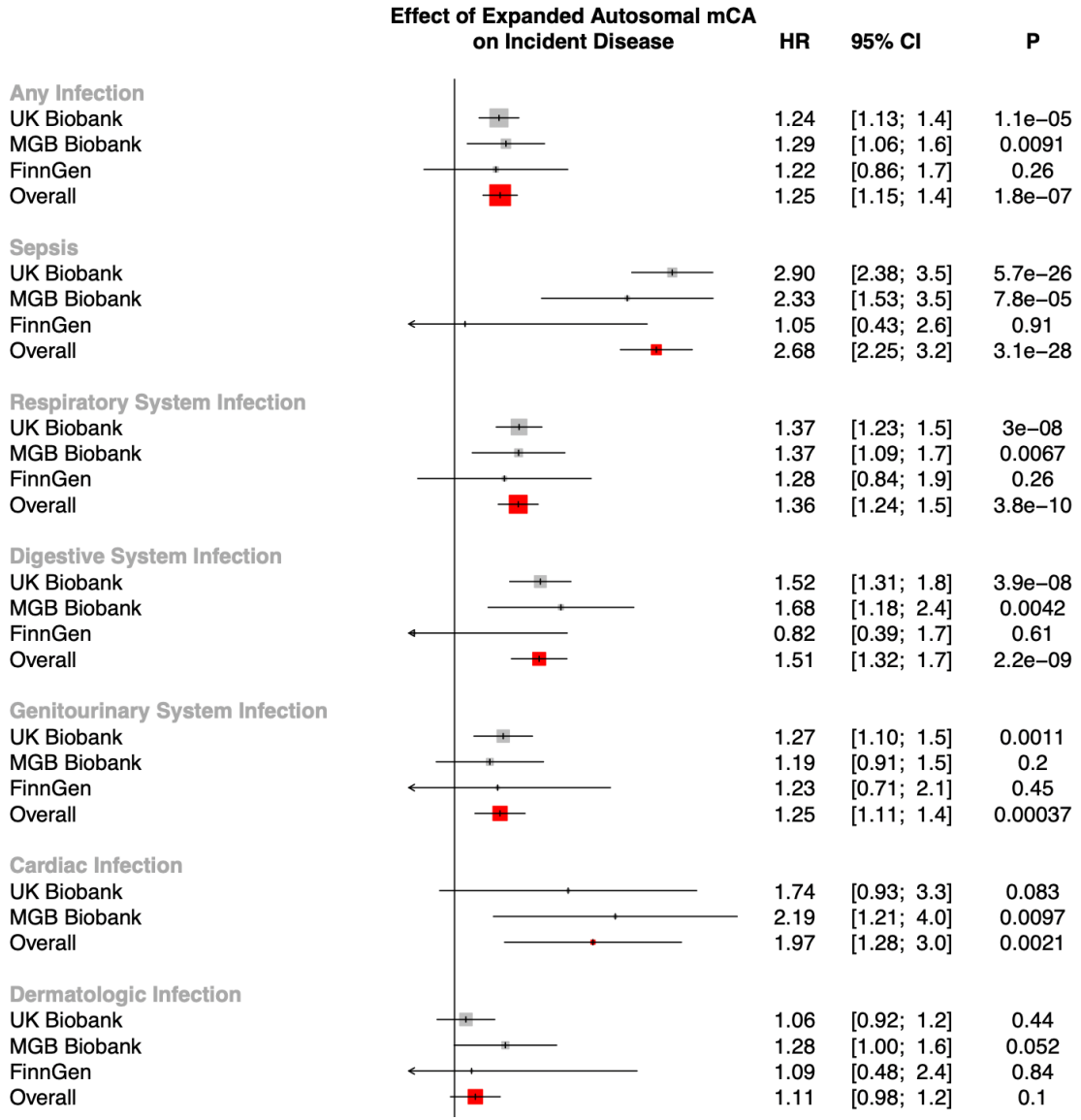
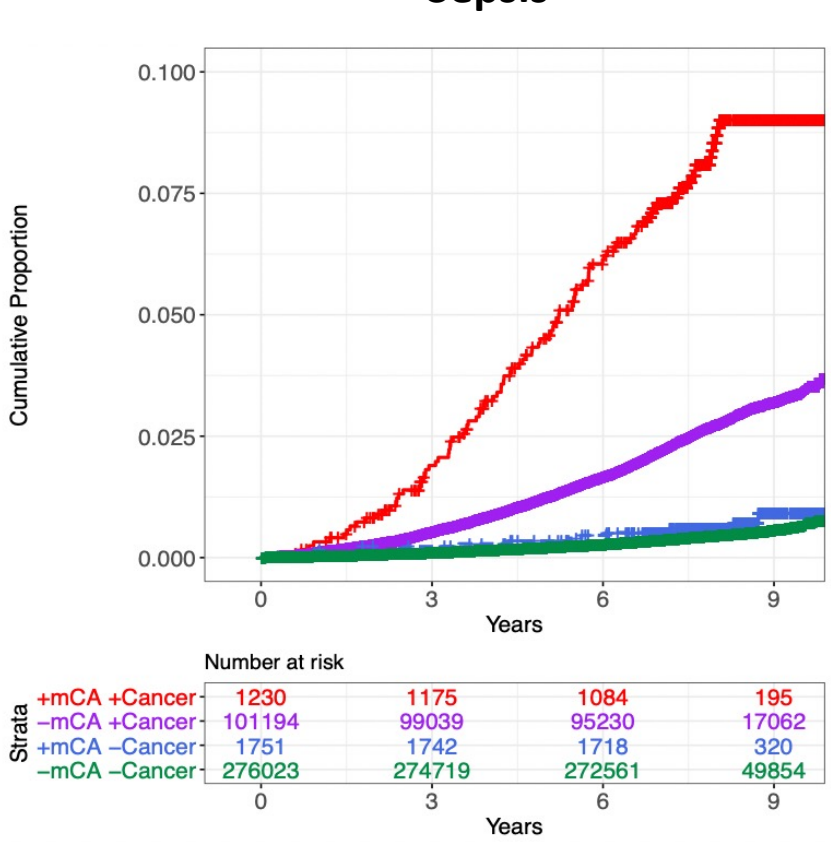
Consequences of mCAs – Increased Infectious Disease Risk





Individuals with prior cancer and detectable mCAs are at the highest risk

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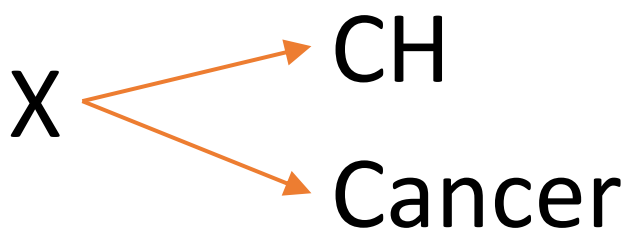
Thinking Causally

CH and Cancer (or another outcome)

(1) Risk Factor



(2) Common Cause



e.g., X = incorrectly modeling age, residual effects of smoking

(3) Reverse Causation



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State of Knowledge of mCAs in Circulating Leukocytes

- mCAs are detectable in healthy individuals with the frequency and expansion rate dependent on the chromosome affected
  - individual characteristics (e.g., age, sex, smoking, environmental exposures) can alter frequency or rate
- Germline variation has been associated with mCAs
  - Genetic susceptibility loci identified for mLOY and mLOX
  - Germline variation on specific chromosomes predisposes to mCA acquisition on those chromosomes
- mCAs are associated with hematologic malignancies – both lymphoid and myeloid
  - Not all individuals with mCAs will develop a hematologic malignancy
  - Which mCAs are concerning/high risk?
- mCAs co-occurrences with other types of CH are highly enriched
  - Select co-occurrences are associated with stronger phenotypic effects or high hematologic cancer risk
- mCAs are associated with select solid tumors and infection risk
  - Ongoing studies to disentangle whether associations are causal
- Performing integrative studies to shed light on potential biologic mechanisms promoting acquisition and clonal expansion of mCAs

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And many others…

https://authorarranger.nci.nih.gov/

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