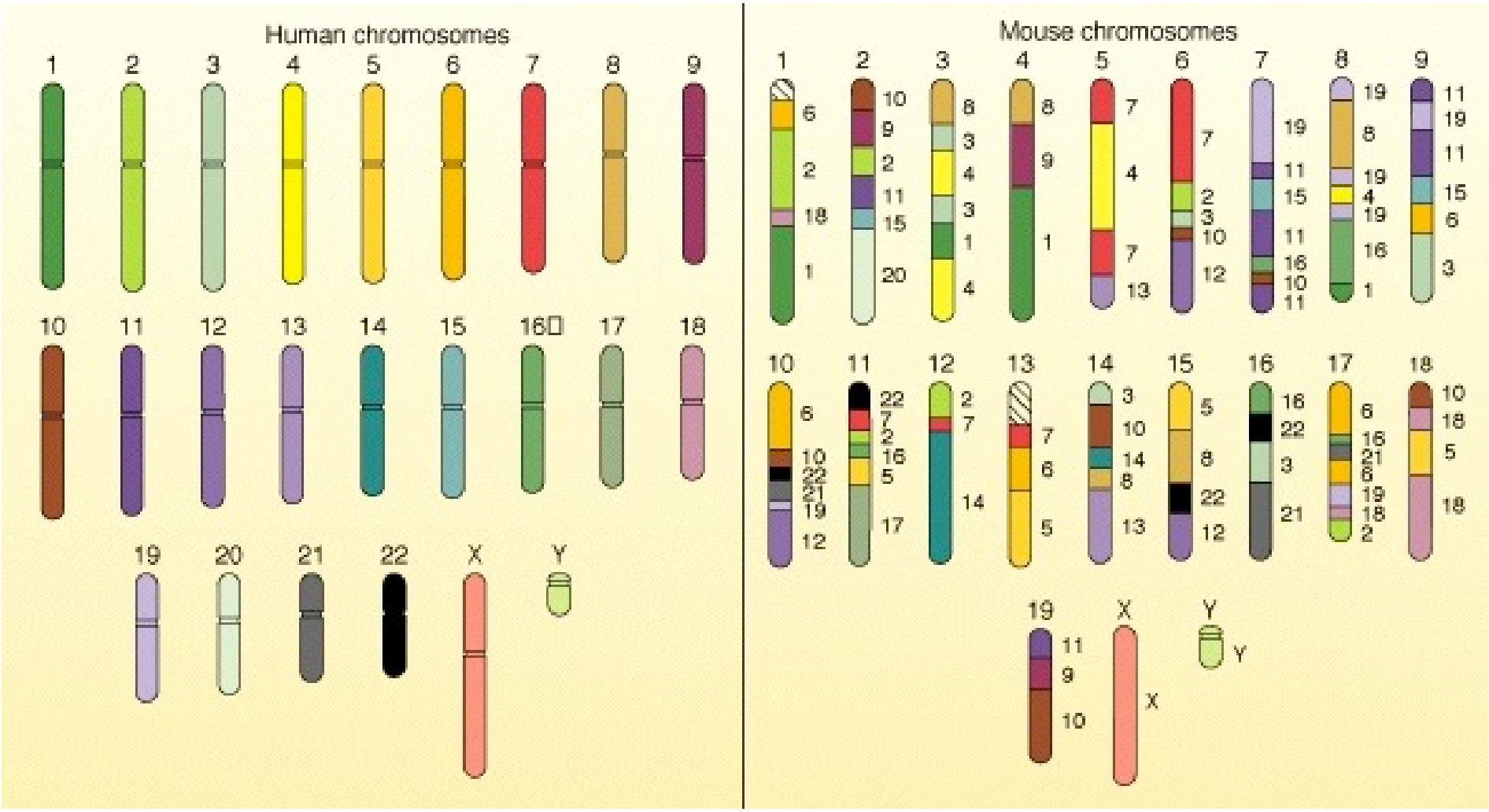
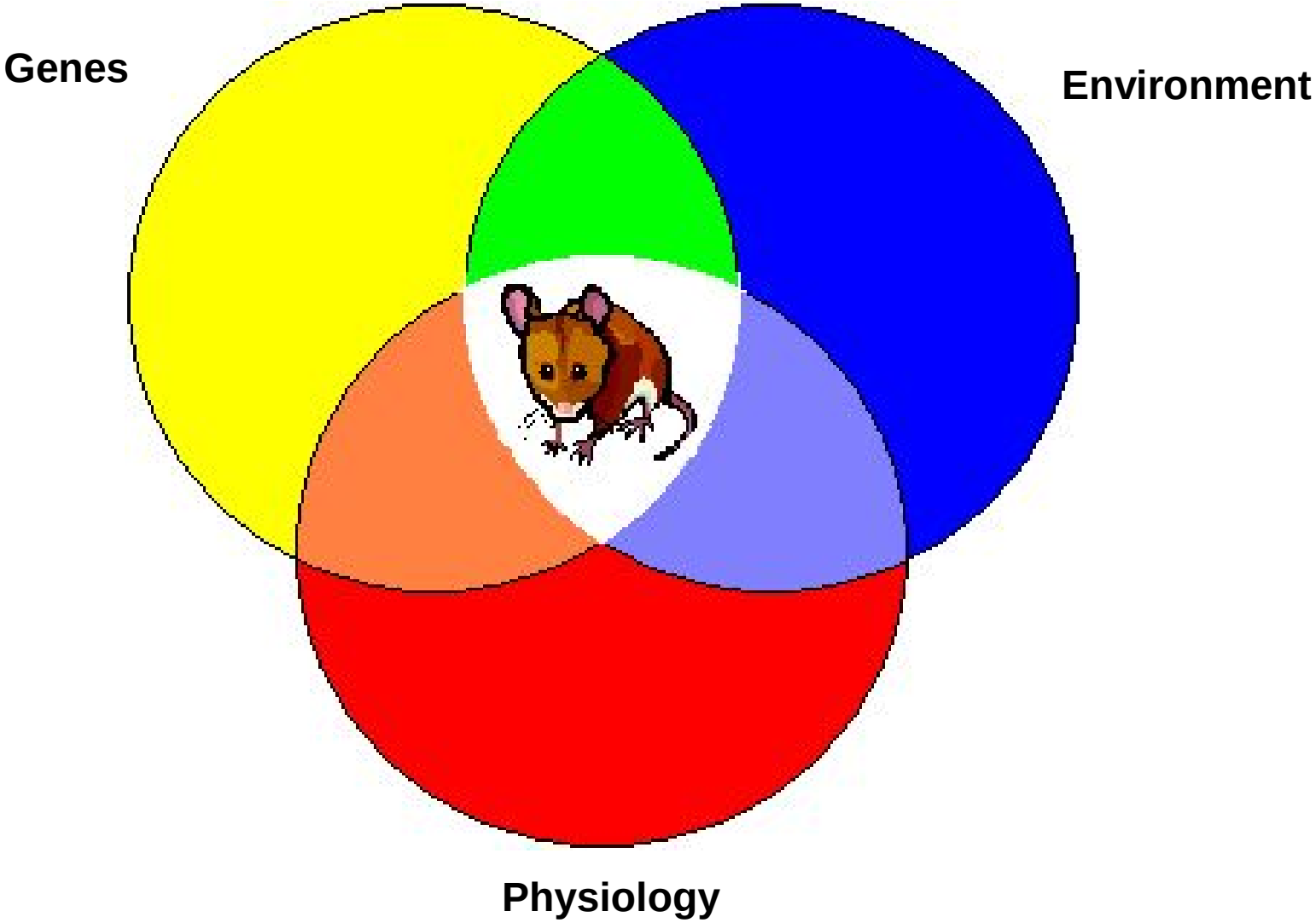
Reinventing the Laboratory Mouse

Gary A. Churchill
The Jackson Laboratory

Why Mice?

Human-Mouse Orthology



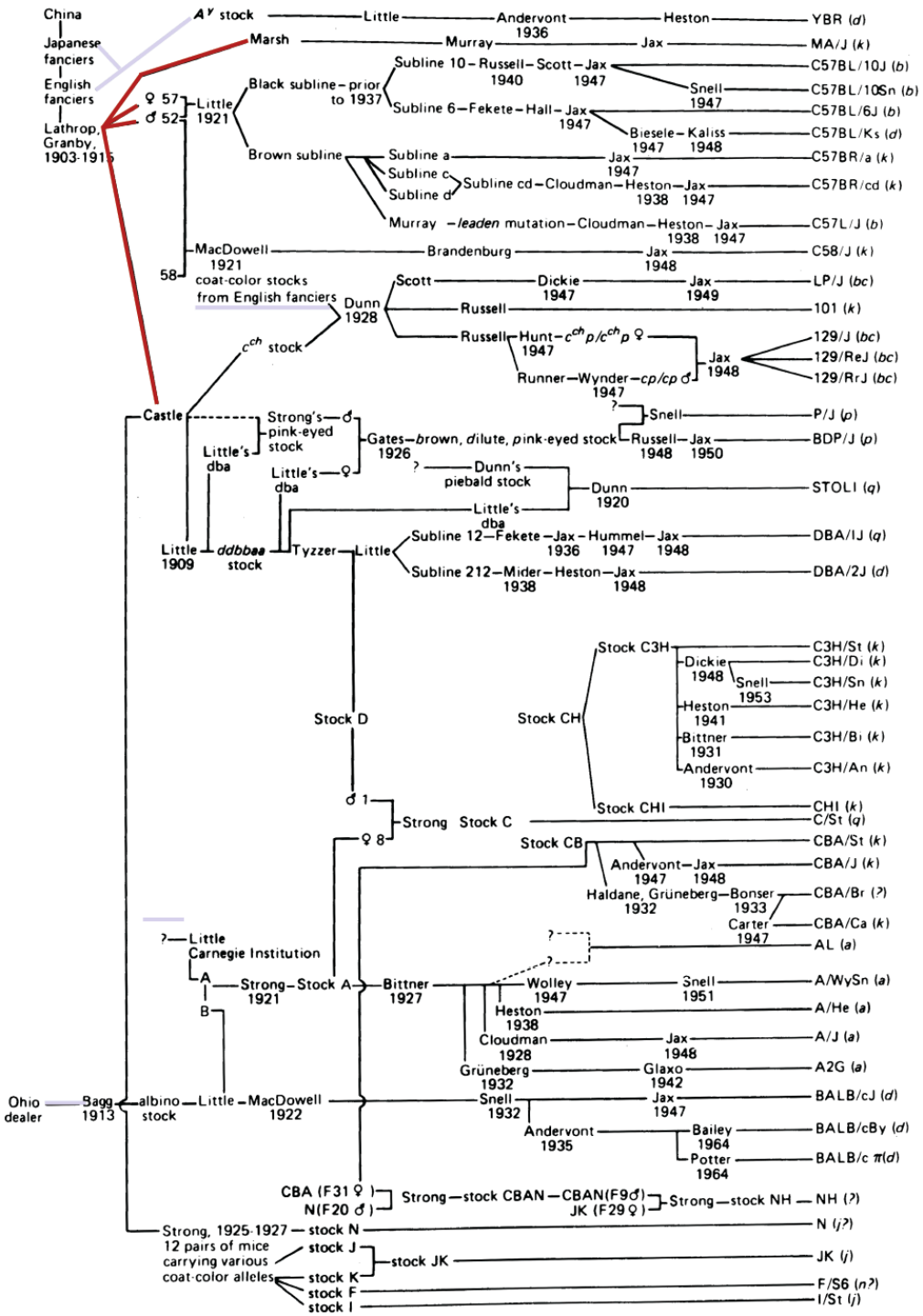

Genes
Environment
Physiology

What are Laboratory Mice?

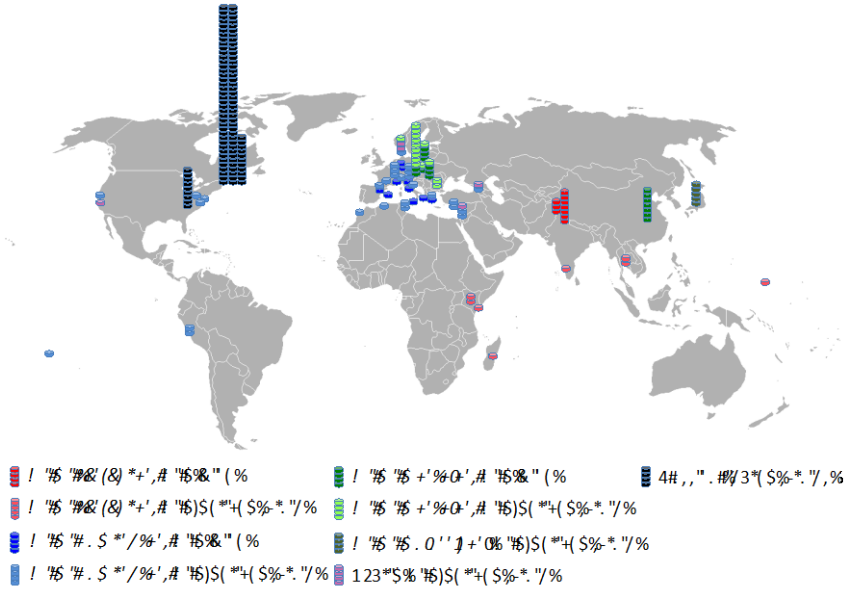
GURE 5. Miss Abbie E.C. Lathrop at Granby. Redrawn
he Springfield Sunday Republican, October 5, 1913.

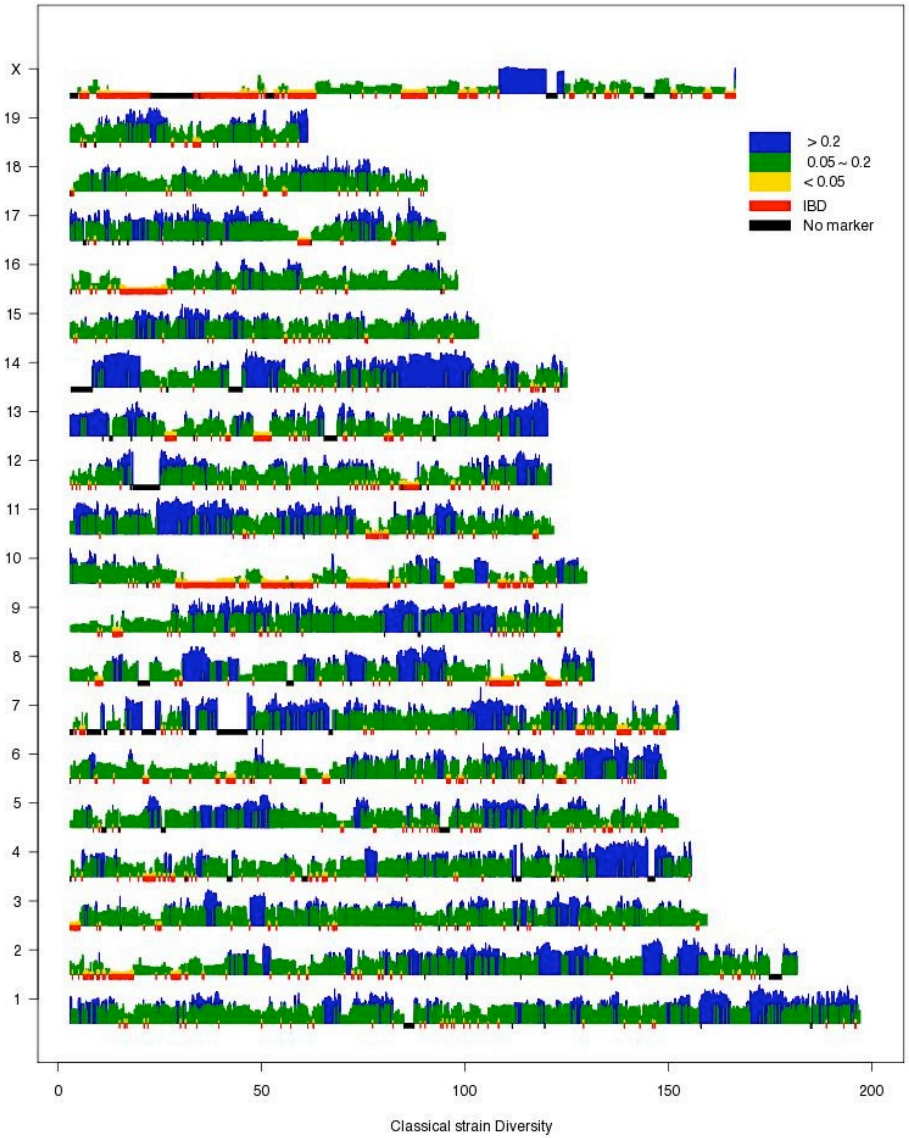
Abbie Lathrop - Pet vendor who supplied animals that founded many inbred strains

The market price varied according to the age of the mice, their color and other points, ranging from $10 to $20 per hundred. (Morse, 1978)



Genetic Diversity in 100 Inbred Strains





Yang et al Nat. Gen. 2007, 2011

Genomic responses in mouse models poorly mimic human inflammatory diseases

Junhee Seok[a,1], H. Shaw Warren[b,1], Alex G. Cuenca[c,1], Michael N. Mindrinos[a], Henry V. Baker[c], Weihong Xu[a], Daniel R. Richards[d], Grace P. McDonald-Smith[e], Hong Gao[a], Laura Hennessy[f], Celeste C. Finnerty[g], Cecilia M. López[c], Shari Honari[f], Ernest E. Moore[h], Joseph P. Minei[i], Joseph Cuschieri[j], Paul E. Bankey[k], Jeffrey L. Johnson[h], Jason Sperry[l], Avery B. Nathens[m], Timothy R. Billiar[l], Michael A. West[n], Marc G. Jeschke[o], Matthew B. Klein[j], Richard L. Gamelli[p], Nicole S. Gibran[j], Bernard H. Brownstein[q], Carol Miller-Graziano[k], Steve E. Calvano[r], Philip H. Mason[e], J. Perren Cobb[s], Laurence G. Rahme[t], Stephen F. Lowry[r,2], Ronald V. Maier[j], Lyle L. Moldawer[c], David N. Herndon[g], Ronald W. Davis[a,3], Wenzhong Xiao[a,t,3], Ronald G. Tompkins[t,3], and the Inflammation and Host Response to Injury, Large Scale Collaborative Research Program[4]

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Genomic responses in mouse models greatly mimic human inflammatory diseases

Keizo Takao[a,b] and Tsuyoshi Miyakawa[a,b,c,1]

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Edited by Ruslan Medzhitov, Yale University School of Medicine, New Haven, CT, and approved June 11, 2014 (received for review January 31, 2014)

The use of mice as animal models has long been considered essential in modern biomedical research, but the role of mouse models in research was challenged by a recent report that genomic responses in mouse models poorly mimic human inflammatory diseases. Here we reevaluated the same gene expression datasets used in the previous study by focusing on genes whose expression levels were significantly changed in both humans and mice. Contrary to the previous findings, the gene expression levels in the mouse models showed extraordinarily significant correlations with those of the human conditions (Spearman's rank correlation

to the stimulus would generally decrease the sensitivity to detect the responses shared by the disorders and their models. For this reason, we excluded such genes from our analysis. Second, we compared each of the conditions in a single mouse study independently with the human reference conditions. Mouse studies, such as GSE7404 and GSE19668, included multiple conditions or gene sets. For example, GSE19668 contains multiple datasets, including those for two different mouse strains and multiple time-course data points after infection. Because humans and mice are expected to be quite different, the optimal conditions/parameters

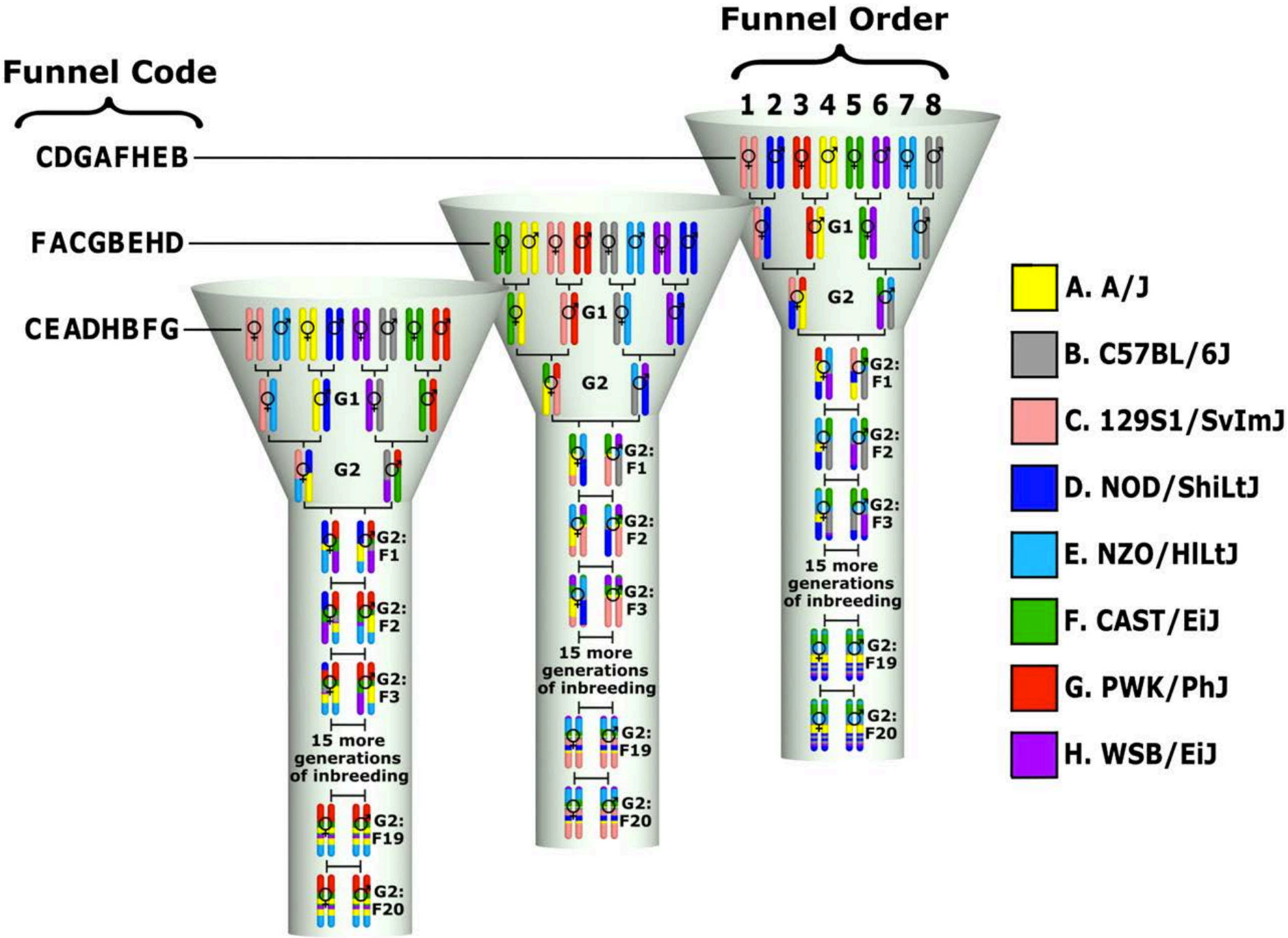
Why didn’t I see this adverse effect in my mouse model?

Our monogamous(e) relationship with C57BL/6J.

The Collaborative Cross

Founder Strains of the CC

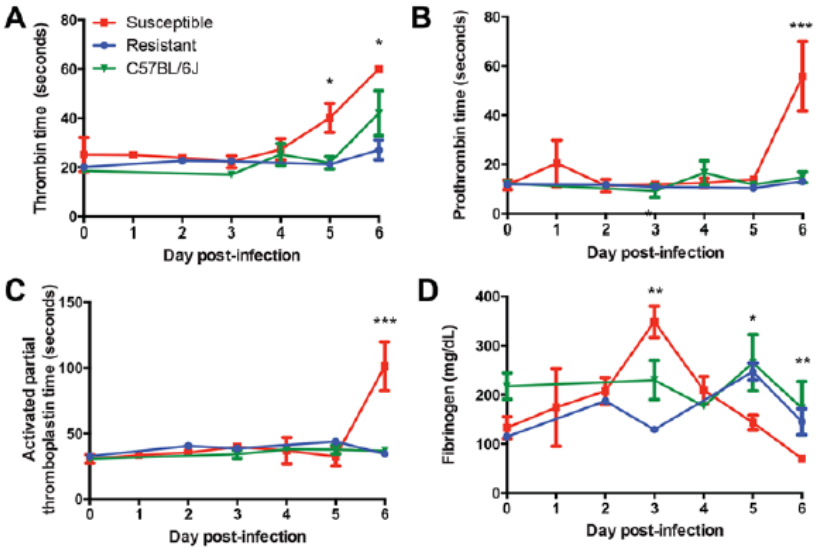
Brynn Voy


Funnel Code
Funnel Order
1 2 3 4 5 6 7 8
CDGAFHEB
FACGBEHD
CEADHBFG
G1
G2
G2: F1
G2: F2
G2: F3
15 more generations of inbreeding
G2: F19
G2: F20
A. A/J
B. C57BL/6J
C. 129S1/SvImJ
D. NOD/ShiLtJ
E. NZO/HILtJ
F. CAST/EiJ
G. PWK/PhJ
H. WSB/EiJ

Sciencexpress

Host genetic diversity enables Ebola hemorrhagic fever pathogenesis and resistance

Angela L. Rasmussen,*[1] Atsushi Okumura,*[1,4] Martin T. Ferris,[2] Richard Green,[1] Friederike Feldmann,[3] Sara M. Kelly,[1] Dana P. Scott,[3] David Safronetz,[4] Elaine Haddock,[4] Rachel LaCasse,[3] Matthew J. Thomas,[1] Pavel Sova,[1] Victoria S. Carter,[1] Jeffrey M. Weiss,[1] Darla R. Miller,[2] Ginger D. Shaw,[2] Marcus J. Korth,[1] Mark T. Heise,[2,5] Ralph S. Baric,[5] Fernando Pardo-Manuel de Villena,[2] Heinz Feldmann,[4] Michael G. Katze[1]†

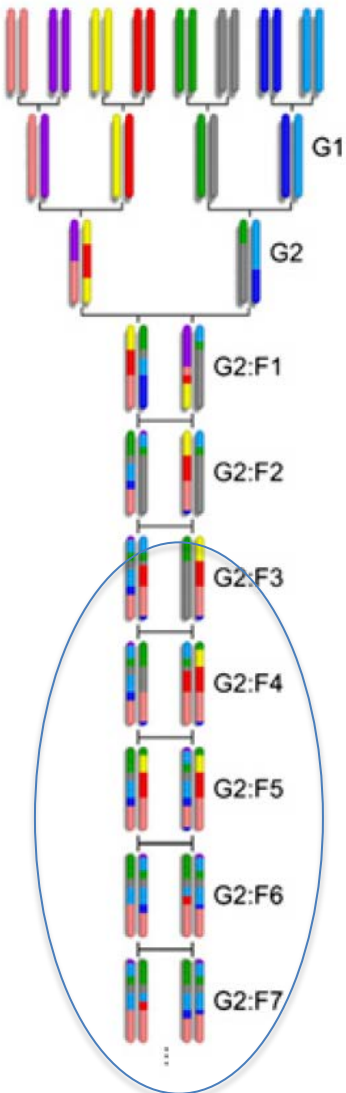


Existing mouse models of lethal Ebola virus infection do not reproduce hallmark symptoms of Ebola hemorrhagic fever, neither delayed blood coagulation and disseminated intravascular coagulation, nor death from shock, thus restricting pathogenesis studies to non-human primates. Here we show that mice from the Collaborative Cross exhibit distinct disease phenotypes following mouse-adapted Ebola virus infection. Phenotypes range from complete resistance to lethal disease to severe hemorrhagic fever characterized by prolonged coagulation times and 100% mortality. Inflammatory signaling was associated with vascular permeability and endothelial activation, and resistance to lethal infection arose by induction of lymphocyte differentiation and cellular adhesion, likely mediated by the susceptibility allele *Tek*. These data indicate that genetic background determines susceptibility to Ebola hemorrhagic fever.

Diversity Outbred Mice

The Diversity Outbred (DO) heterogeneous stock.

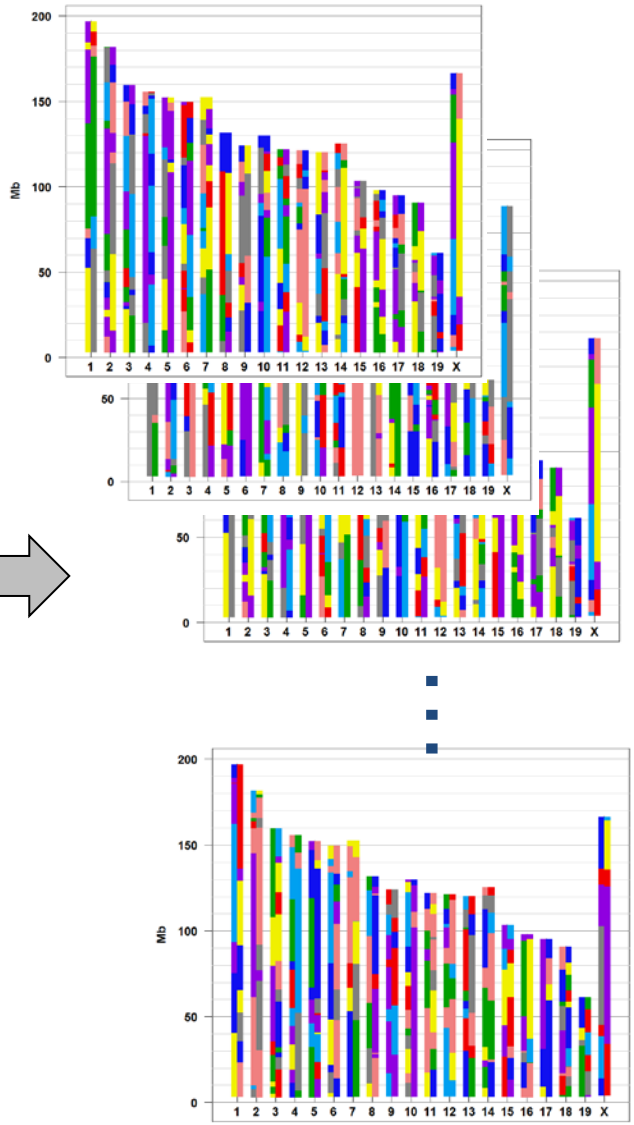
Collaborative Cross Funnel



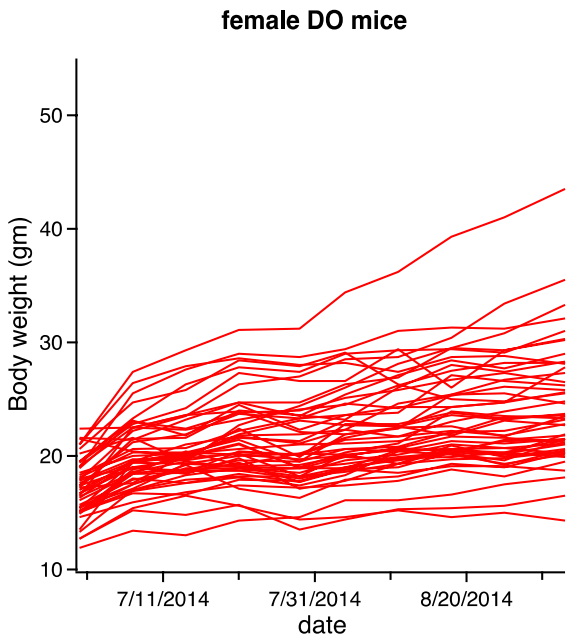
Pre-CC mice from 144 different funnels

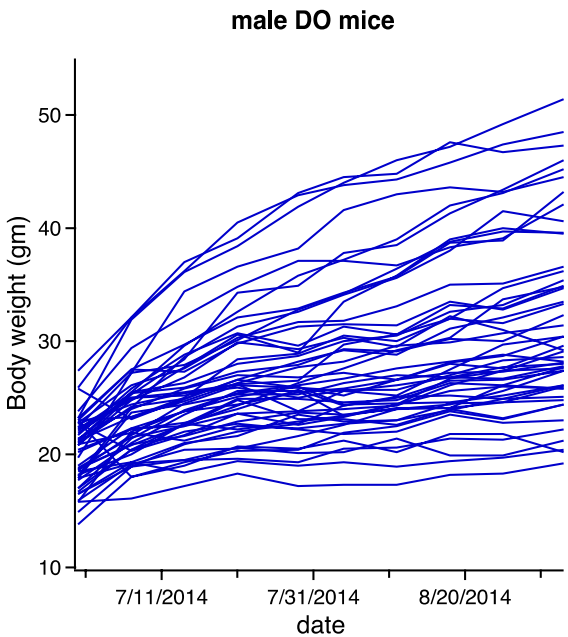
Random Outbreeding

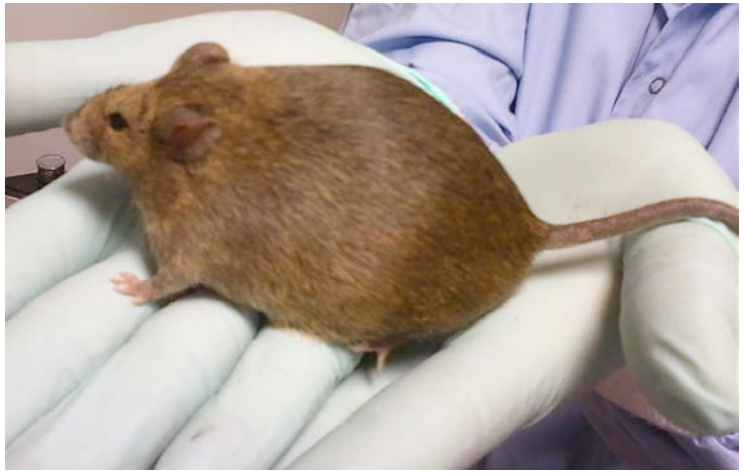
Diversity Outbred

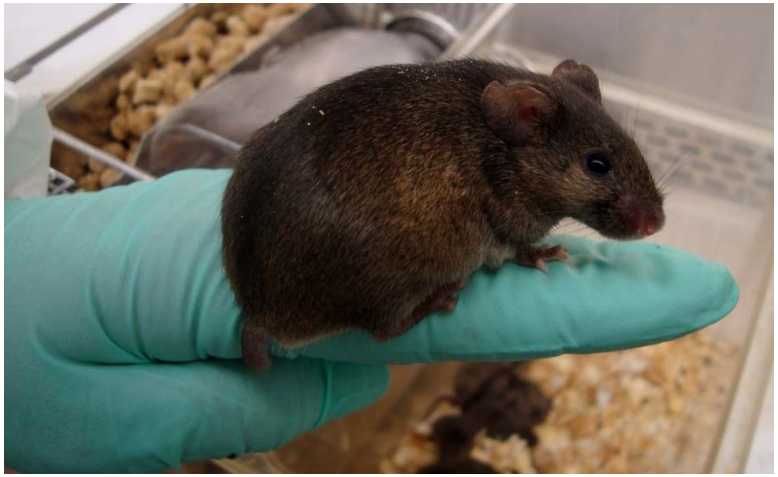


DO are genetically and phenotypically diverse

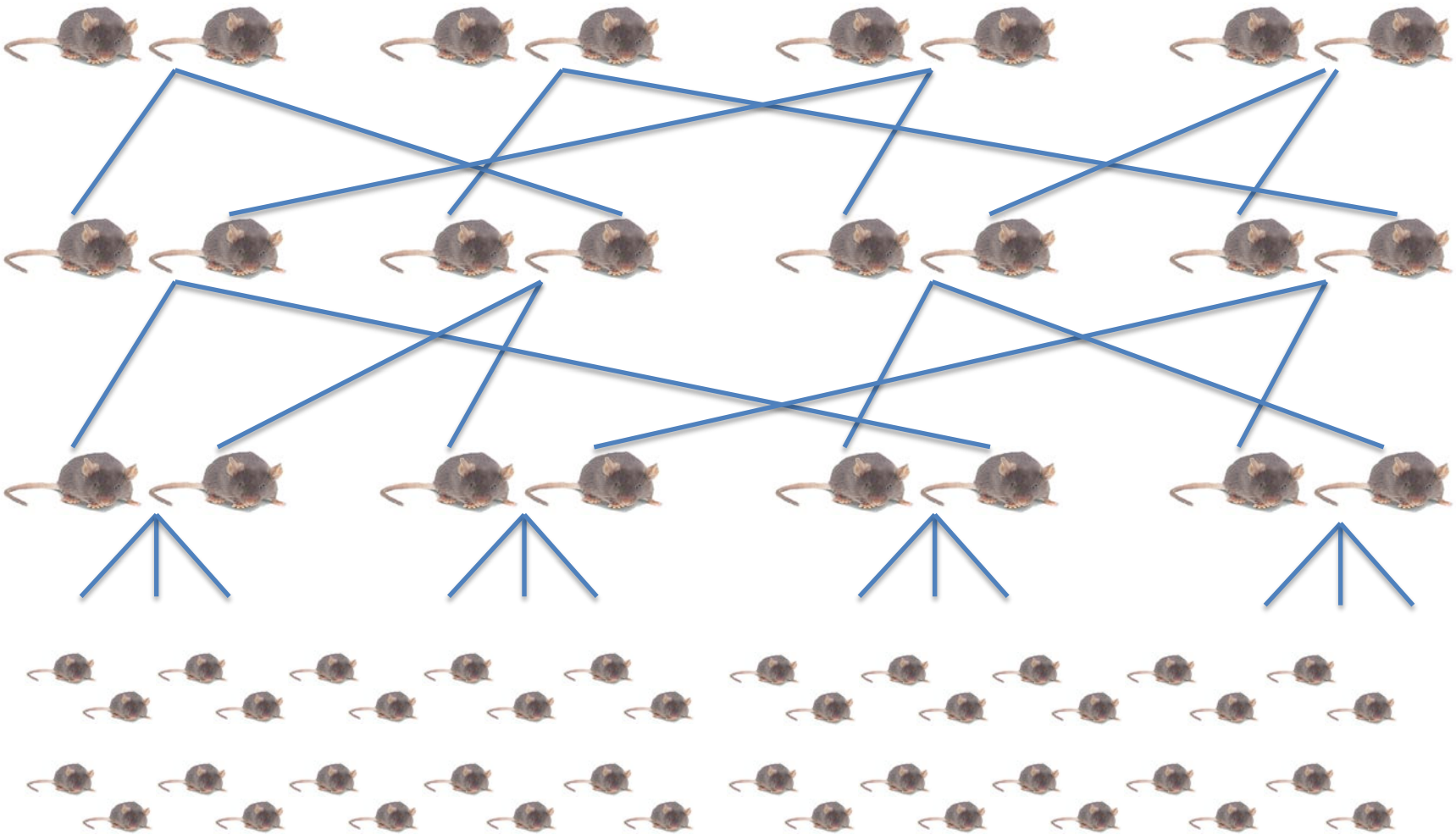




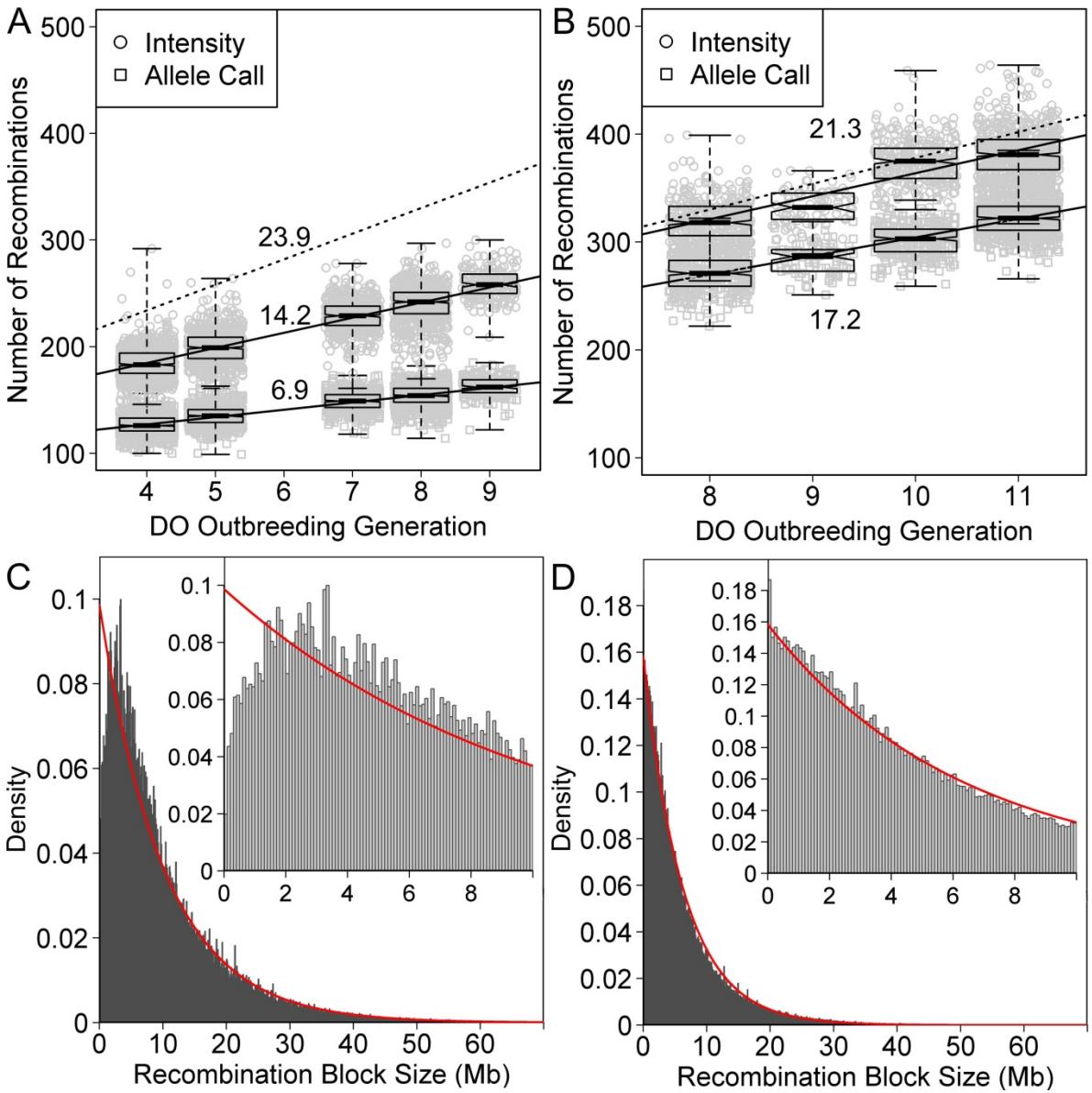




Randomized Outcrossing & Balanced Selection



Accumulation of Recombination in DO Mice



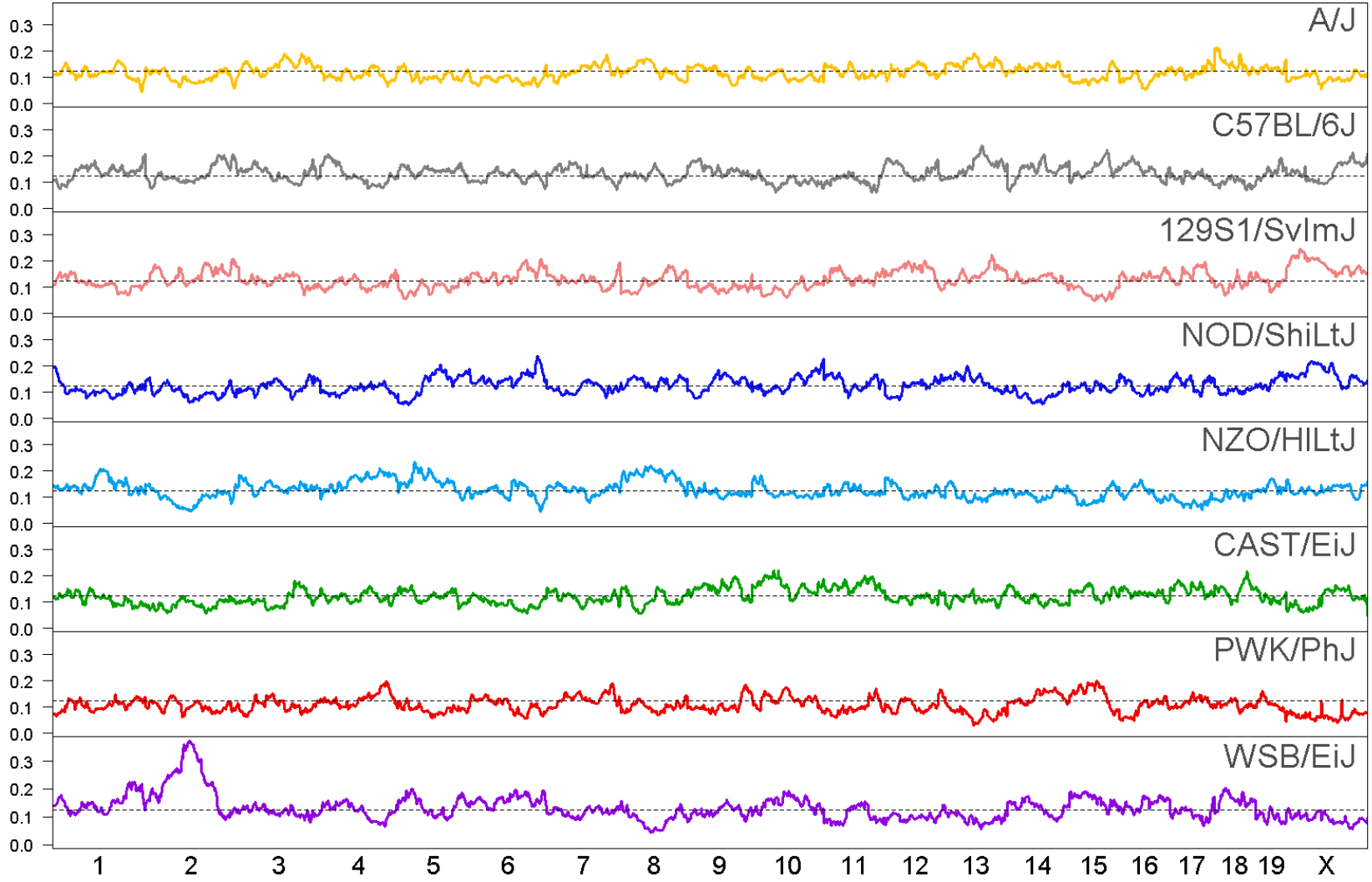
Transmission Ratio Distortion in the DO

Elissa Chesler

Dan Gatti

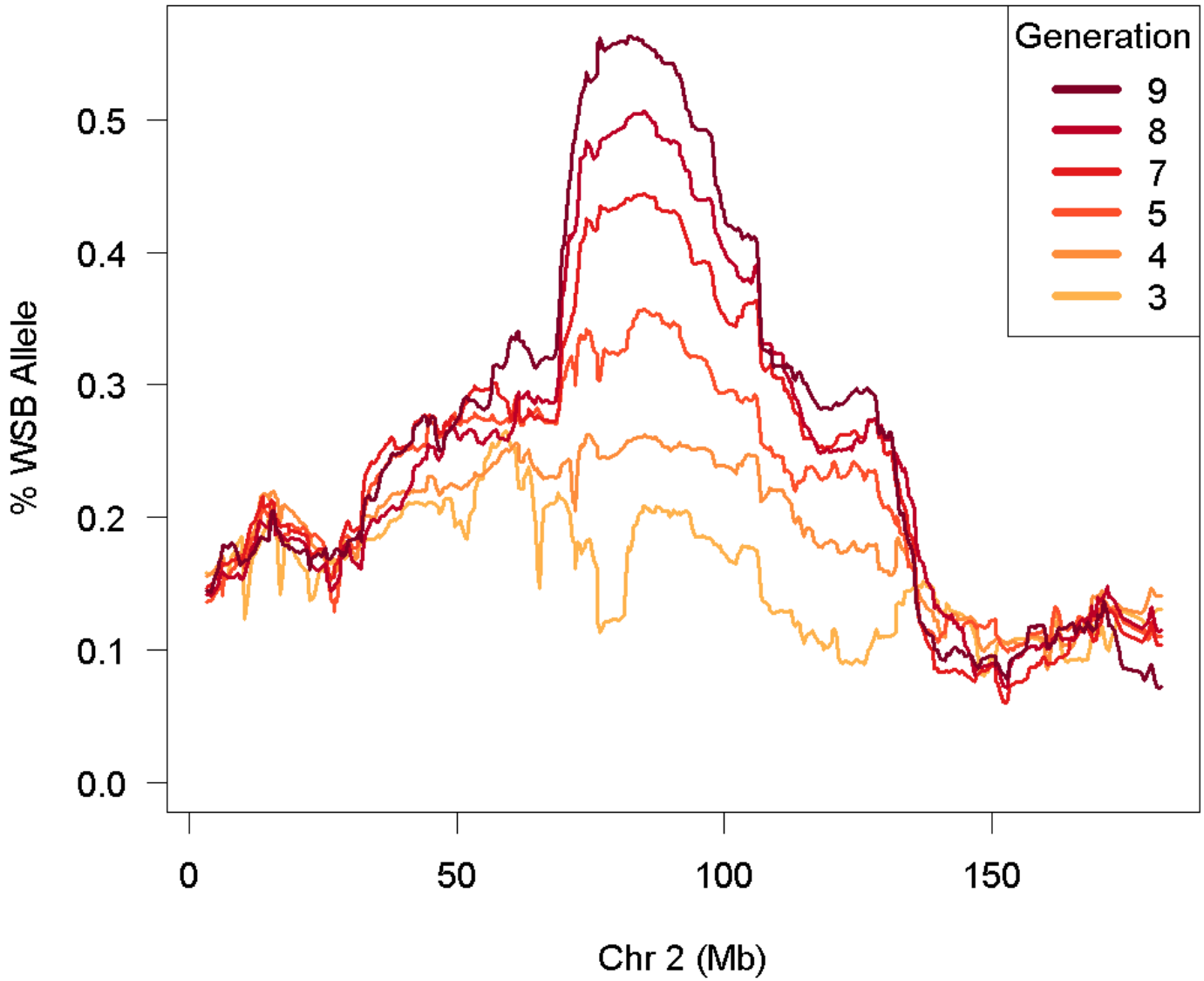
Fernando

Founder allele frequencies across the genome



Generations 3 – 9, averaged

The WSB allele on Chr 2 was increasing with each generation



G12 x G12 350 pairs

♂ \ ♀	WW	Wa	aa
WW	4	4	3
Wa	4	2c	2a
aa	3	2b	1

④ no genotyping ship

③ all Wa no genotyping use only as needed ship rest

② genotype + hold select aa use Wa if none

① no genotyping ship extra

production breeders → 175 production pairs

randomization pool F 175 M 175 F → 175 Core Colony Pairs

Any Wa matings will need progeny typing to proceed to G14 mating

G13 x G13

Za > Zb > Zc

50% 40% 20%

Fixing the DO in two generations!

Table 1: Expected frequencies of the various mating types in the DO population at the G12 intervention.

			Female			
			WW	Wa	aa	
			0.36	0.48	0.16	
	WW	0.36	**0.1296**	**0.1728**	**0.0576**	0.36
Male	Wa	0.48	**0.1728**	**0.2304**	**0.0768**	0.48
	aa	0.16	**0.0576**	**0.0768**	**0.0256**	0.16
			0.36	0.48	0.16	1

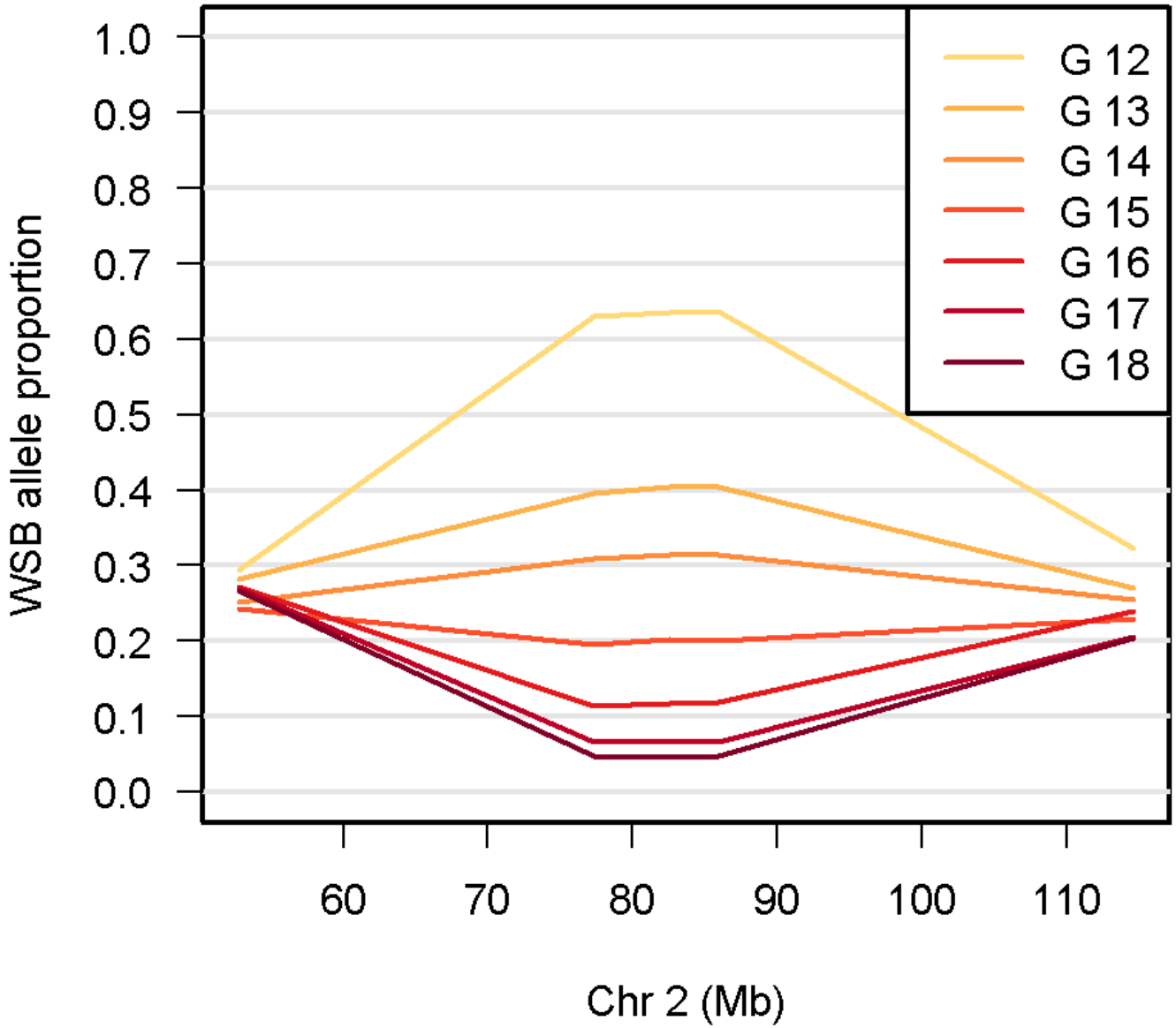
Table 2: First generation intervention

		Female		
		WW (.36)	Wa (.48)	Aa (.16)
	WW (.36)	~~WW~~	~~WW~~ Wa*	Wa
Male	Wa (.48)	~~WW~~ Wa*	~~WW~~ ~~Wa~~ aa	~~Wa~~ aa
	aa (.16)	Wa	~~Wa~~ aa	aa

Table 3: Second generation intervention

		Female	
		Wa (.35)	aa (.65)
Male	Wa (.35)	~~WW~~ ~~Wa~~ aa	~~Wa~~ aa
	aa (.65)	~~Wa~~ aa	aa

Purging the WSB allele



PLOS | GENETICS

RESEARCH ARTICLE

A Multi-Megabase Copy Number Gain Causes Maternal Transmission Ratio Distortion on Mouse Chromosome 2

John P. Didion[1,2,3], Andrew P. Morgan[1,2,3], Amelia M.-F. Clayshulte[1,2,3], Rachel C. Mcmullan[1,2,3], Liran Yadgary[1], Petko M. Petkov[4], Timothy A. Bell[1,2,3], Daniel M. Gatti[4], James J. Crowley[1], Kunjie Hua[1,2,3,5], David L. Aylor[6], Ling Bai[7], Mark Calaway[1,2,3], Elissa J. Chesler[4], John E. French[8], Thomas R. Geiger[7], Terry J. Gooch[1,2,3], Theodore Garland Jr.[9], Alison H. Harrill[10], Kent Hunter[7], Leonard McMillan[11], Matt Holt[11], Darla R. Miller[1,2,3], Deborah A. O'Brien[2,5], Kenneth Paigen[4], Wenqi Pan[2,5], Lucy B. Rowe[4], Ginger D. Shaw[1,2,3], Petr Simecek[4], Patrick F. Sullivan[1], Karen L. Svenson[4], George M. Weinstock[12], David W. Threadgill[13], Daniel Pomp[1,2,3,5], Gary A. Churchill[4], Fernando Pardo-Manuel de Villena[1,2,3]*

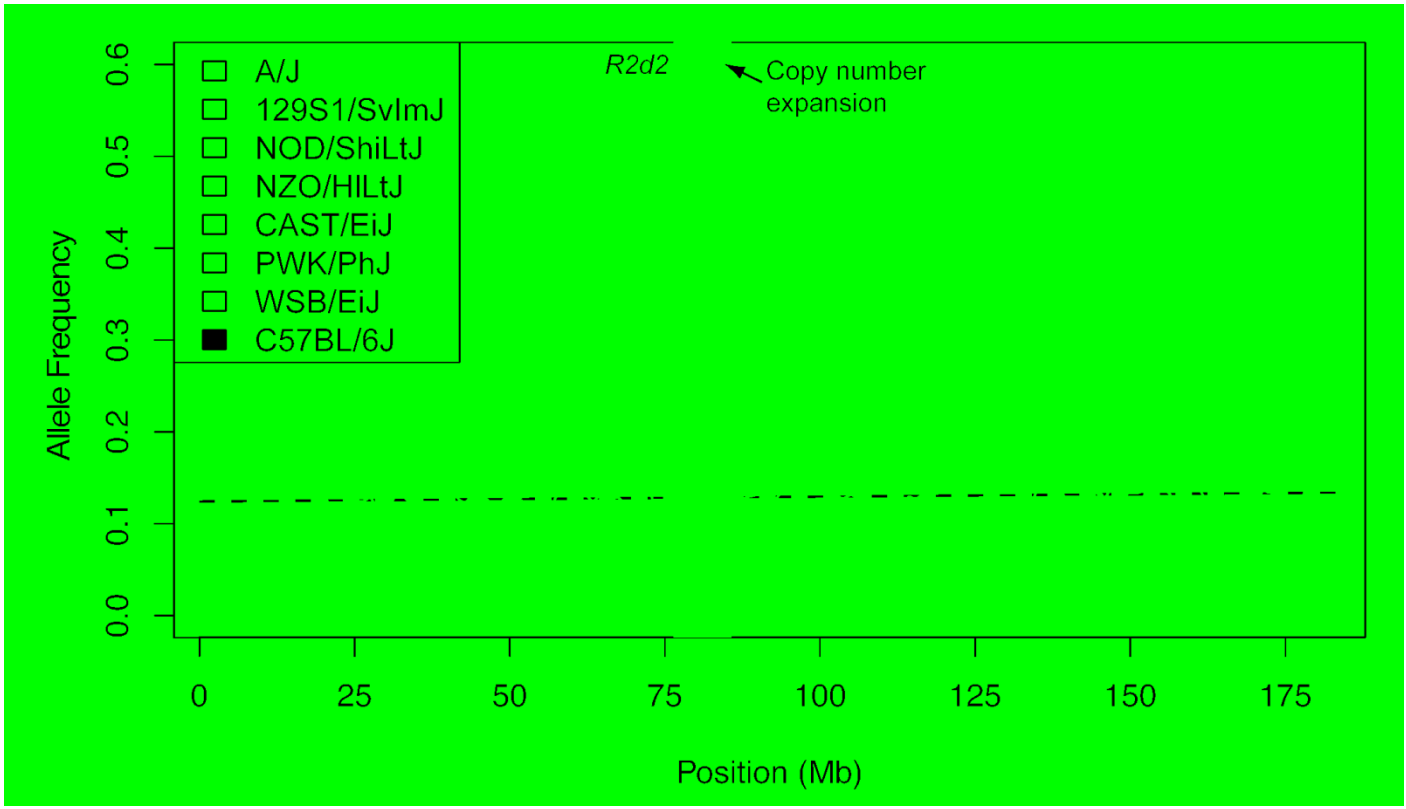
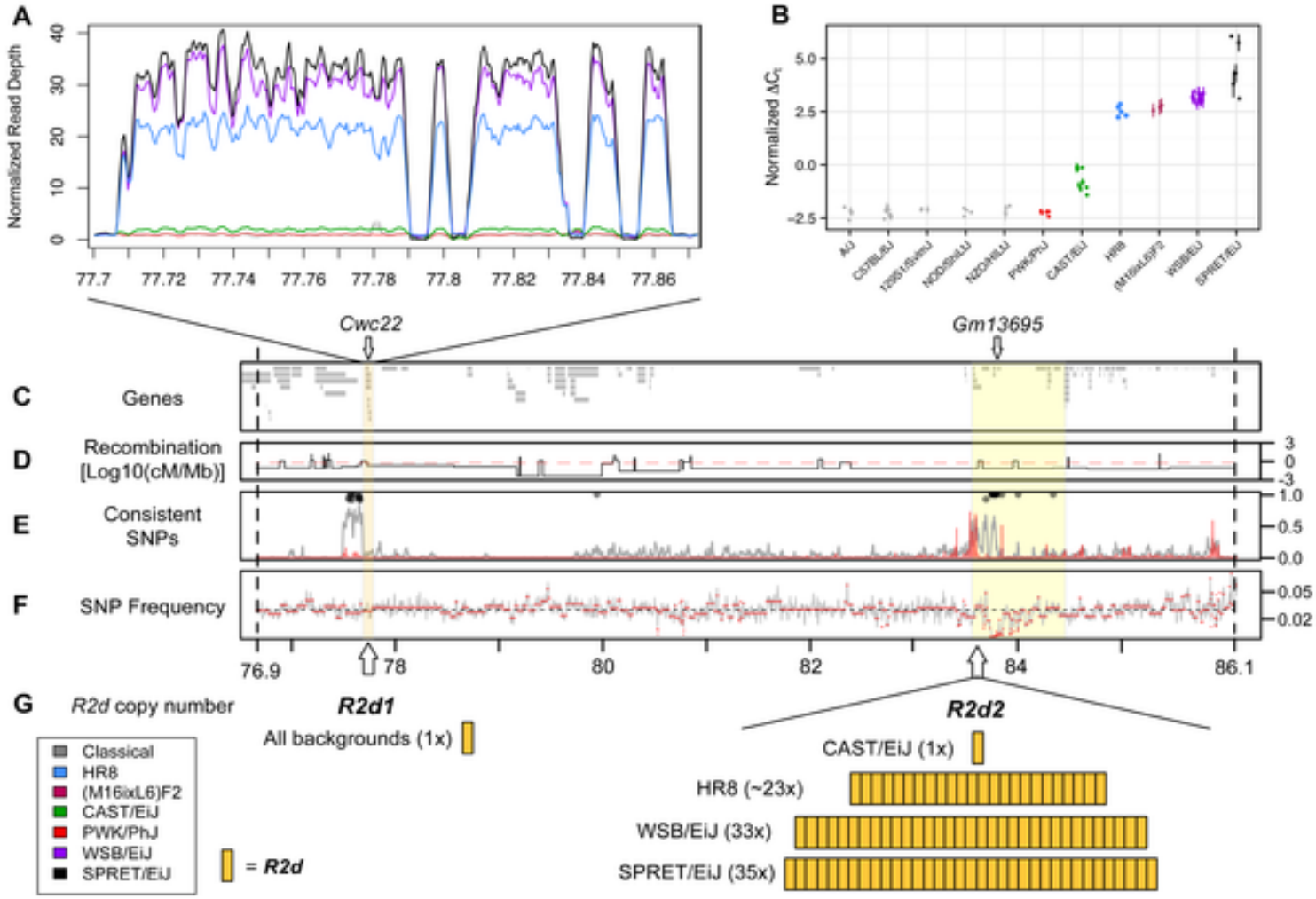


Figure 2. A large copy number gain is present in strains with maternal TRD.



Didion JP, Morgan AP, Clayshulte AMF, Mcmullan RC, Yadgary L, et al. (2015) A Multi-Megabase Copy Number Gain Causes Maternal Transmission Ratio Distortion on Mouse Chromosome 2. PLoS Genet 11(2): e1004850. doi:10.1371/journal.pgen.1004850
http://127.0.0.1:8081/plosgenetics/article?id=info:doi/10.1371/journal.pgen.1004850

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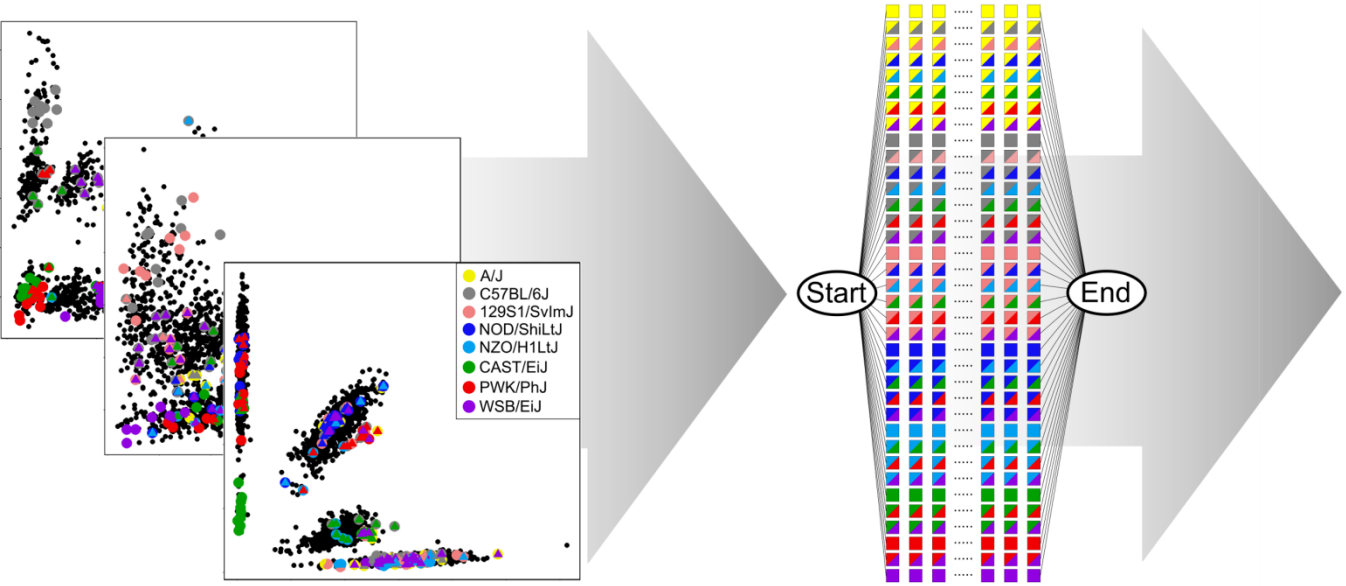
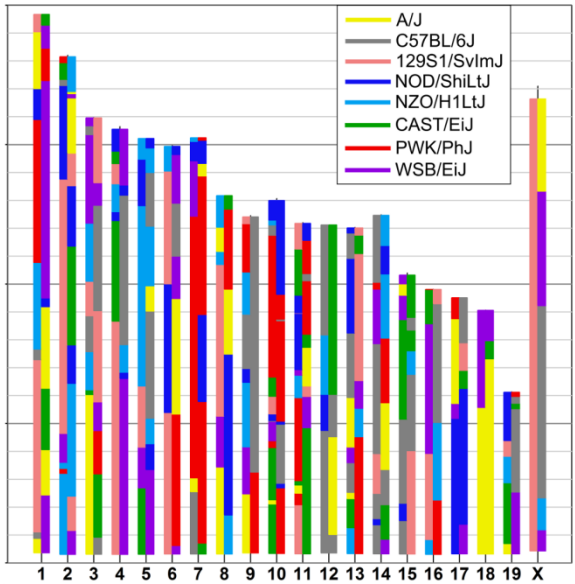
DO QTL

Genetic Mapping in the DO





Genotyping Arrays

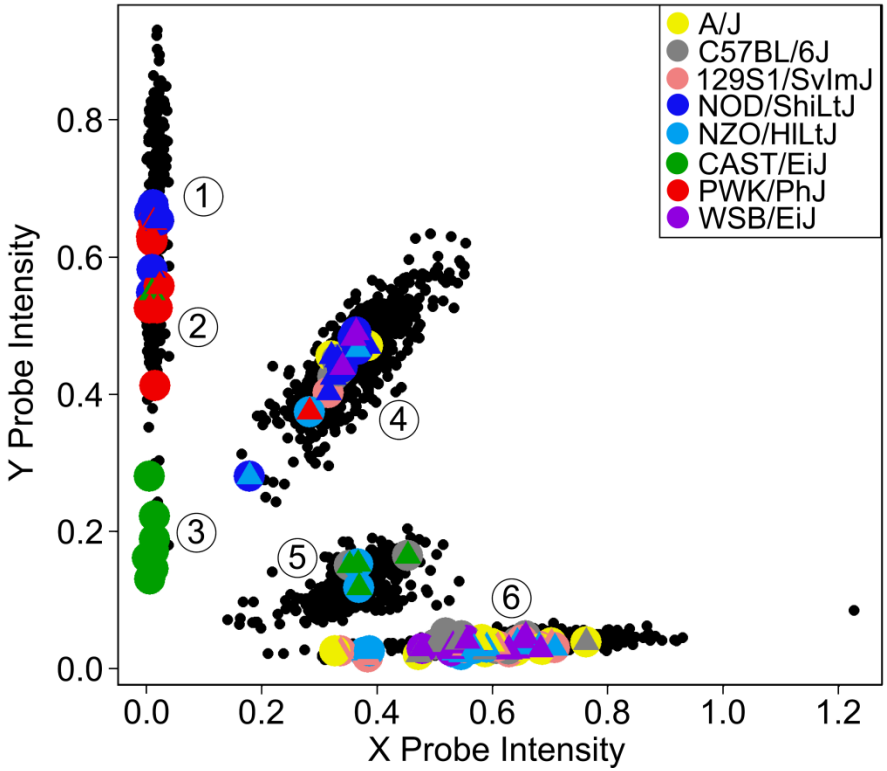
Hidden Markov Model

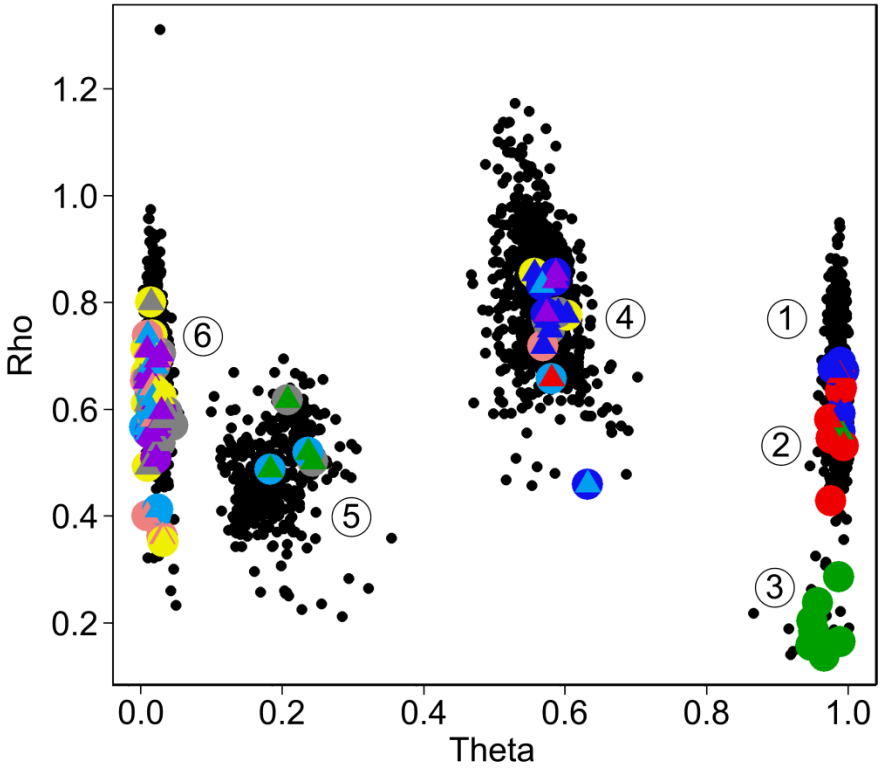
Genome Reconstruction

Linkage and Association:

$$y_i = \sum_{j=1}^{c} x_{ij}\alpha_j + \sum_{k=1}^{8} g_{ik}\beta_k + \gamma_i + \varepsilon_i$$

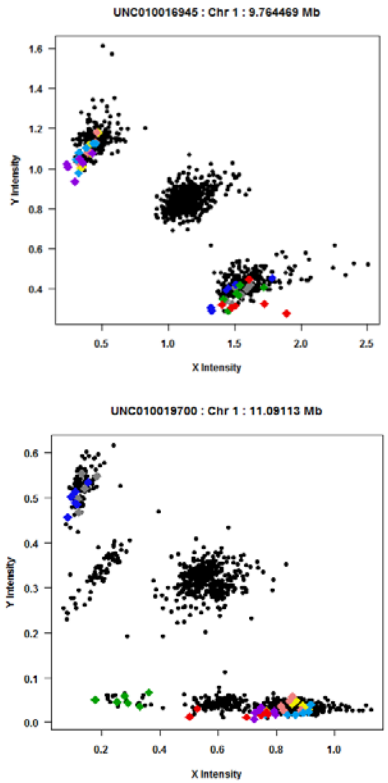
High-density Array Genotyping





Building an individualized diploid genome.

Genotype DNA @ 8-80k informative SNPs



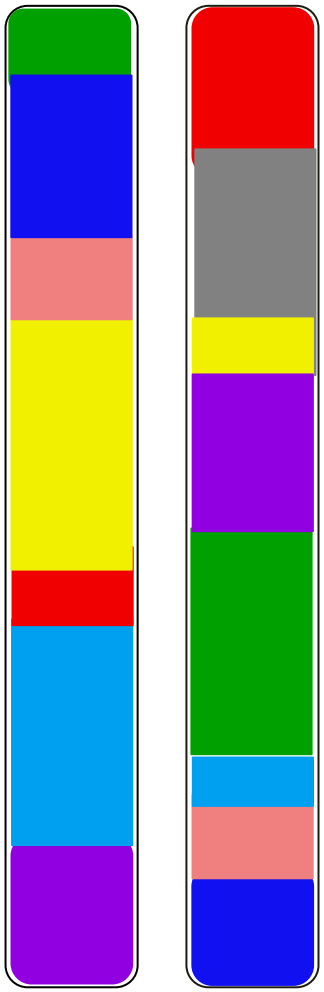
Infer 36-state genotypes

FG
FG
DG
DG
DB
DB
DB
CB
CB
AA
AA
AH
AH
AH
AH
AF
GF
GF
EF
EF
EF
EE
EE
EC
HC
HC
HD
HD

Psuedo-phased Chromosomes

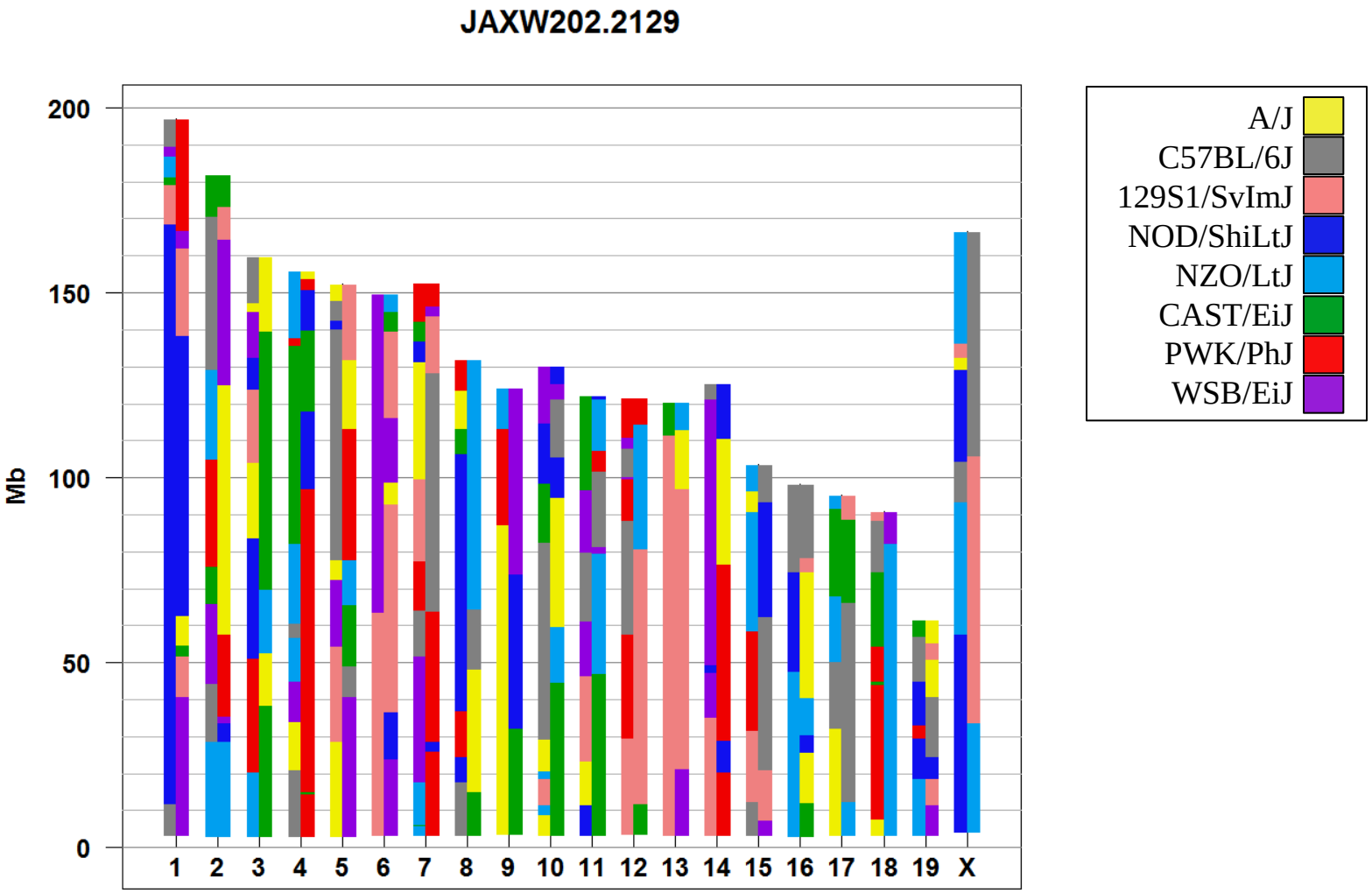
F	G
F	G
D	G
D	G
D	B
D	B
D	B
C	B
C	B
A	A
A	A
A	H
A	H
A	H
A	H
A	F
G	F
G	F
E	F
E	F
E	F
E	E
E	C
H	C
H	C
H	D
H	D

Two Haploid Chromosomes



Munger et al., *submitted*

Reconstructed DO Genome



8 Haplotype States

A/J A	BL6 B	129 C	NOD D	NZO E	CAST F	PWK G	WSB H

36 Diplotype States

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36
A	A	A	A	A	A	A	A	B	B	B	B	B	B	B	C	C	C	C	C	C	D	D	D	D	D	E	E	E	E	F	F	F	G	G	H
A	B	C	D	E	F	G	H	B	C	D	E	F	G	H	C	D	E	F	G	H	D	E	F	G	H	E	F	G	H	F	G	H	G	H	H

Allelic Dosage

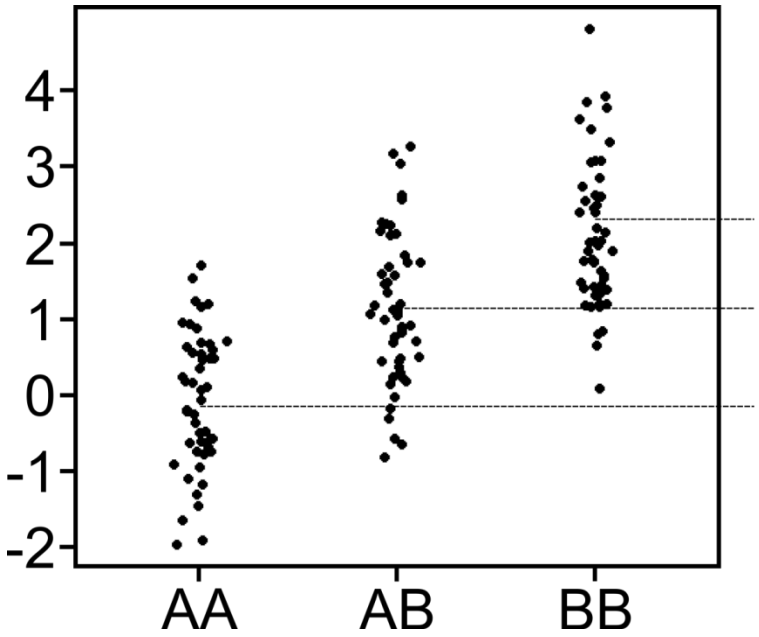
For each mouse at each SNP, we have 36 diplotype probabilities.

AA	AB	AC	AD	AE	AF	AG	AH	BB	BC	BD	BE	BF	BG	BH	CC	CD	CE	CF	CG	CH	DD	DE	DF	DG	DH	EE	EF	EG	EH	FF	FG	FH	GG	GH	HH
0	0	0	0	0	0	0	0	**1**	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	0	**1**	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0	**0.5**	**0.5**	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

These collapse to 8 founder allele dosages.

A	B	C	D	E	F	G	H
0	**1**	0	0	0	0	0	0
0	**0.5**	0	0	0	0	0	**0.5**
0	**0.5**	0	0	0	0	**0.25**	**0.25**

Additive Genetics Effects in an F2



$Y = \mu + 2\beta$

$Y = \mu + \beta$

$Y = \mu$

$Y = \mu + \beta g$

where g is “dosage” of the B allele

Additive Genetic Effects in DO

$$y_i = \beta_s s_i + \sum_{j=1}^{8} \beta_j g_{ij} + \gamma_i$$

y_i = phenotype of animal i

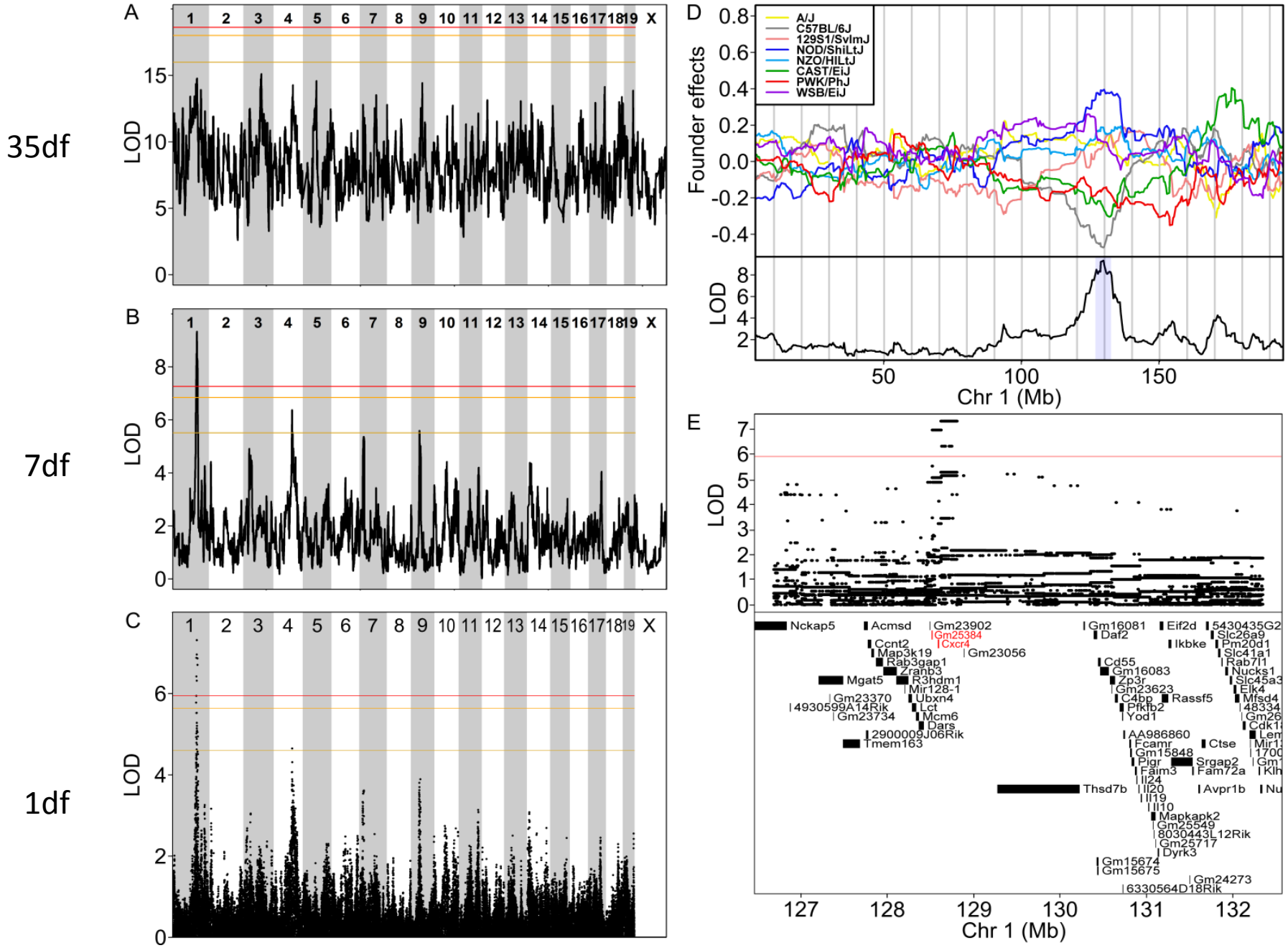
β_s = sex effect

s_i = sex of animal i

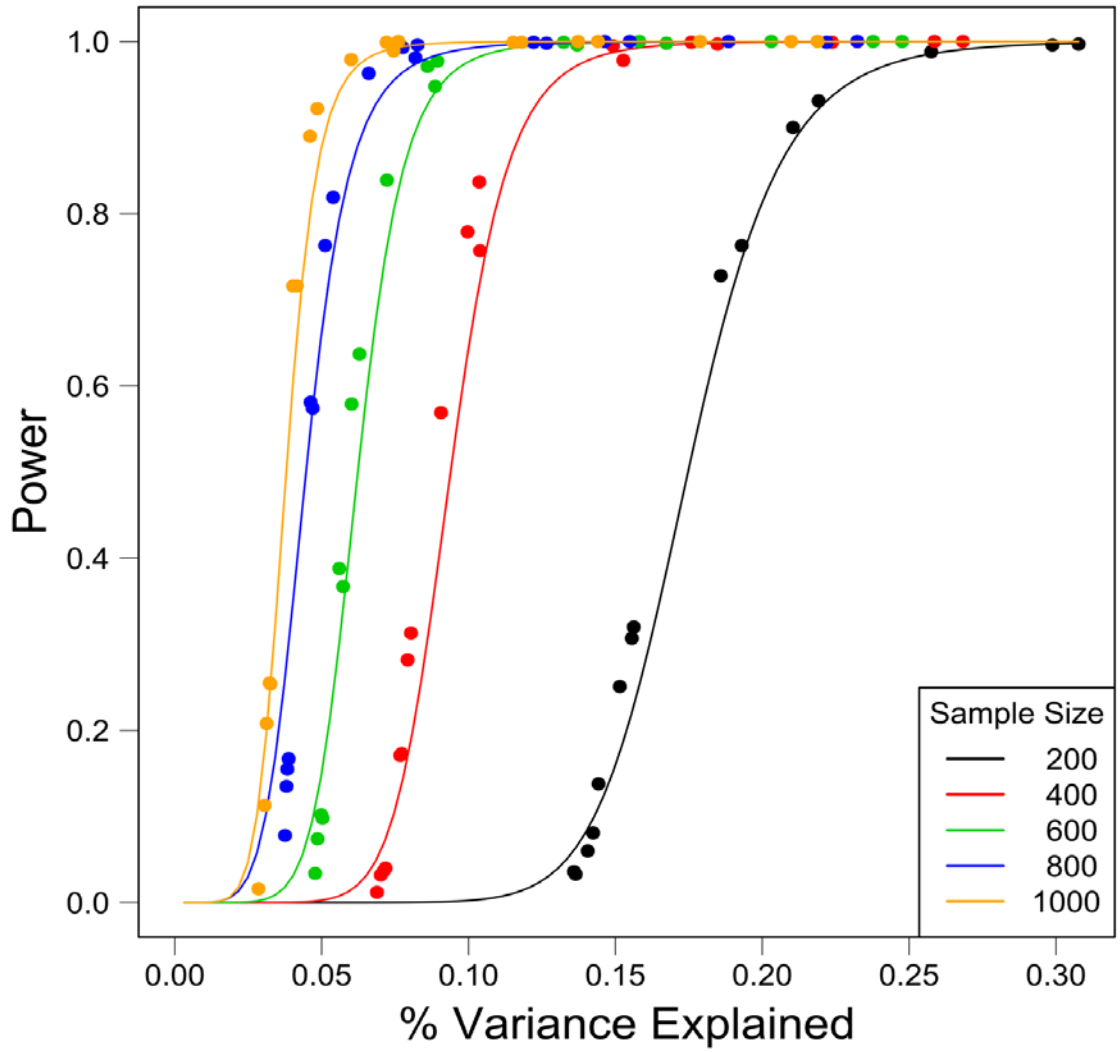
$\beta_{1\text{-}8}$ = founder effect

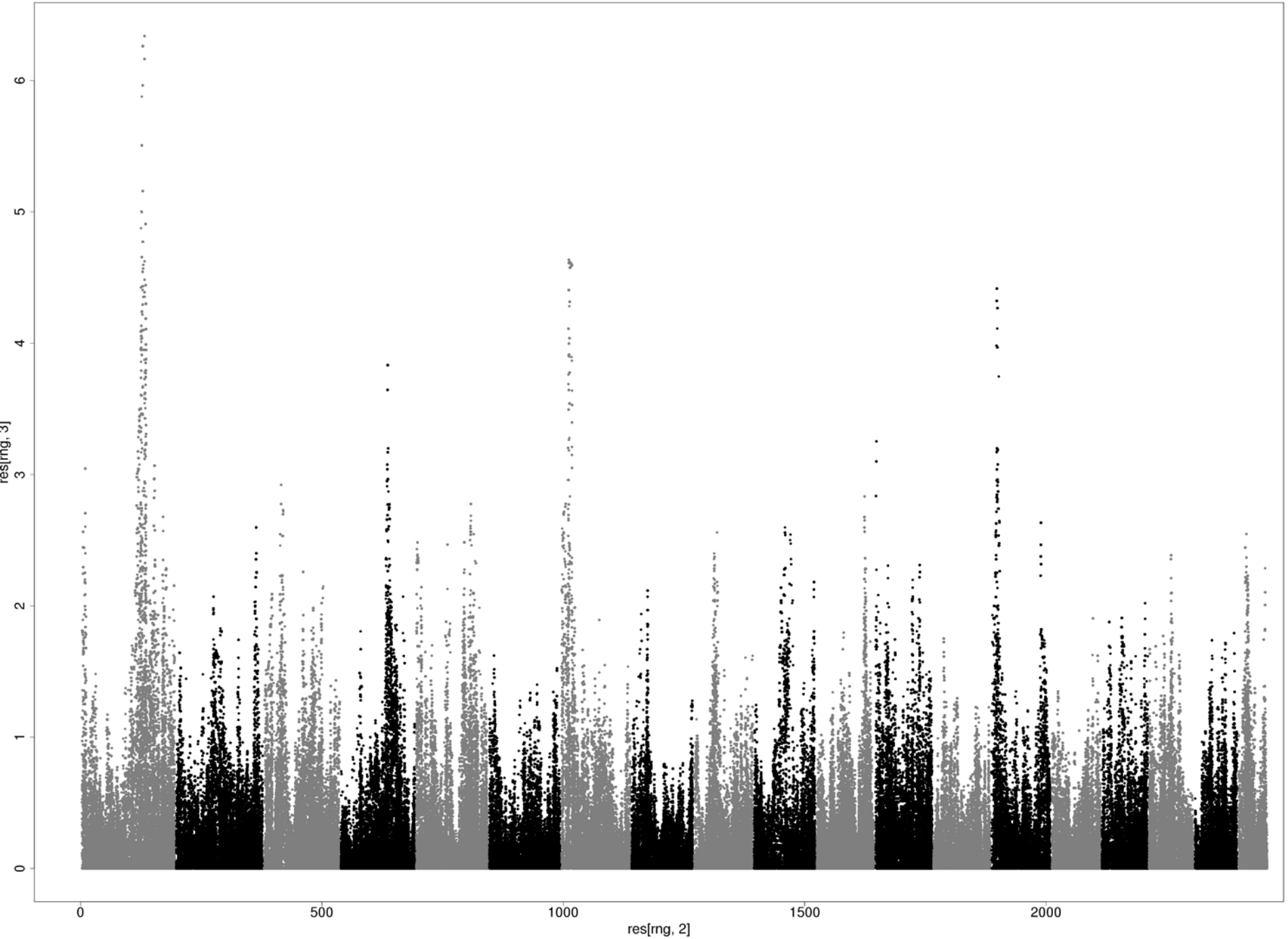
g_{ij} = dosage of haplotype j in animal i

γ = adjustment for population structure.


35df
7df
1df
A
B
C
D
E
LOD
Founder effects
A/J
C57BL/6J
129S1/SvImJ
NOD/ShiLtJ
NZO/HILtJ
CAST/EiJ
PWK/PhJ
WSB/EiJ
Chr 1 (Mb)
Nckap5
Acmsd
Gm23902
Gm25384
Cxcr4
Ccnt2
Map3k19
Gm23056
Rab3gap1
Zranb3
Mgat5
R3hdm1
Mir128-1
Gm23370
Ubxn4
4930599A14Rik
Lct
Gm23734
Mcm6
Dars
2900009J06Rik
Tmem163
Gm16081
Daf2
Eif2d
5430435G2
Slc26a9
Ikbke
Pm20d1
Slc41a1
Cd55
Rab7l1
Gm16083
Nucks1
Zp3r
Slc45a3
Gm23623
Elk4
C4bp
Rassf5
Mfsd4
Pfkfb2
Yod1
AA986860
Fcamr
Ctse
Gm15848
Pigr
Srgap2
Faim3
Fam72a
Il24
Thsd7b
Il20
Avpr1b
Il19
Il10
Mapkapk2
Gm25549
8030443L12Rik
Gm25717
Dyrk3
Gm15674
Gm15675
Gm24273
6330564D18Rik

How Many DO?


Power
1.0
0.8
0.6
0.4
0.2
0.0
0.00
0.05
0.10
0.15
0.20
0.25
0.30
% Variance Explained
Sample Size
200
400
600
800
1000


res[rng, 3]
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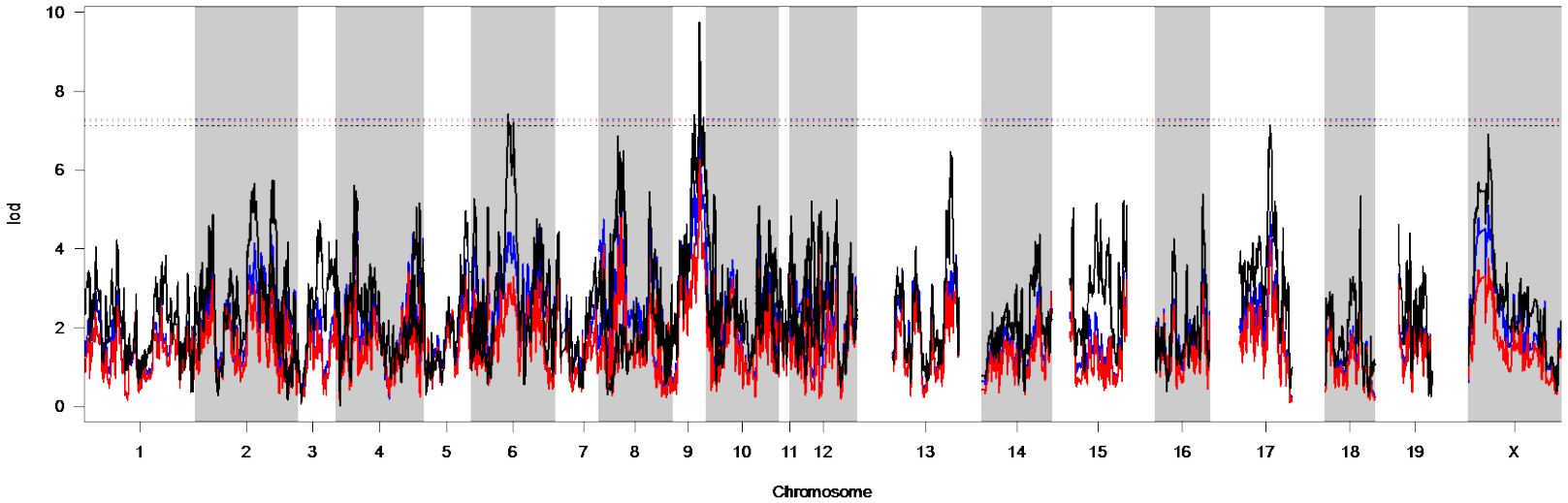
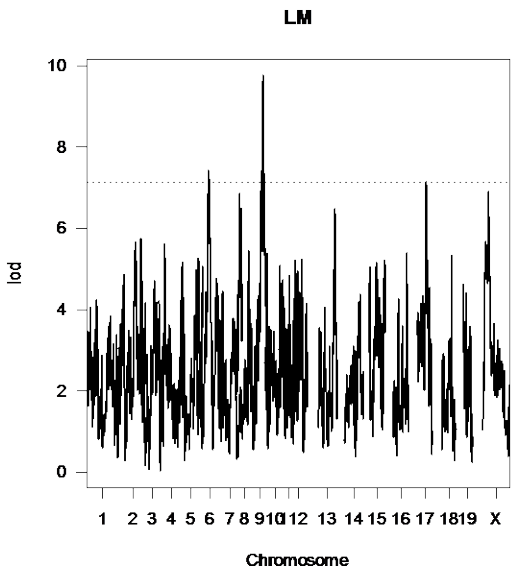
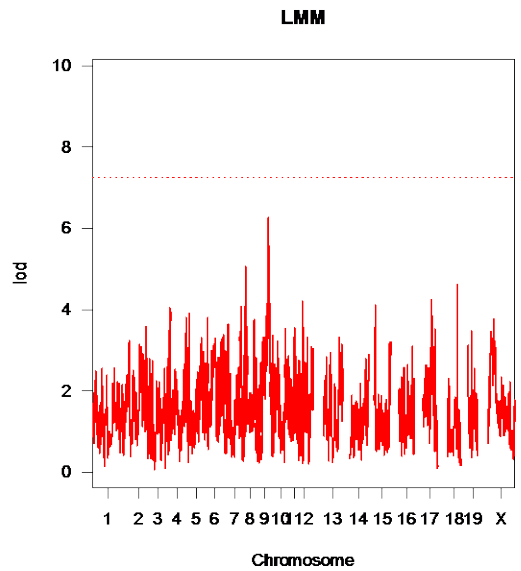
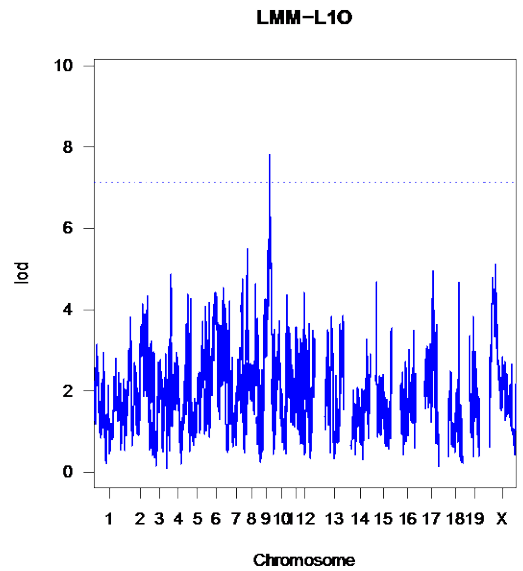
1
2
3
4
5
6
0
500
1000
1500
2000
res[rng, 2]

Impact of Kinship Correction on QTL Mapping



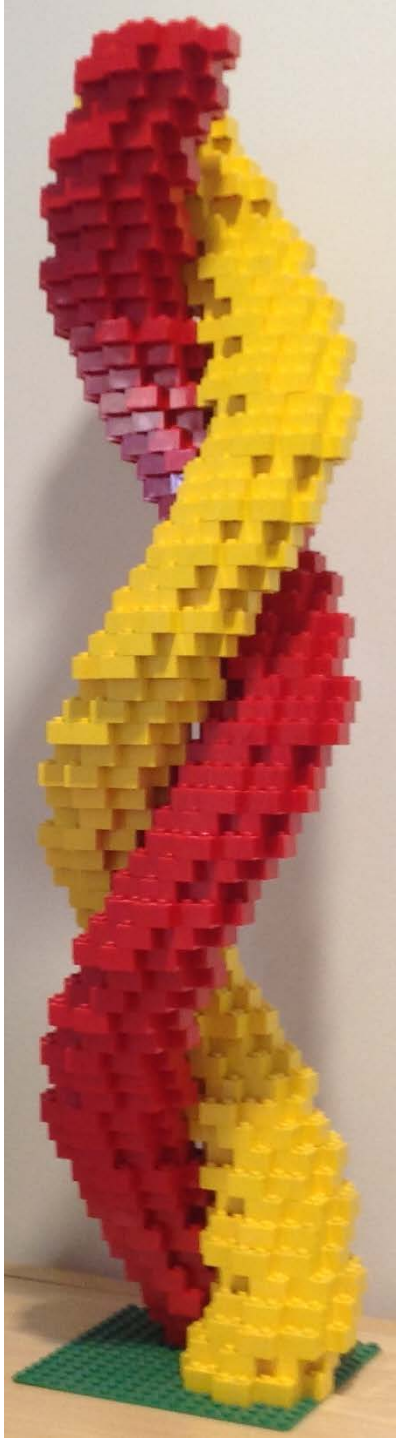






Missing Heritability

Petr Simecek



Linear mixed models for structured mapping populations

$$Y = X\beta + Zu + \varepsilon,$$

where $u \sim N\left(0, \sigma_g^2 K\right), \varepsilon \sim N(0, \sigma_e^2 I),$

K is a kinship matrix and I is an identity matrix.

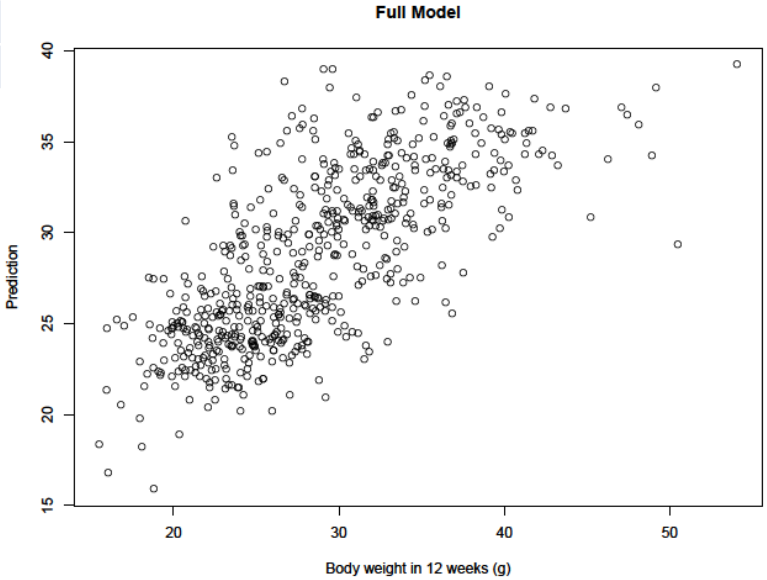
Heritability estimated as $\dfrac{\widehat{\sigma_g^2}}{\widehat{\sigma_g^2} + \widehat{\sigma_e^2}},$

Body Weight Heritability

Name	#missing	sigma_g^2	sigma_e^2	Heritability	beta(NZO)	SE(NZO)
BW.10	1	7.850698	10.302831	43%	31.667545	8.34738
BW.11	1	9.634533	11.4269019	46%	32.879816	9.019688
BW.12	1	10.383394	12.171026	46%	33.407795	9.337154
BW.13	1	9.8972628	13.3368905	43%	34.688206	9.435187
BW.14	4	11.552609	14.4687721	44%	32.649167	10.01988
BW.15	5	13.261251	15.5131729	46%	36.096457	10.58802
BW.16	5	15.636686	16.5555212	49%	35.745239	11.23048
BW.17	6	17.046866	18.0390107	49%	40.055002	11.73381
BW.18	7	19.932604	20.0998211	50%	39.700635	12.57485
BW.19	7	21.6743	21.5578365	50%	41.600712	13.05236
BW.20	7	22.919733	23.9057386	49%	36.261463	13.56802
BW.21	8	24.824493	25.1181201	50%	40.131266	14.02999

Heritability
40 - 50%

% NZO predicts
Body Weight



Phenotyping the DO

Karen Svenson

Lisa Somes

Dan Gatti

Center for Genome Dynamics

Resources from Svenson 850 DO Project

650 Diversity Outbred animals have been phenotyped for clinically relevant traits. QTL analysis is underway.

Animal Information							Diet
Genotype	Sex	Generation (4-9)	Litter No.	Born Date	Coat Color	Other Coat Color Feature	Chow or HF

Urinalysis				
Urinary Glu 1	ACR1	Urinary Glu2	ACR2	Change in ACR

Electrocardiogram												
HR	HRV	PQ	PR	QRS	QTc	RR	ST	QTc dispersion	mean SR amplitude	mean R amplitude	pNN50 (>6ms)	rMSSD

Plasma chemistries at two ages: (1) 8 weeks; (2) 19 weeks												
CHOL1	HDLD1	TG1	Glu1	NEFA1	Ca1	Phos1	GLDH1	BUN1	*Change in*			
CHOL2	HDLD2	TG2	Glu2	NEFA2	Ca2	Phos2	GLDH2	BUN2	*Chol*	*TG*	*NEFA*	*Glu*

Insulin (17w)	Leptin (17w)

Hematological parameters at two ages: (1) 10 weeks; (2) 22 weeks																		
WBC1	RBC1	mHGB1	HCT1	MCV1	MCH1	MCHC 1	CHCM1	RDW1	HDW1	PLT1	MPV1	NEUT1	LYM1	MONO1	EOS1	LUC1	BASO1	Retic1	cHGB1
WBC2	RBC2	mHGB2	HCT2	MCV2	MCH2	MCHC 2	CHCM2	RDW2	HDW2	PLT2	MPV2	NEUT2	LYM2	MONO2	EOS2	LUC2	BASO2	Retic2	cHGB2

Body Composition at two ages: (1) 12 weeks; (2) 21 weeks																					
Length 1	Weight 1	% Fat 1	LTM1	BMD1	BMC1	B Area1	T Area1	RST1	TTM1	Length 2	Weight 2	% Fat 2	LTM2	BMD2	BMC2	B Area2	T Area2	RST2	TTM2	Change in Weight	Change in % Fat

Weekly Body Weights; Growth Curve																					
BW 4	BW 5	BW 6	BW 7	BW 8	BW 9	BW 10	BW 11	BW 12	BW 13	BW 14	BW 15	BW 16	BW 17	BW 18	BW 19	BW 20	BW 21	BW 22	BW 23	BW 24	BW 25	BW 26

Organ Weights			
Heart	Spleen	Kidney L	Kidney R

Liver Harvest		
RNAseq	Metabol-omics	Protein processing

Other Tissue Harvest	
Kidney/R RNAseq	Muscle (gastrocnemius) RNAseq

Pellets: Microbiome	
Gen 8 Ltr 2 (*n*=100) 18w	Gen 9 Ltr 1 (*n*=100) 18w, 26w

Karen Svenson and Lisa Somes
15 August 2012

The Jackson Laboratory

***N* = 146* traits**

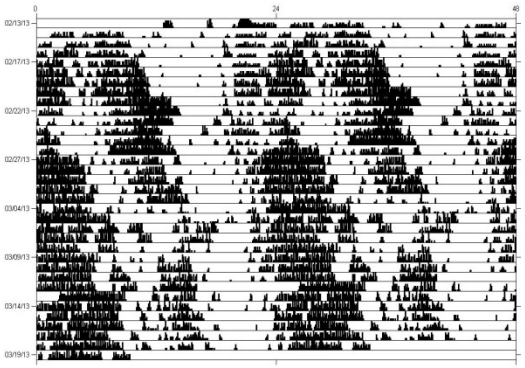
**Other calculated traits can be derived*

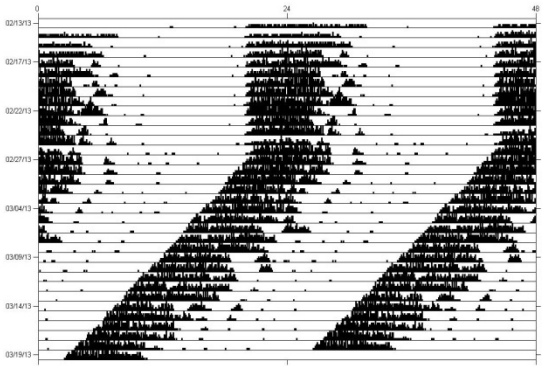
Circadian and Sleep Phenotyping

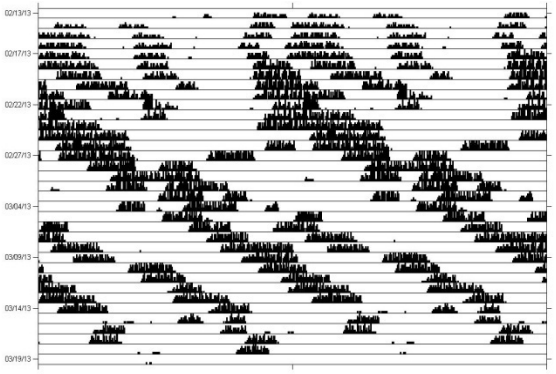
Alan Pack
Karen Svenson

Variations in Circadian Rhythm in Diversity Outbred Mice

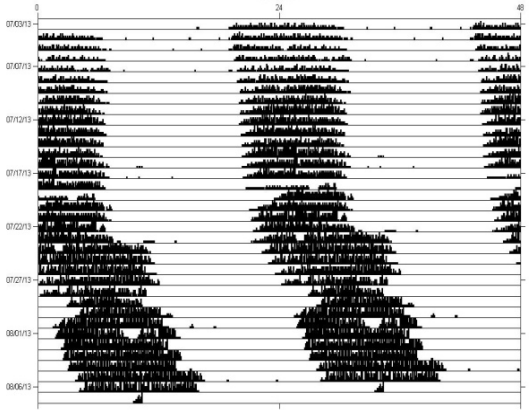
JAXDO800#22C5BlackLeft LD 24.01 DD 23.07 0.94

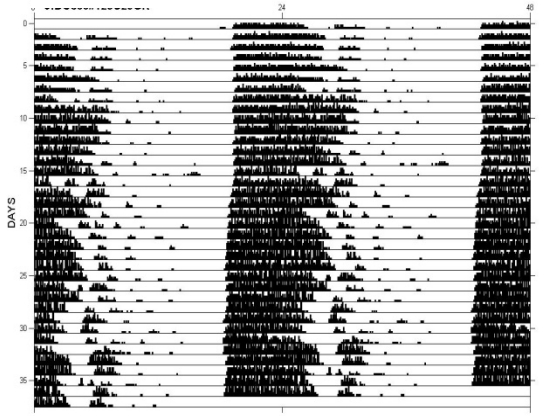




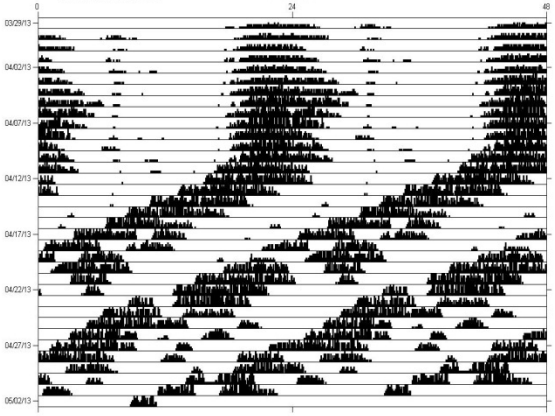


JAXDO800#70C14GreyLeft LD 24.02 DD 24.44 -0.42

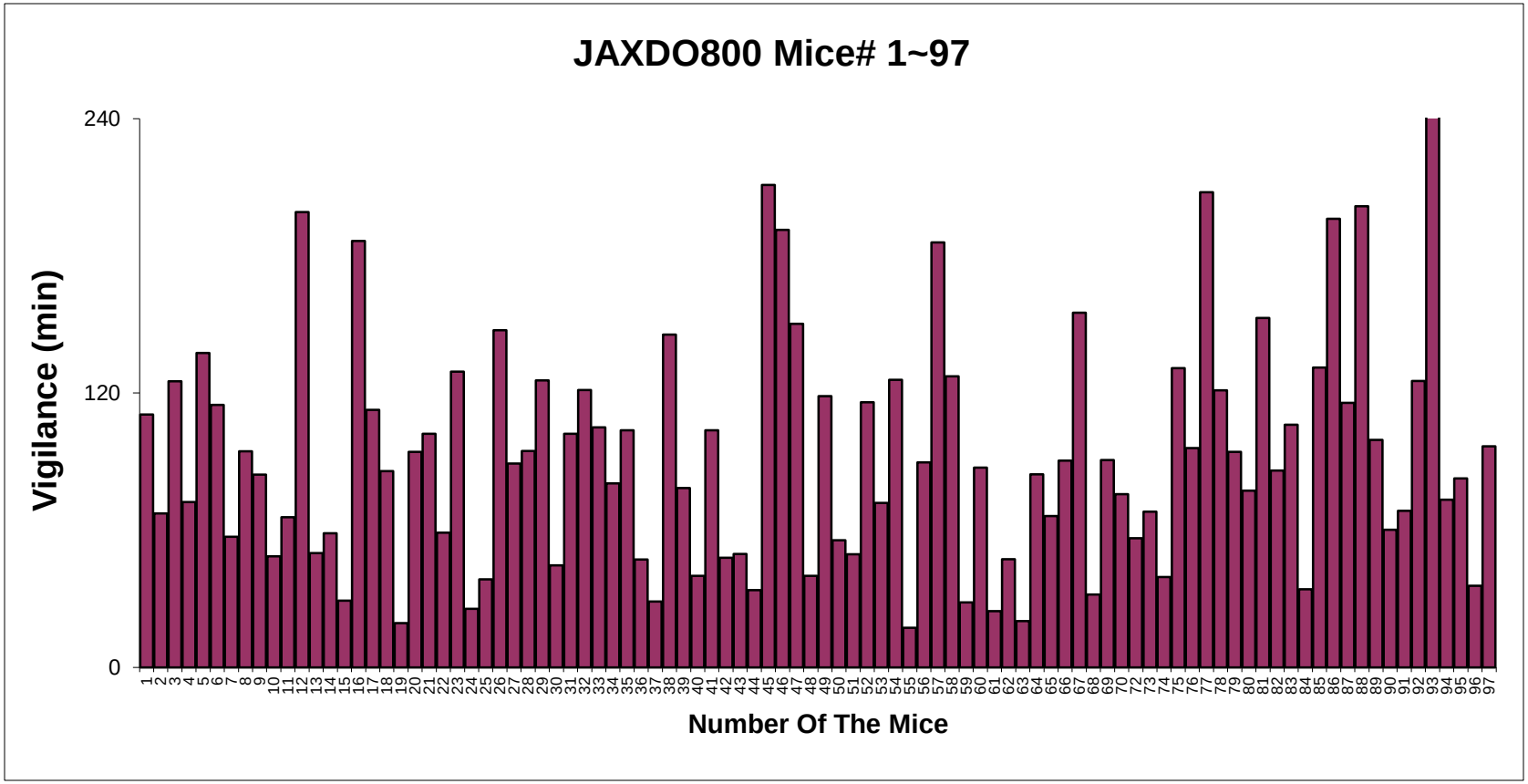




JAXDO800#31C7White LD 24.00 DD 21.71 2.29



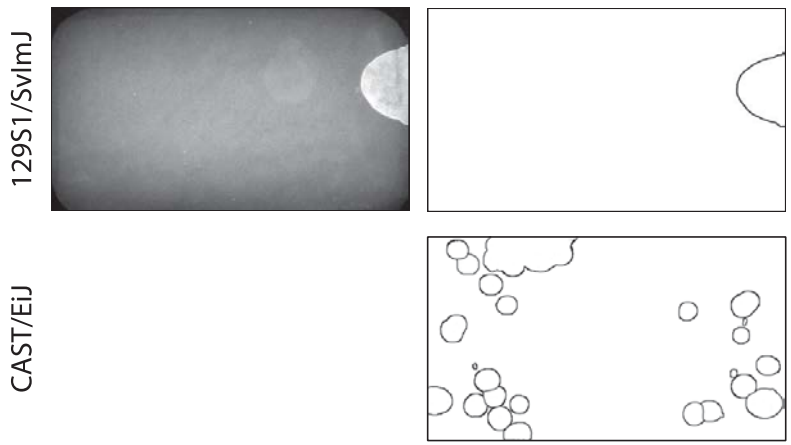
Variation in Vigilance in DO Mice Time-to-Fall-Asleep in New Cage

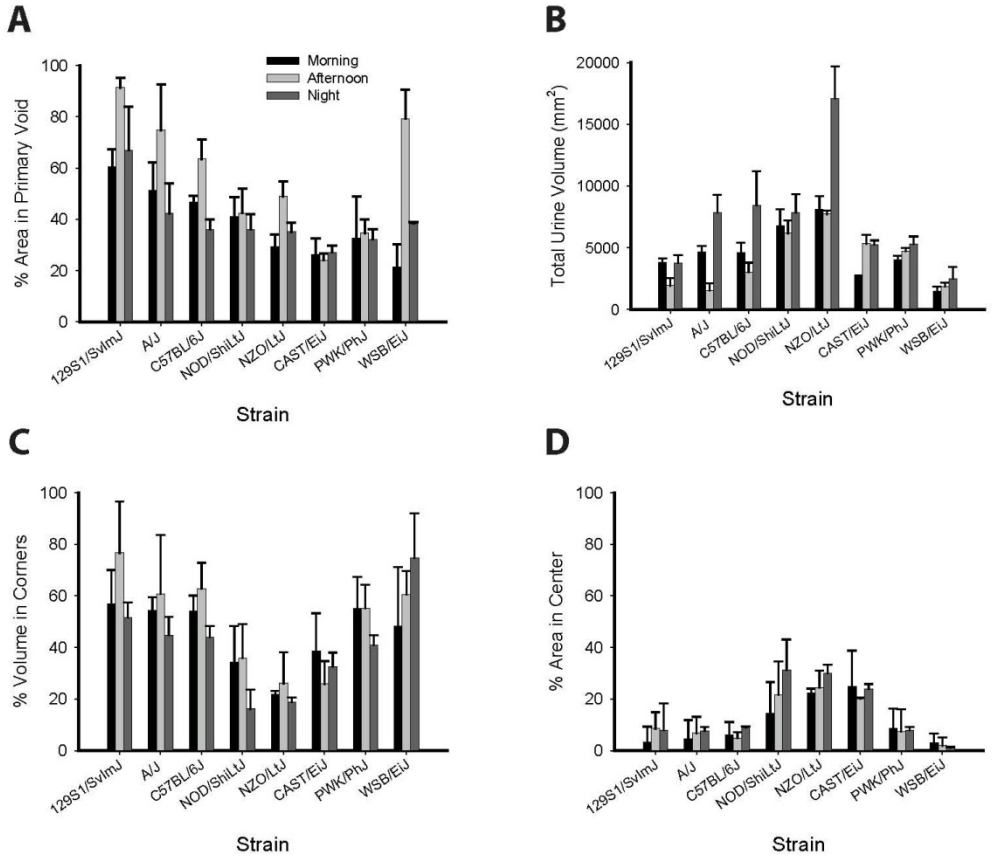


Urinary Spotting and Bladder Function

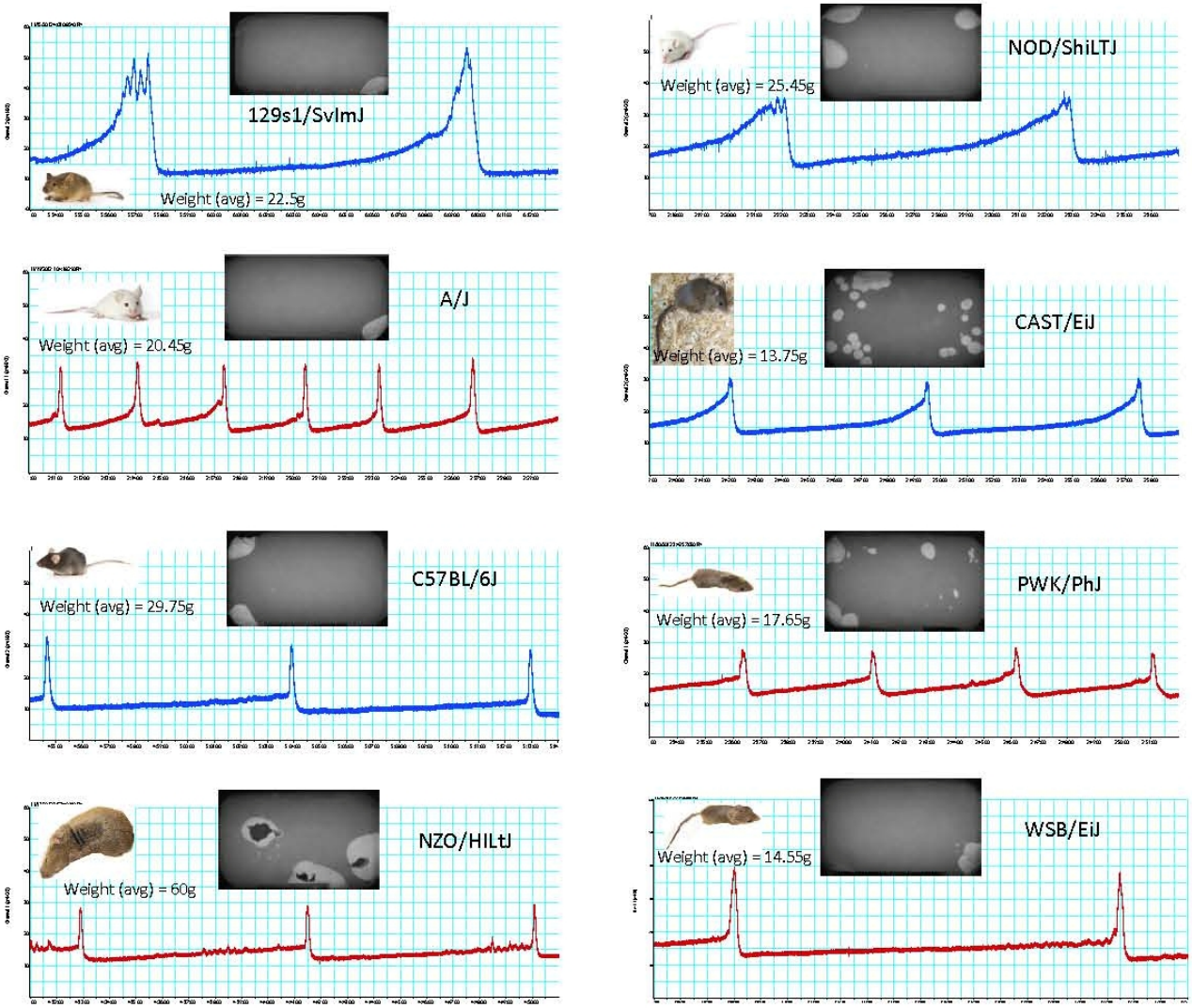
Mark Zeidel, Warren Hill
Cheryl Ackert-Bicknell

Quantitative Assay of Urinary Spotting





Bladder Function in DO Founders

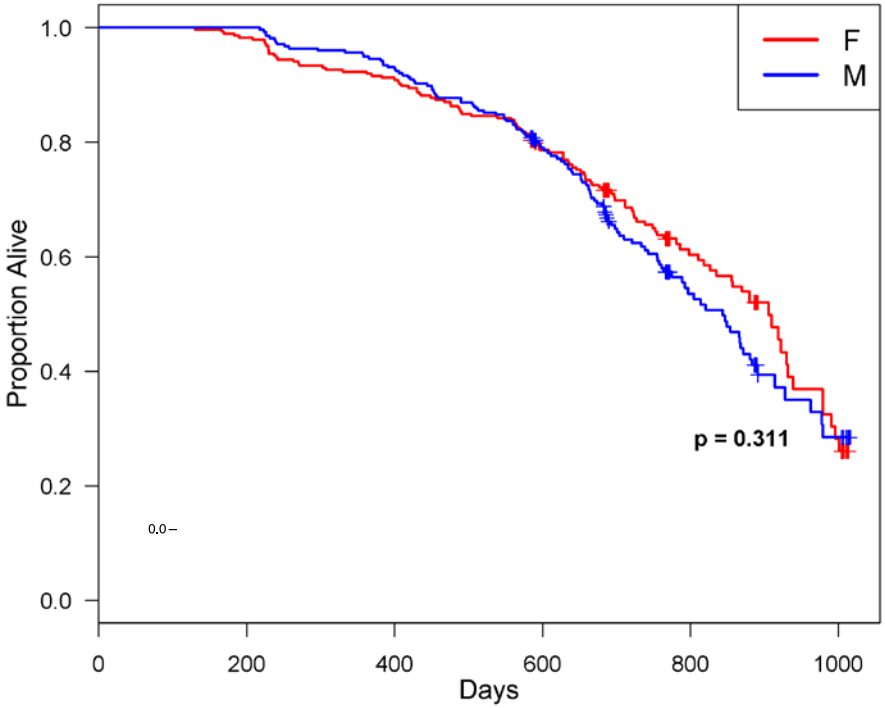


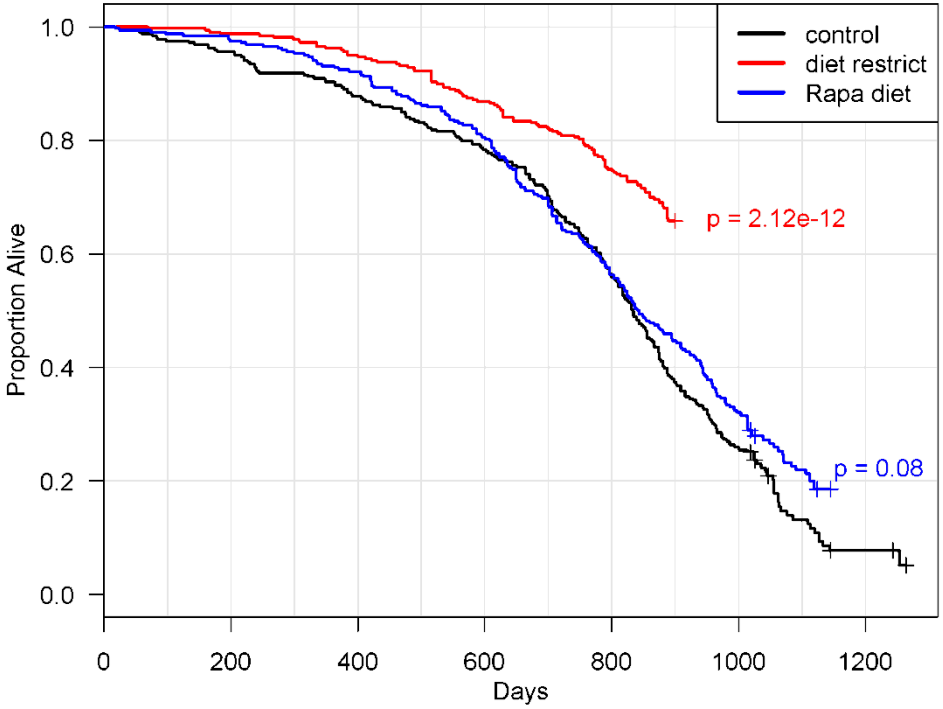
Lifespan Interventions

Karen Svenson

Lisa Somes

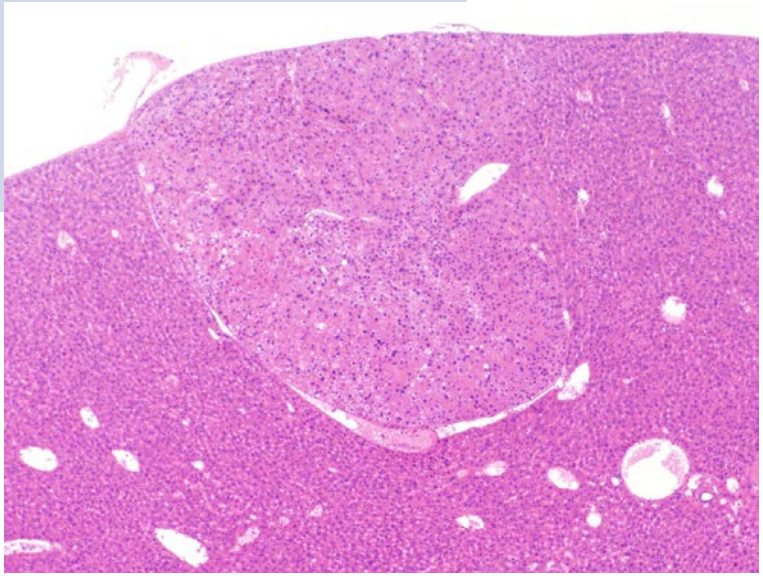
Lifespan of DO mice is genetically regulated and responsive to interventions



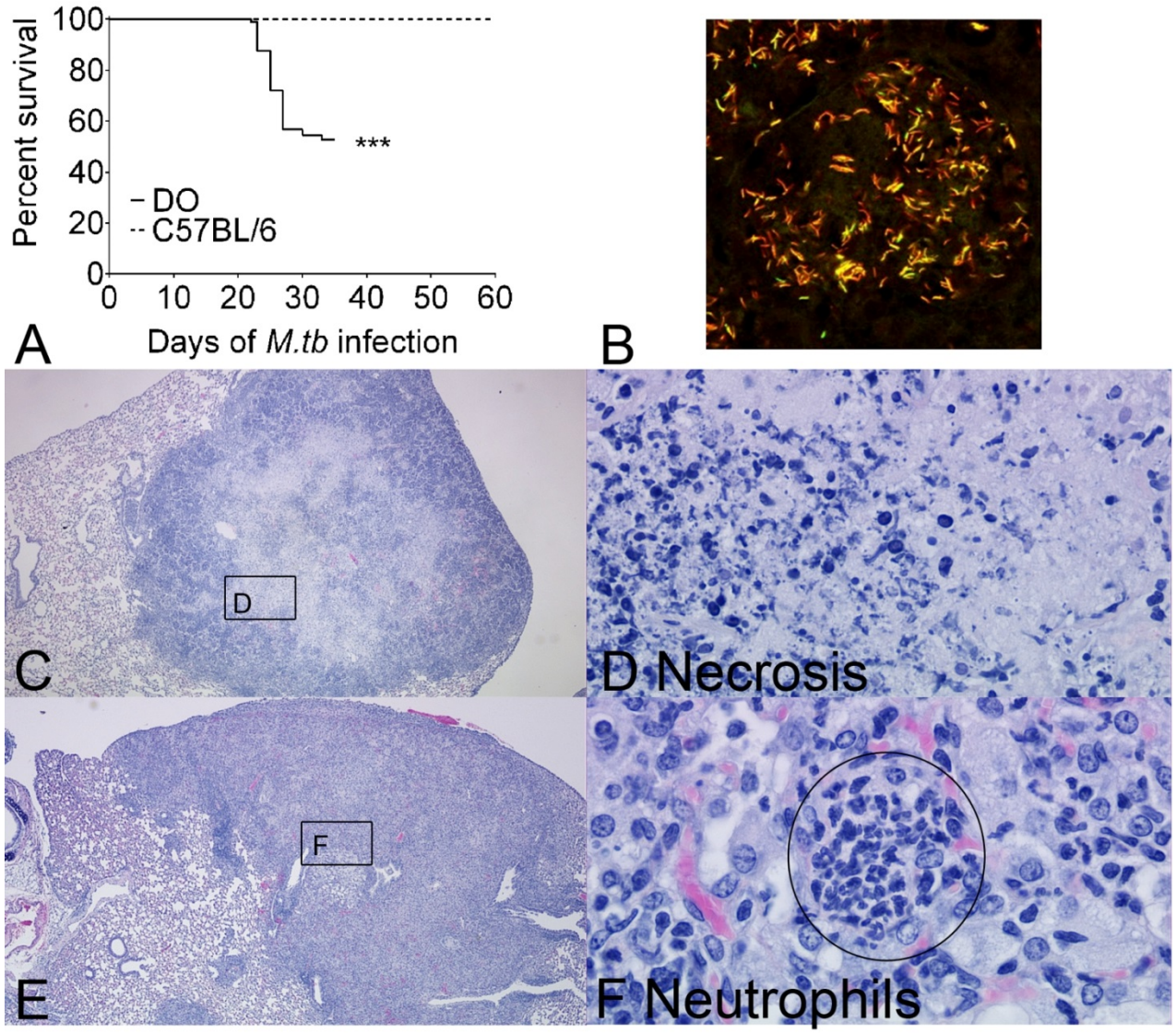


Cancer

Type of tumor	Incidence	Notes
Hepatocellular Adenoma	3% of all mice examined (5.15% of all males and 1% of females)	Incidence increases with age. Do not metastasize. Can grow in excess of 10% of mouse's body weight in size.
Hepatocellular Carcinoma	2% of all males examined. Not seen in females.	Likely incidence increases with age. Can metastasize to the lungs.
Fatty Metamorphosis	0.5% of female mice (not seen in males).	



Infectious Disease

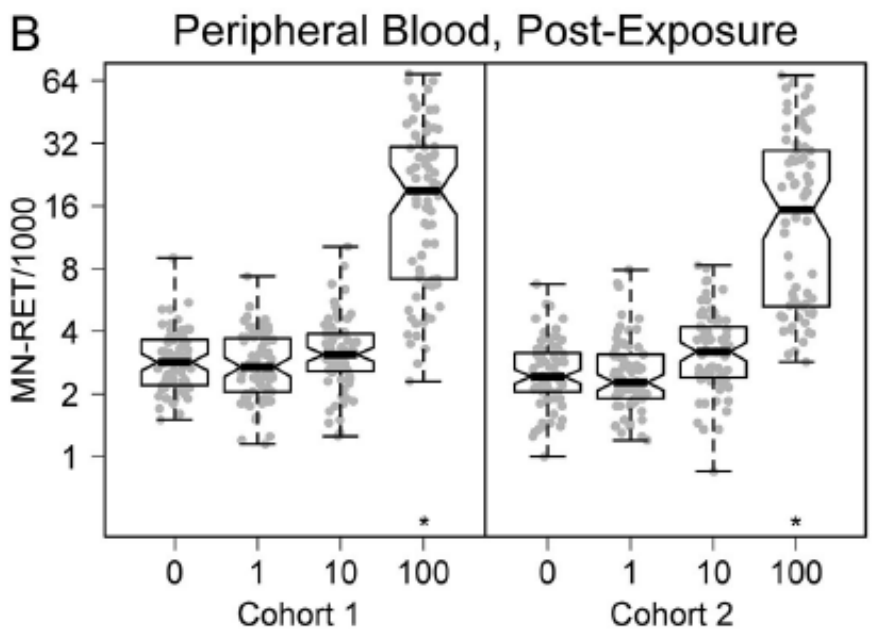


Toxicology

Diversity Outbred Mice Identify Population-Based Exposure Thresholds and Genetic Factors that Influence Benzene-Induced Genotoxicity

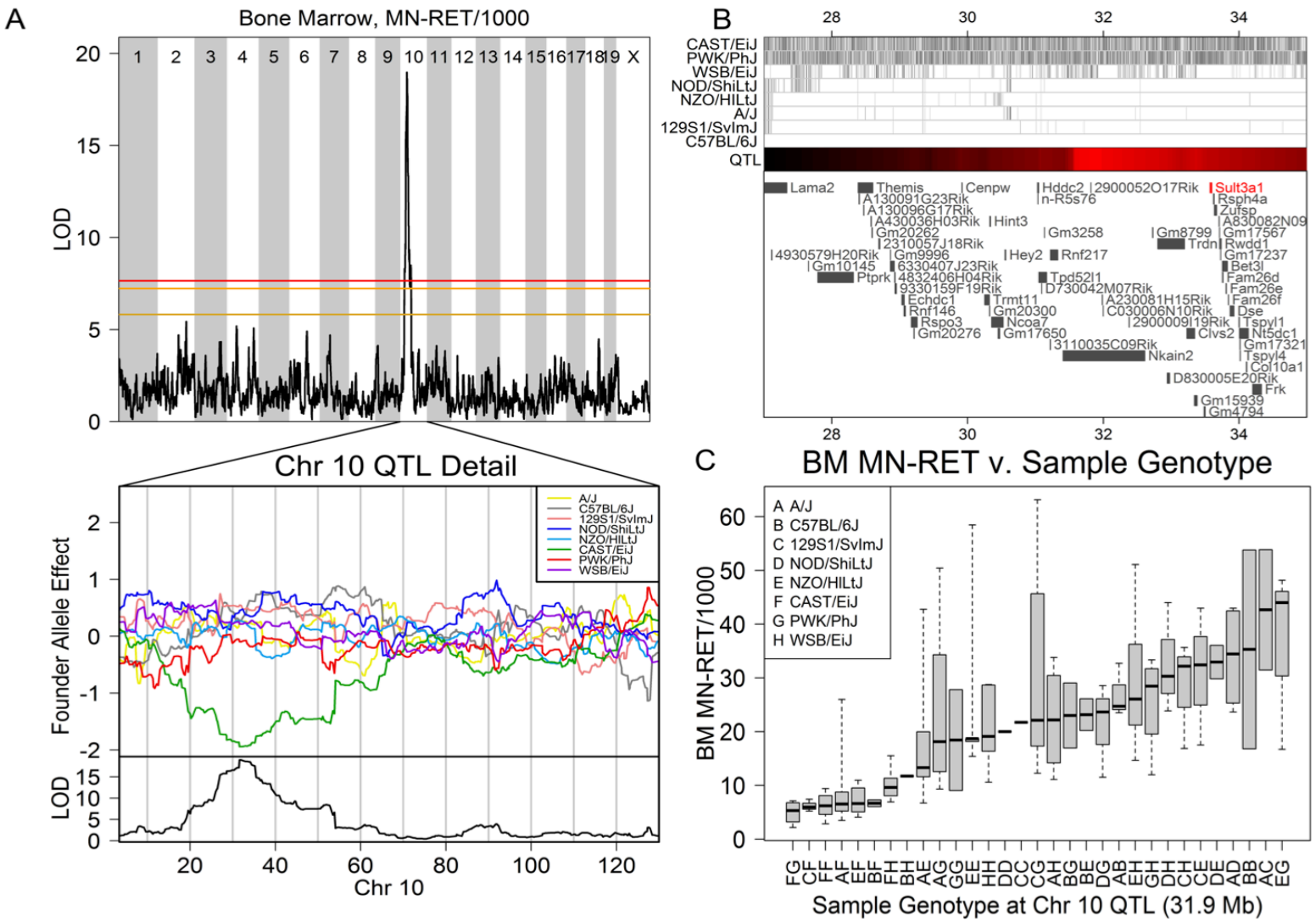
John E. French,[1] Daniel M. Gatti,[2] Daniel L. Morgan,[1] Grace E. Kissling,[3] Keith R. Shockley,[3] Gabriel A. Knudsen,[4] Kim Shepard,[5] Herman C. Price,[6] Deborah King,[1] Kristine L. Witt,[1] Lars C. Pedersen,[3] Steven C. Munger,[2] Karen L. Svenson,[2] and Gary A. Churchill[2]

Environmental Health Perspectives 2014

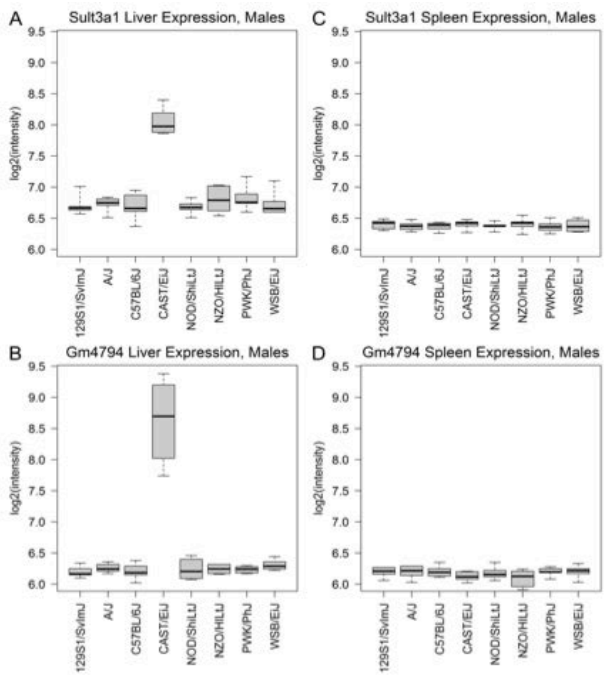


Genetically diverse DO mice provide a reproducible response to benzene exposure variation that reflects the range of responses that can be expected in human populations.

QTL mapping in DO mice defines candidate gene region for susceptibility to benzene exposure.



Expression of Sult3a1 and Gm4794 in liver is specific to CAST mice.



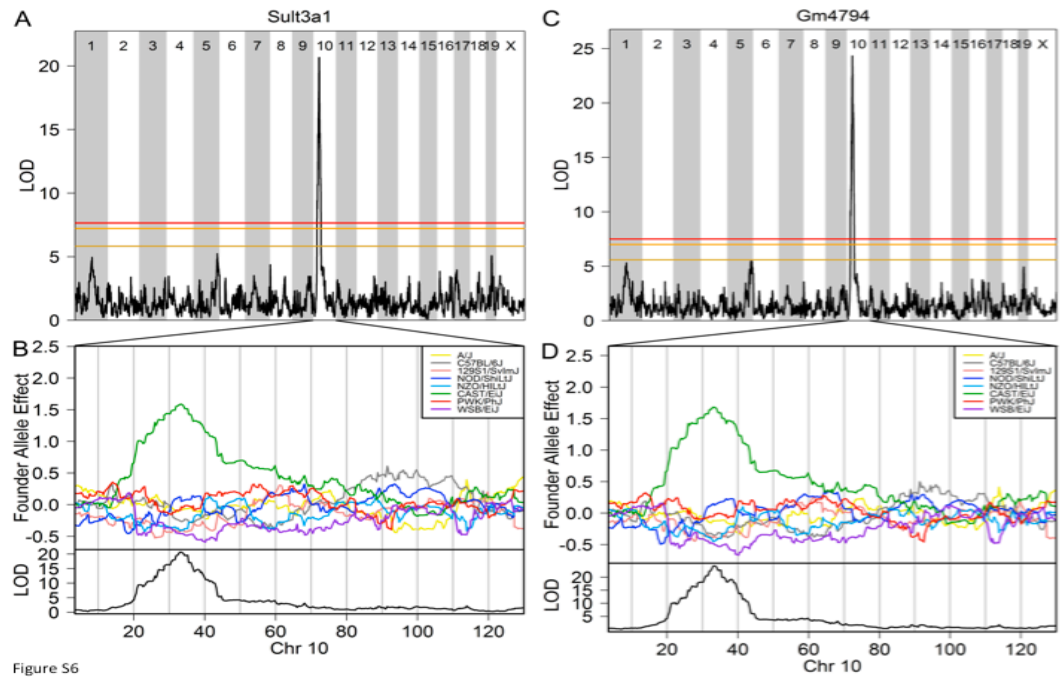
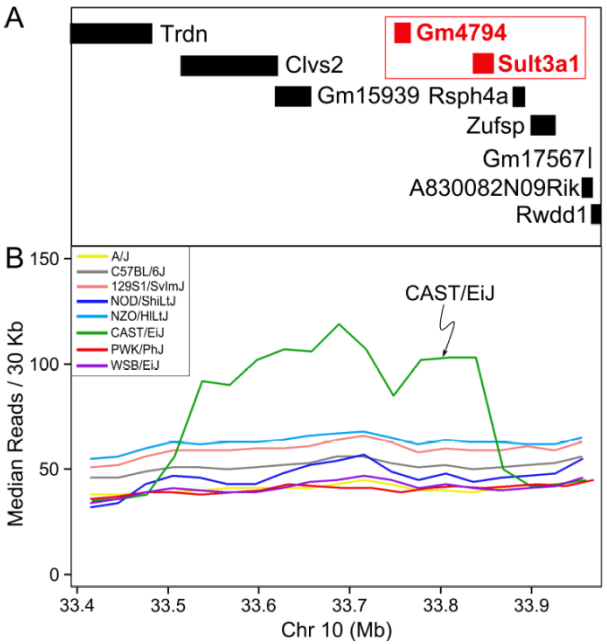


Figure S6

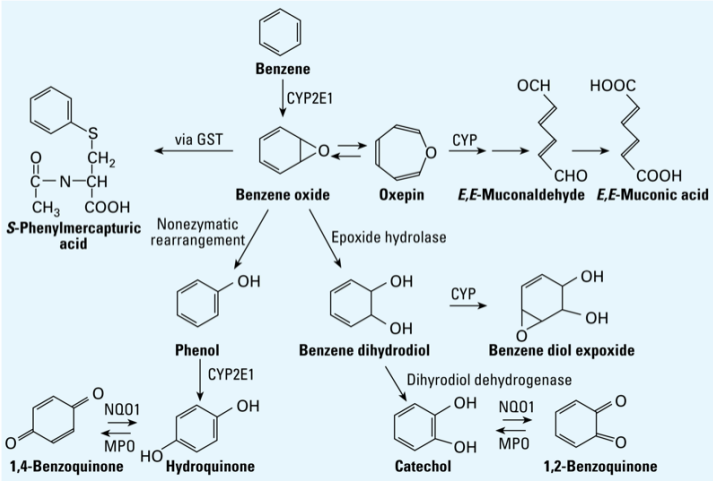
Sult3a1 and Gm4794 are duplicated in the CAST genome.



Genetic Polymorphisms in *CYP1A1*, *CYP2D6*, *UGT1A6*, *UGT1A7*, and *SULT1A1* Genes and Correlation with Benzene Exposure in a Chinese Occupational Population

Copy number variation in sulfotransferases has been associated with benzene toxicity in human studies

Sulfotransferase activity represents a novel pathway for benzene detoxification.



DO Resources

http://do.jax.org

The Jackson Laboratory
Leading the search for tomorrow's cures

The Churchill Group

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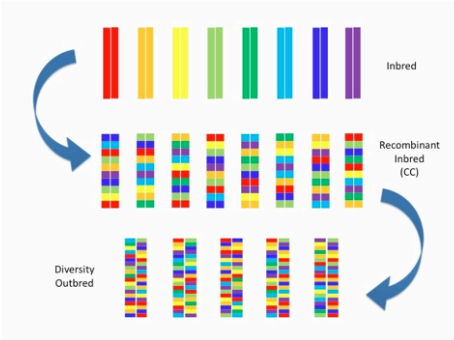
- Graphical Models
- Software Development
- QTL
- Microarray
- Center for Genome Dynamics
- Collaborative Cross
- Diversity Outbred

The Diversity Outbred (DO)

The Diversity Outbred (DO) is a heterogeneous stock derived from the same eight founder strains as the **Collaborative Cross (CC)** inbred strains: A/J, C57BL/6J, 129S1/SvImJ, NOD/ShiLtJ, NZO/HiLtJ, CAST/EiJ, PWK/PhJ, and WSB/EiJ.



In 2009, animals representing 144 independent lineages from the CC breeding colony at The Oak Ridge National Laboratory (Chesler et al. 2008) were used to seed the DO population, which was then maintained as a randomized breeding colony with a population size of 175 pairs.

One female and one male from each first litter are randomly assigned to a new breeding pair to make the next generation. This mating scheme doubles the effective population size and minimizes the effects of drift and selection on allele frequencies in the DO (Rockman and Kruglyak 2008; unpublished simulations). Each DO mouse is a unique individual with a high level of allelic heterozygosity, and the DO population provides an effectively unlimited source of novel allelic combinations.

The CC strains are being inbred to produce stable clones. The DO mice, on the other hand, are being maintained as an outbred stock. Advantages of outbreeding include, normal levels of heterozygosity, similar to the human genetic condition, and substantially increased mapping resolution. A drawback of the DO is that each animal is genetically unique and thus not reproducible. Combinations of genetic loci that are discovered in the DO mice can be replicated in CC strains or in their reproducible F1 progeny. In this regard, the CC and DO populations are complementary. Together these new resources will revolutionize our understanding, treatment and ultimately, prevention of pervasive human diseases.

Papers and Datasets

Churchill GA, Gatti DM, Munger SC, Svenson KL
The Diversity Outbred Mouse Population
Mammalian Genome 2012 Oct;23(9-10):713-8. **[datasets]**

Svenson KL, Gatti DM, Valdar W, Welsh CE, Cheng R, Chesler EJ, Palmer AA, McMillan L, Churchill GA.
High-Resolution Genetic Mapping Using the Mouse Diversity Outbred Population
Genetics. 2012 Feb;190(2):437-47. PMCID: PMC3276626 **[Full Text]** **[datasets]**

Presentations

Genetic analysis of RNA editing in the Diversity Outbred mice (PDF 0.3 MB)
Tongjun Gu, Robert E. Braun, Gary A. Churchill
Gordon Research Conference on RNA Editing, Galveston, TX, Jan 6-11, 2013. Poster

RNA-seq Alignment to Individualized Genomics (PDF 6.5 MB)
Steve Munger
40th Maine Biological and Medical Sciences Symposium, Bar Harbor, ME, April 12-13, 2013. Talk

Mapping Toxicity Traits Using the Diversity Outbred Mice (PDF 5.1 MB)
Daniel Gatti
Genetically Diverse Mouse Models Workshop, ILSI Health and Environmental Sciences Institute, Washington, DC, November 28, 2012. Talk

Developing the Laboratory Mouse as a Tool for Systems Genetics (Video on meeting website)
Gary Churchill
Animal Models and Their Value in Predicting Drug Efficacy and Toxicity
New York Academy of Sciences, New York, NY, September 15-16, 2011. Talk

News Articles

Diversity Outbred and Collaborative Cross Mice to Offer Maximum Allelic Variation
Summer 2009. In its quest to provide even more powerful genetically distinct mouse resources, JAX is developing a population of Diversity Outbred (DO) mice, designed to maximize allelic variation throughout the genome. Each DO mouse will be genetically unique, and groups of them will approximate the genetic diversity found in human populations.

Diversity Outbed Mice

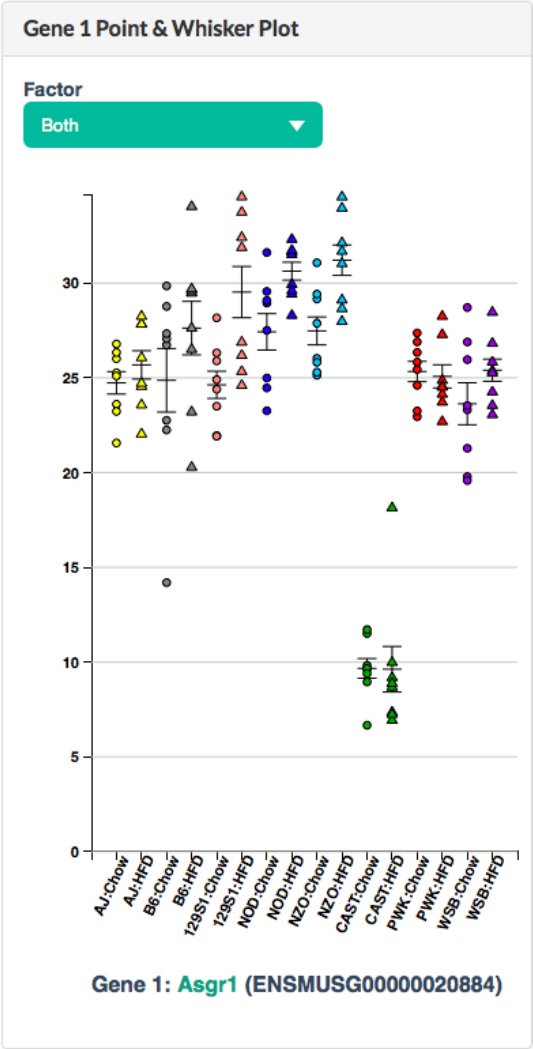
Click here to order mice from JAX® Mice & Services

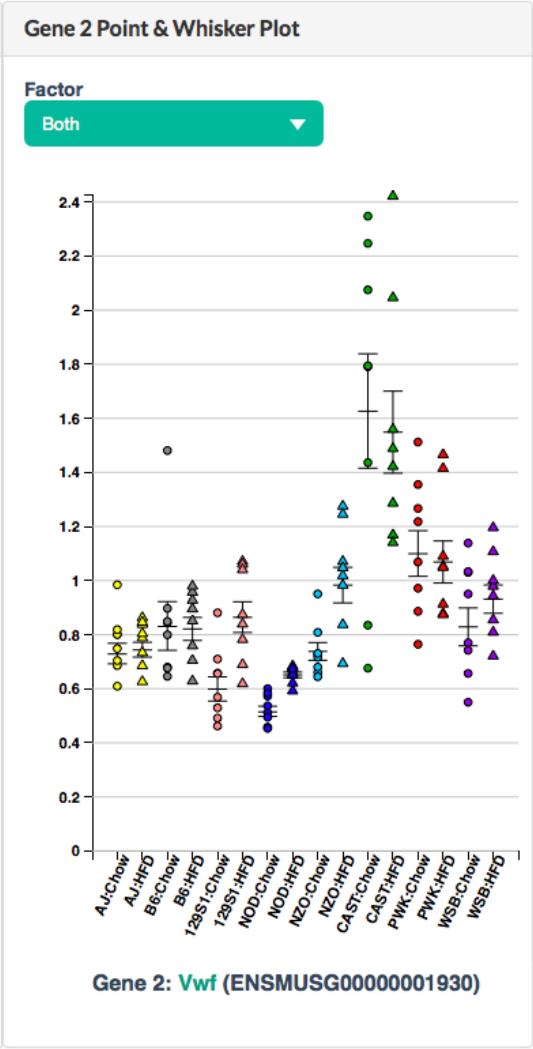
The Diversity Outbred (J:DO) stock is maintained and supplied by The Jackson Laboratory.

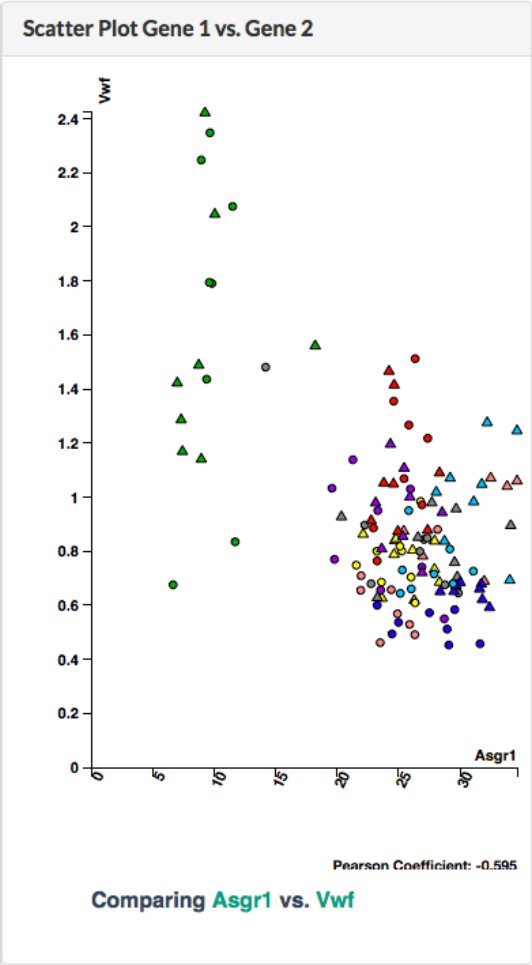
JAX® Mice & Services
Strain and Ordering Information

Home | Site Map | Disclaimer | Churchill wiki

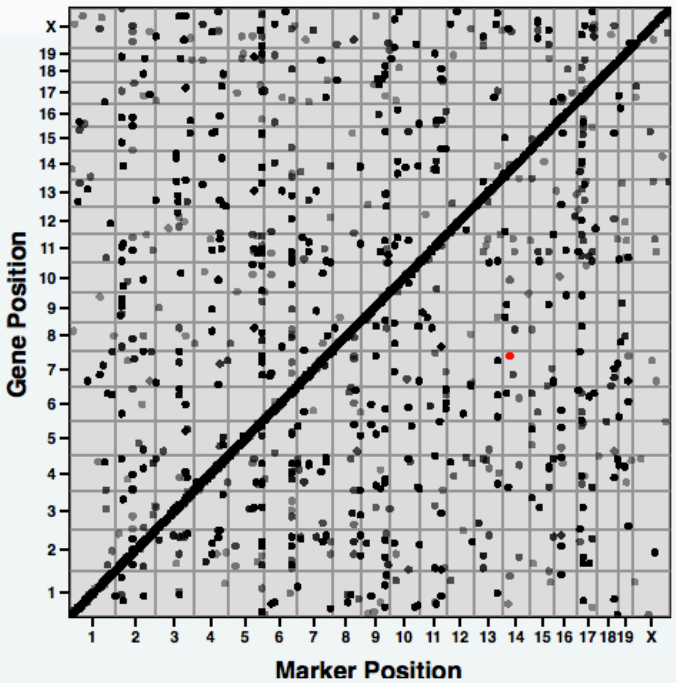
Aim 3A. Interactive data visualization







eQTL Matrix Plot



Gene: Bcl7c (ENSMUSG00000030814)

Gene Search

Type in a Gene Symbol or Ensembl ID to locate the corresponding data in the grid.

Gene Bcl Find

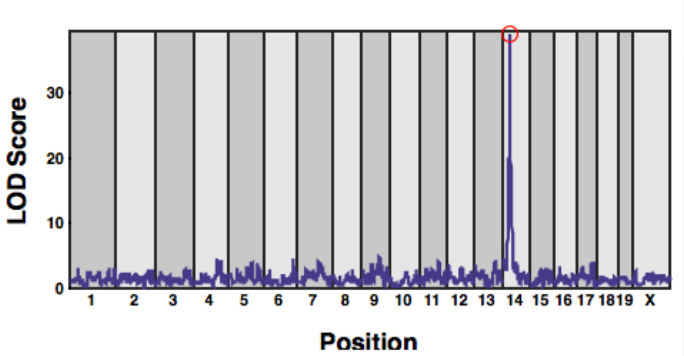
More than 10 matches were found. Only showing the first 10.

Ensembl ID	Symbol	LOD Score
ENSMUSG00000007659	Bcl2l1	19.07
ENSMUSG00000038256	Bcl9	8.65
ENSMUSG00000044165	Bcl2l15	17.12
ENSMUSG00000028191	Bcl10	25.76
ENSMUSG00000029681	Bcl7b	19.93
ENSMUSG00000009112	Bcl2l13	20.01
ENSMUSG00000003190	Bcl2l12	9.48
ENSMUSG00000030814	Bcl7c	39.26
ENSMUSG00000074148	Bcl2a1d	13.56
ENSMUSG00000074147	Bcl2a1a	10.84

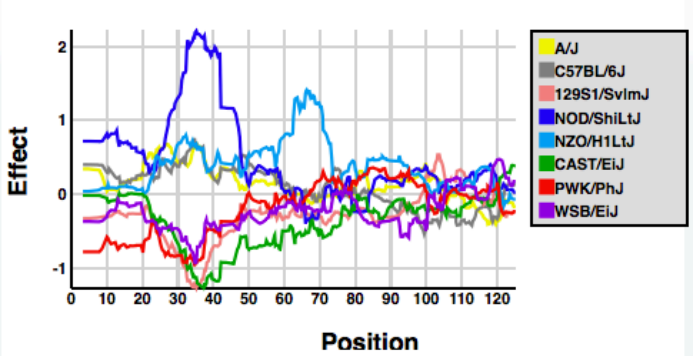
☐ Show ONLY the results

☑ Highlight the results

LOD Score Plot



Effect Plot



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