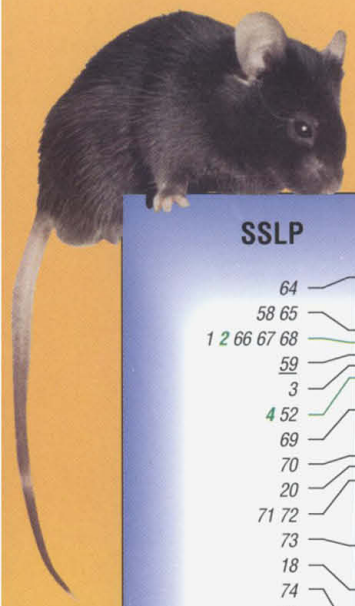

Gus^{mps}*/Gus*^{mps}

GE

The laboratory mouse
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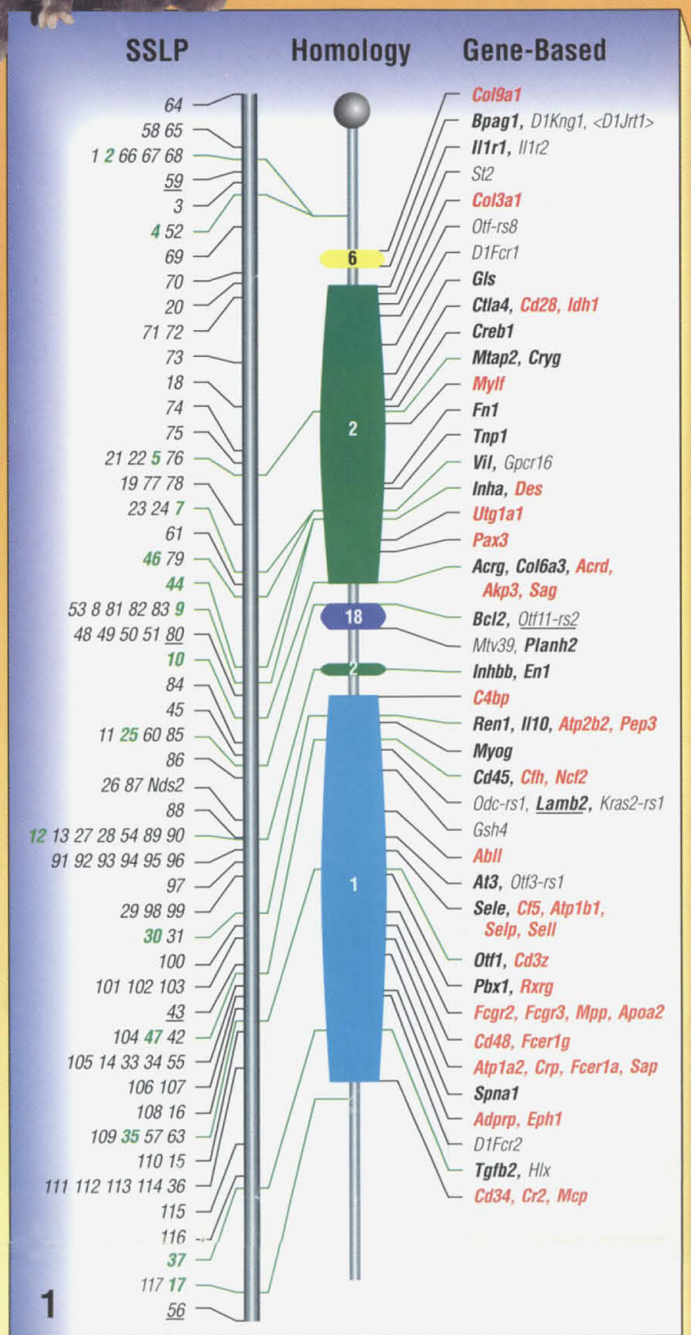
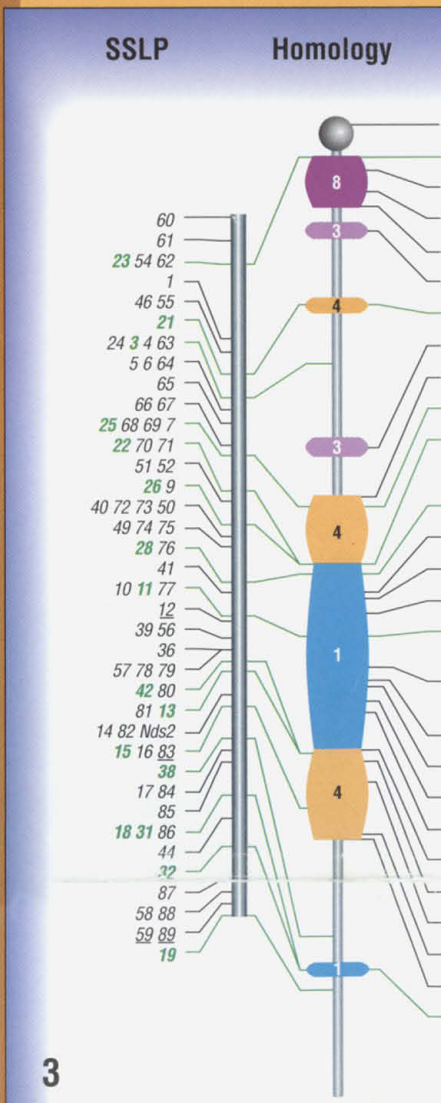
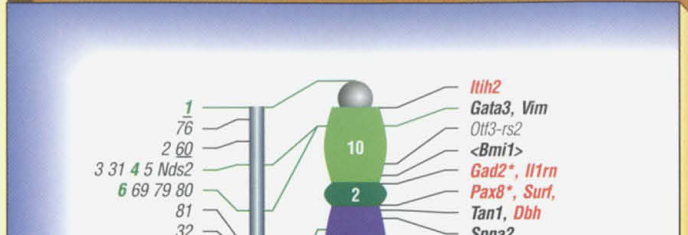
show pathologies cl
mouse is also an exc





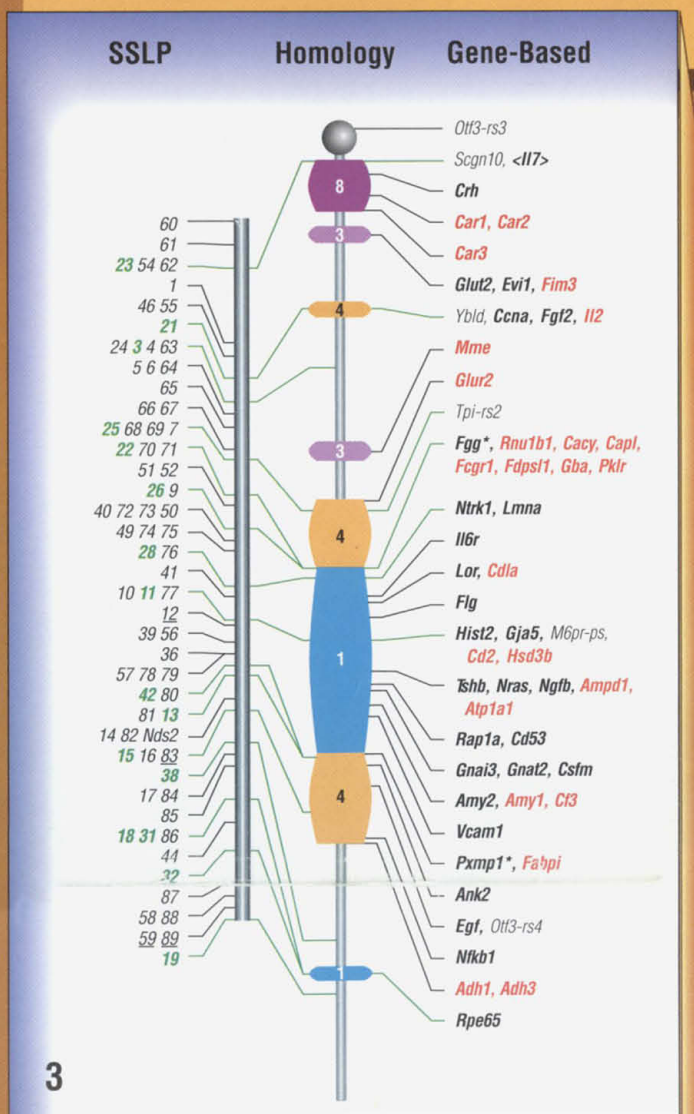


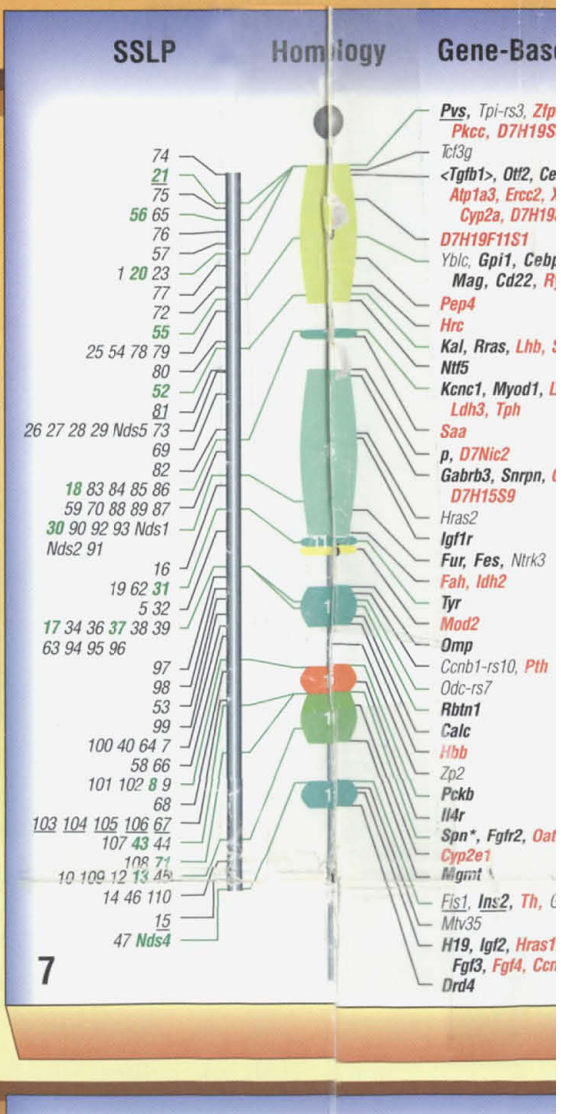
SCIENCE

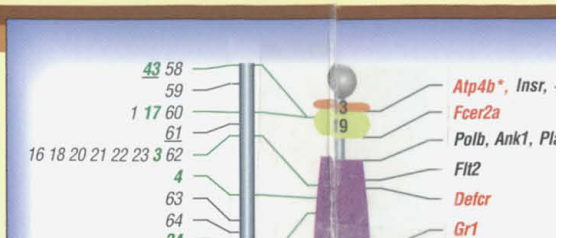
GENOME M

THE MOUSE

The laboratory mouse is the best experimental model for studying many aspects of mammalian physiology and development. Many mouse mutants and strains show pathologies closely resembling those of human genetic diseases. The mouse is also an excellent model at the genomic level: the mouse and human genomes are a genes, and ofte of mouse mod genes in the tw







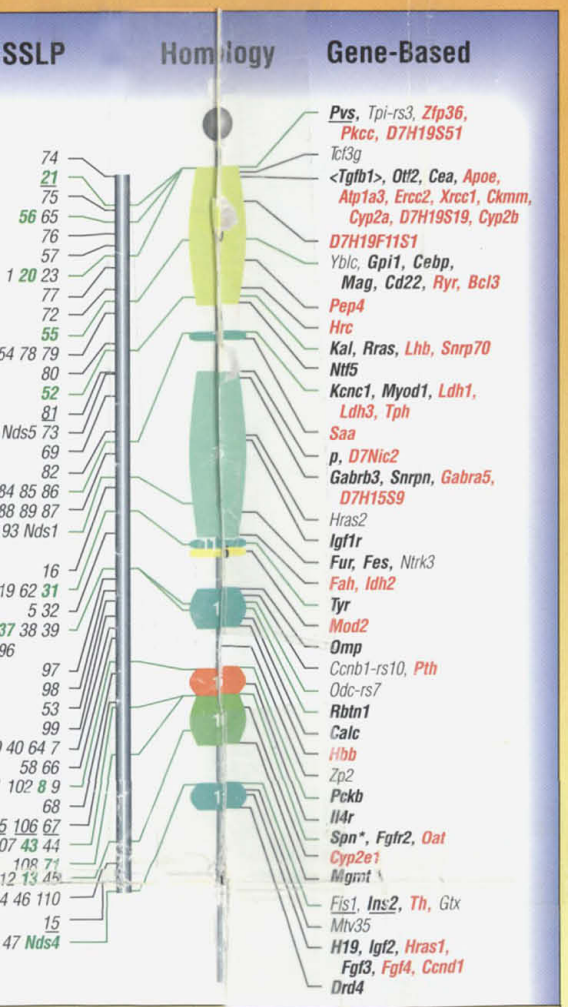
SCIENCE

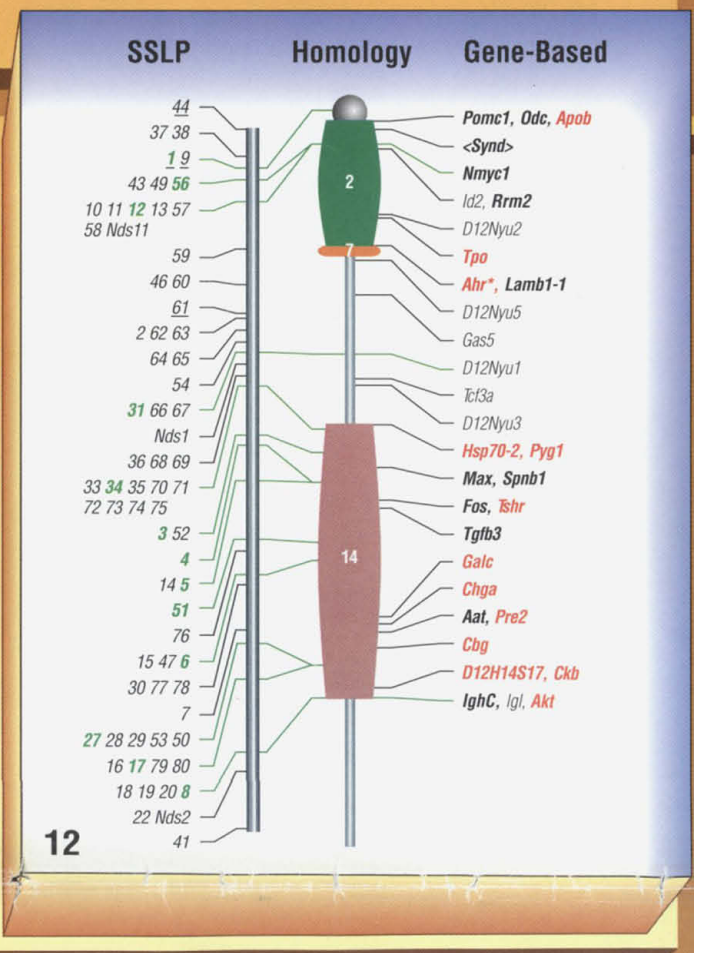
ME MAPS IV

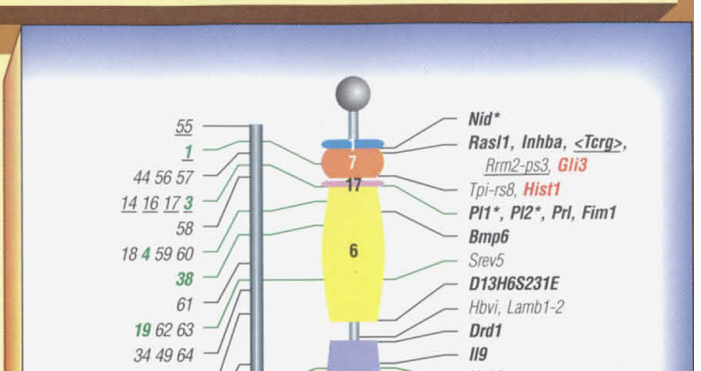
THE MOUSE

g many aspects
ants and strains
iseases. The
se and human

genomes are approximately the same size, contain essentially the same basic collection of genes, and often show evolutionary conservation in gene order. Moreover, in a growing list of mouse models of human disease, the responsible mutations are found in homologous genes in the two species.

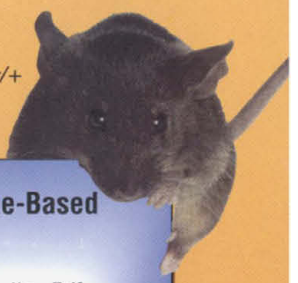


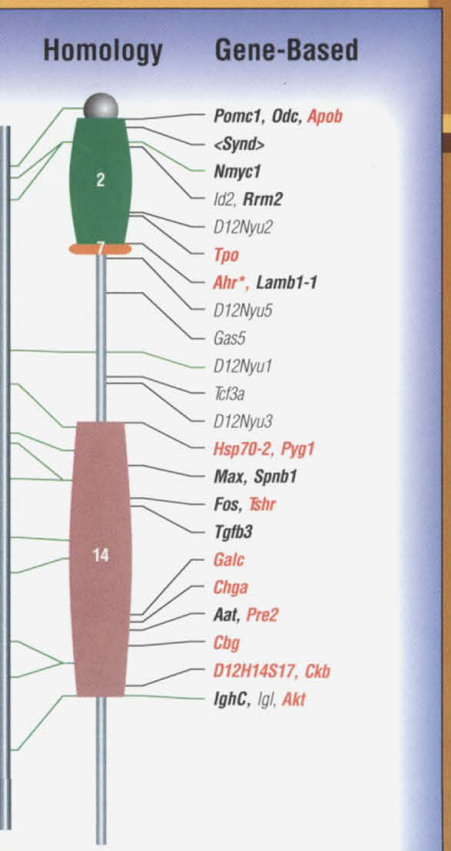


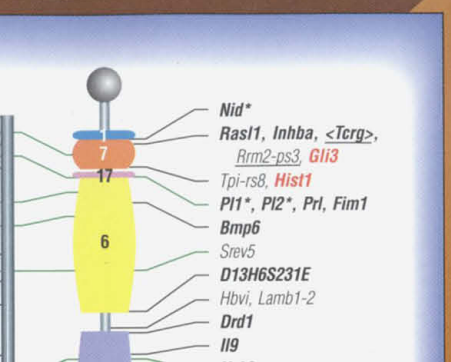


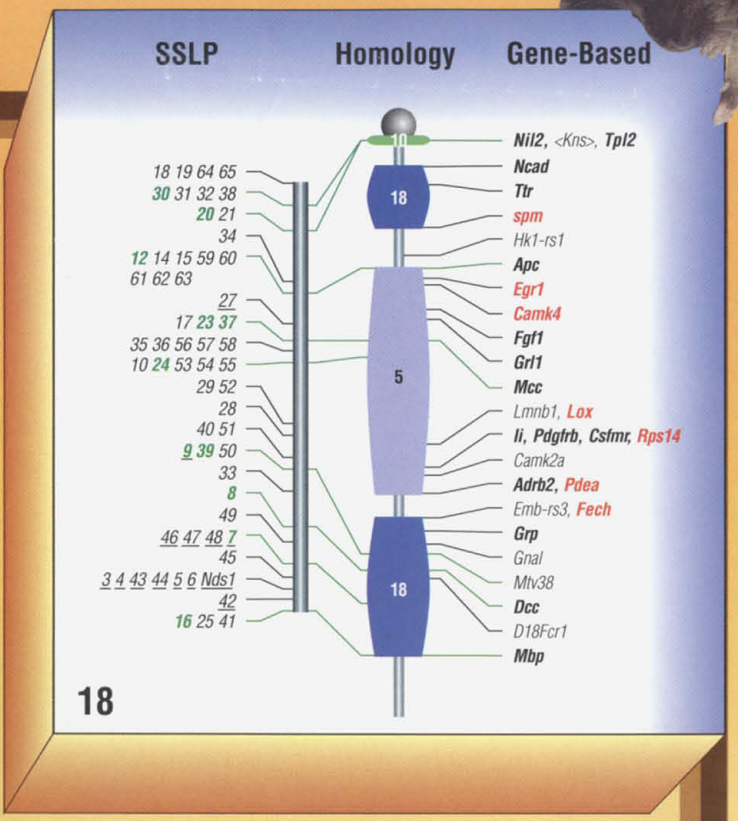
SIV

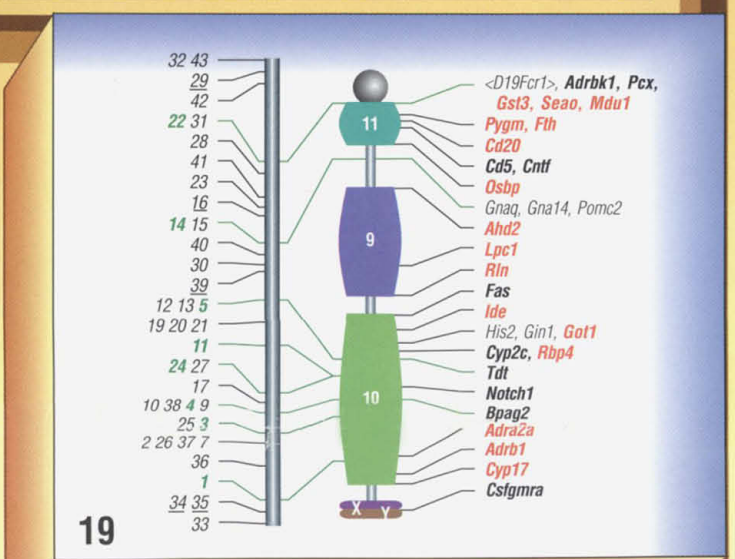
ially the same basic collection of
order. Moreover, in a growing list
tions are found in homologous

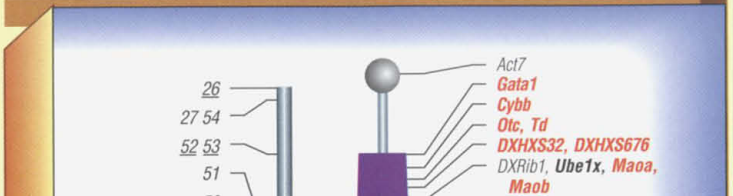


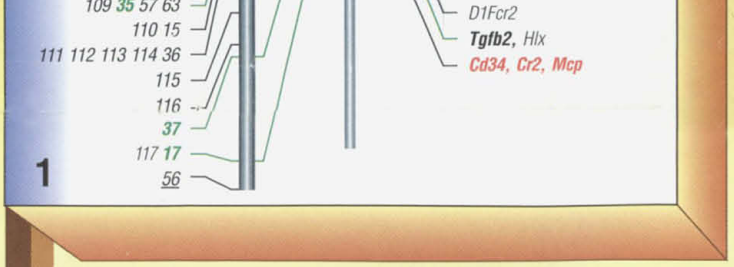


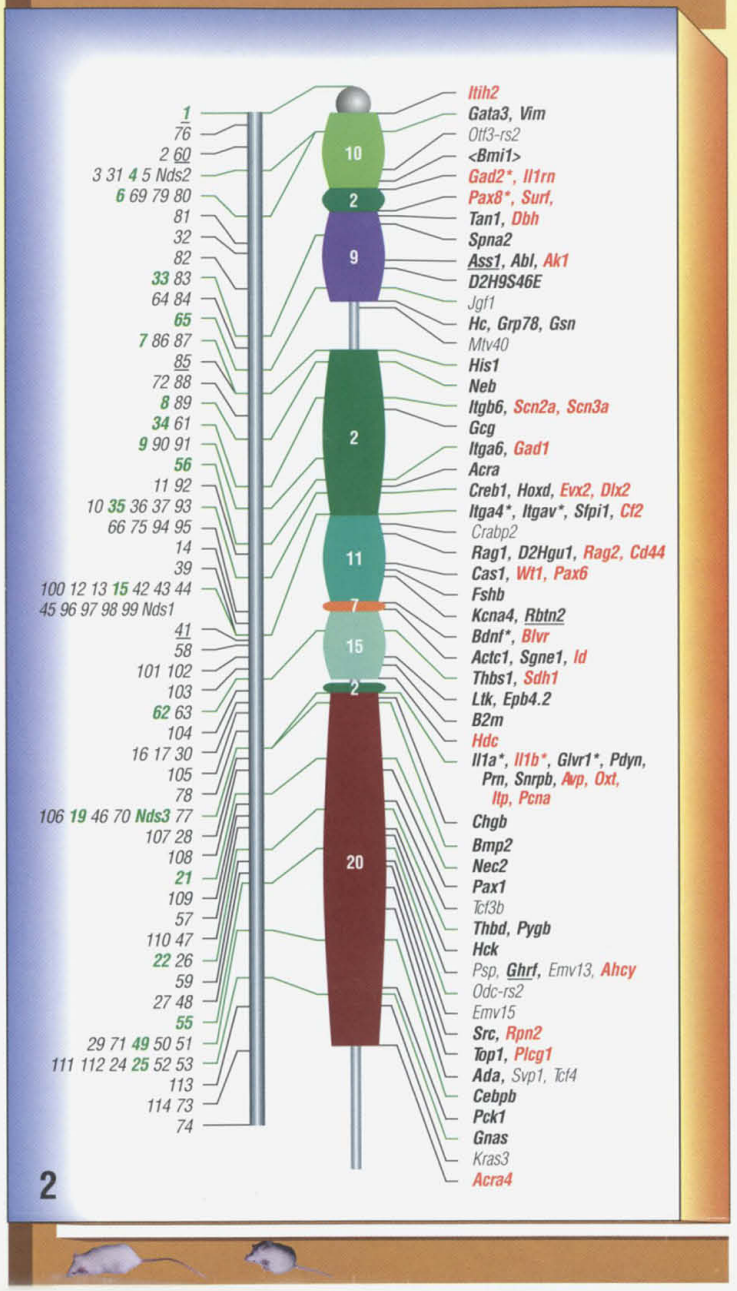


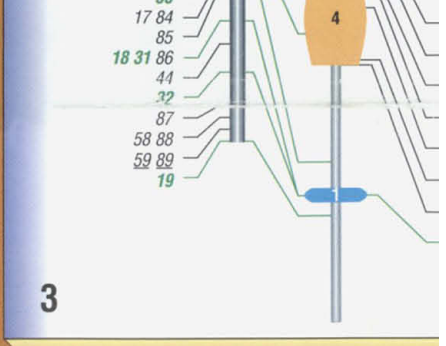


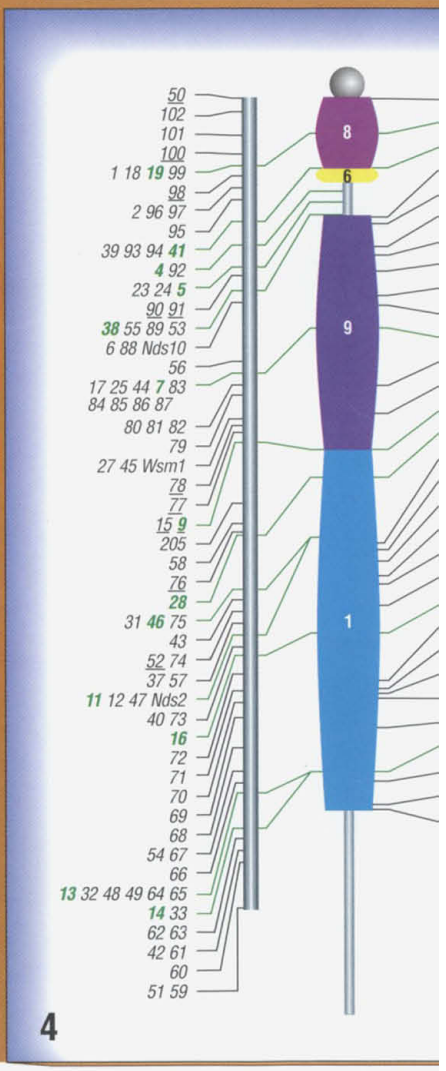


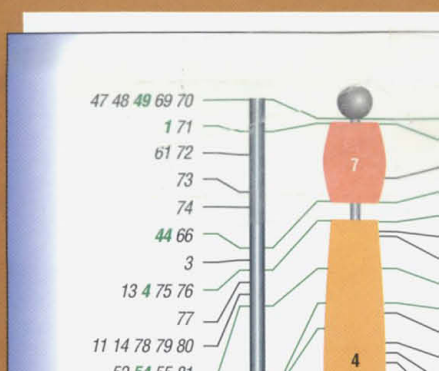






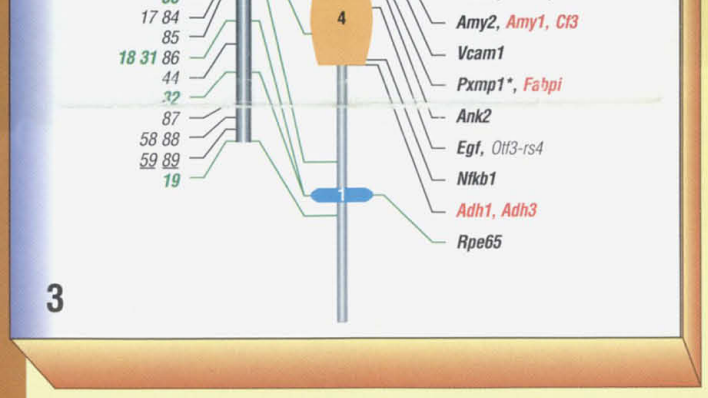






REPRESENTATIVE SINGLE-GENE MOUSE MODELS FOR HUMAN GENETIC DISORDERS

Mouse gene*	Mouse chr	Human condition	Human chr
Apc^{Min}	18	Adenomatous polyposis coli	5q21-q22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32
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58 88
59 89
19
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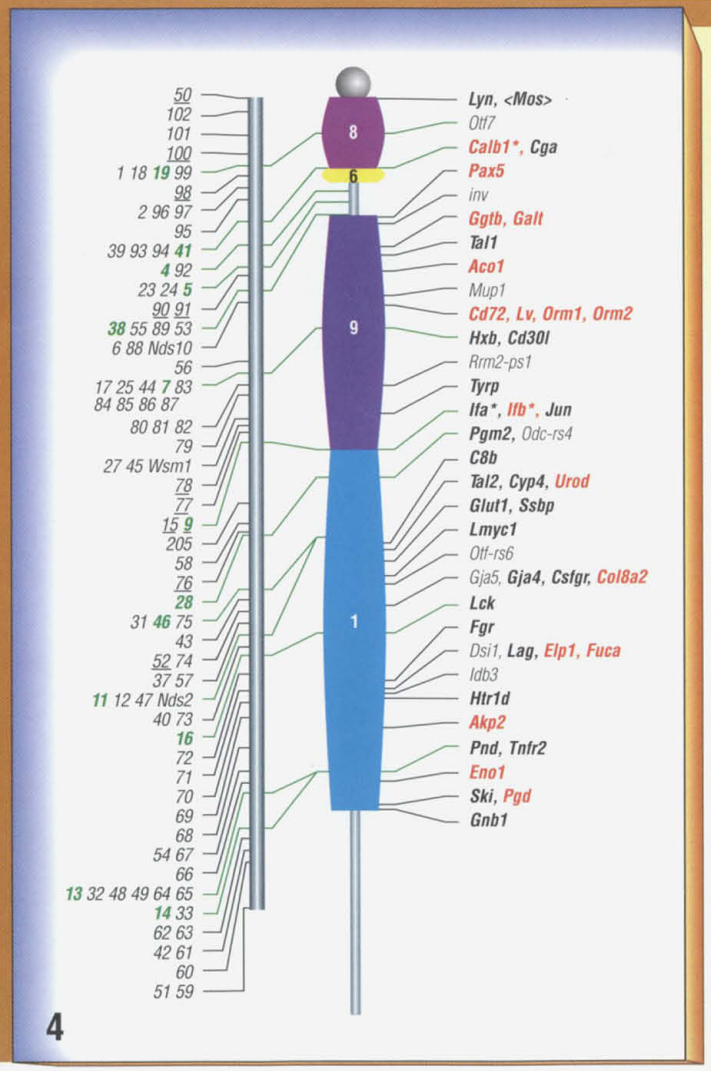
Amy2, Amy1, Cf3
Vcam1
Pxmp1*, Fabpi
Ank2
Egf, Otf3-rs4
Nfkb1
1
Adh1, Adh3
Rpe65

3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101
100
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38 55 89 53
6 88 Nds10
56
17 25 44 7 83
84 85 86 87
80 81 82
79
27 45 Wsm1
78
77
15 9
205
58
76
28
31 46 75
43
52 74
37 57
11 12 47 Nds2
40 73
16
72
71
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54 67
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14 33
62 63
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51 59
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6
9
1
Lyn, <Mos>
Otf7
Calb1*, Cga
Pax5
inv
Ggtb, Galt
Tal1
Aco1
Mup1
Cd72, Lv, Orm1, Orm2
Hxb, Cd30l
Rrm2-ps1
Tyrp
Ifa*, Ifb*, Jun
Pgm2, Odc-rs4
C8b
Tal2, Cyp4, Urod
Glut1, Ssbp
Lmyc1
Otf-rs6
Gja5, Gja4, Csfgr, Col8a2
Lck
Fgr
Dsi1, Lag, Elp1, Fuca
Idb3
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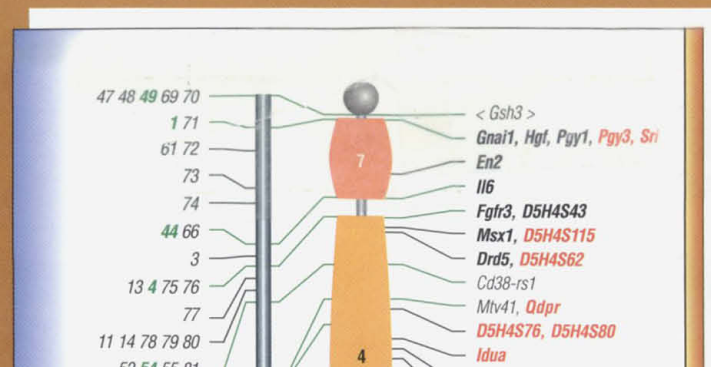
Pnd, Tnfr2
Eno1
Ski, Pgd
Gnb1

4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11 14 78 79 80
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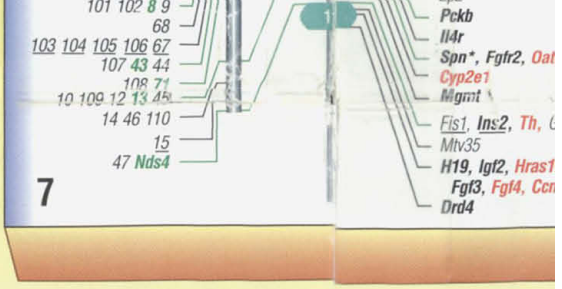
4
< Gsh3 >
Gnai1, Hgf, Pgy1, Pgy3, Sri
En2
Il6
Fgfr3, D5H4S43
Msx1, D5H4S115
Drd5, D5H4S62
Cd38-rs1
Mtv41, Qdpr
D5H4S76, D5H4S80
Idua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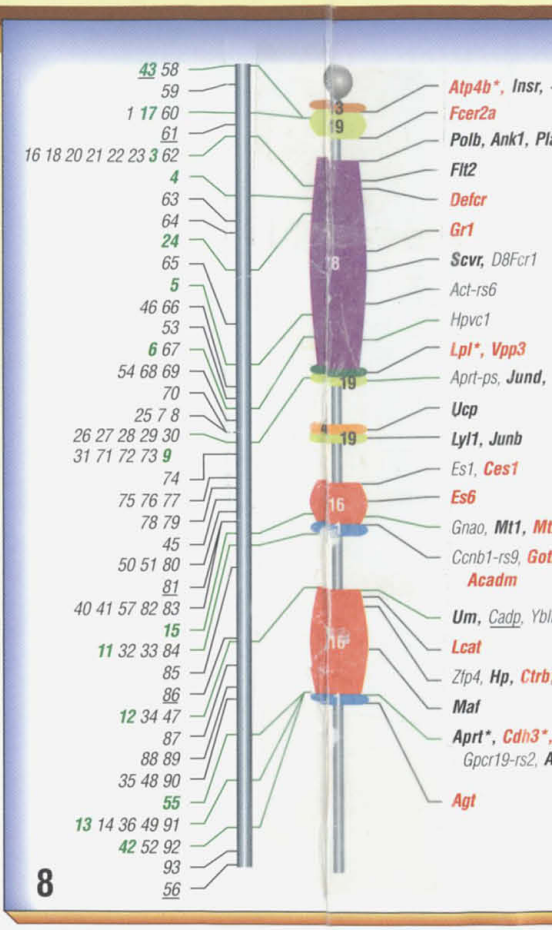
103 104 105 106 67
107 43 44
108 71
10 109 12 13 45
14 46 110
15
47 Nds4
Pckb
Il4r
Spn*, Fgfr2, Oat
Cyp2e1
Mgmt
Fis1, Ins2, Th,
Mtv35
H19, Igf2, Hras1
Fgf3, Fgl4, Ccn
Drd4

7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6 67
54 68 69
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25 7 8
26 27 28 29 30
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74
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78 79
45
50 51 80
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11 32 33 84
85
86
12 34 47
87
88 89
35 48 90
55
13 14 36 49 91
42 52 92
93
56
Atp4b*, Insr,
Fcer2a
Polb, Ank1, Pl
Flt2
Defcr
Gr1
Scvr, D8Fcr1
Act-rs6
Hpvc1
Lpl*, Vpp3
Aprt-ps, Jund,
Ucp
Lyl1, Junb
Es1, Ces1
Es6
Gnao, Mt1, Mt
Ccnb1-rs9, Got
Acadm
Um, Cadp, Ybl
Lcat
Ztp4, Hp, Ctrb
Maf
Aprt*, Cdh3*,
Gpcr19-rs2,
Agt

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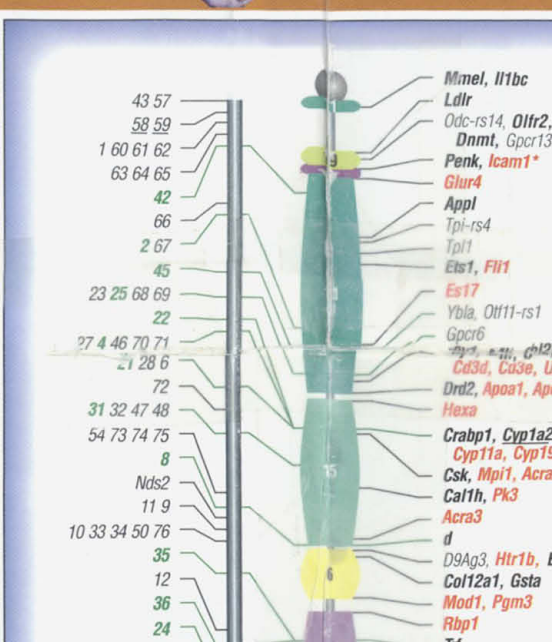
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2 67
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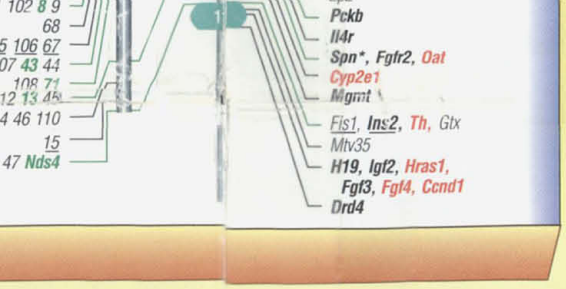
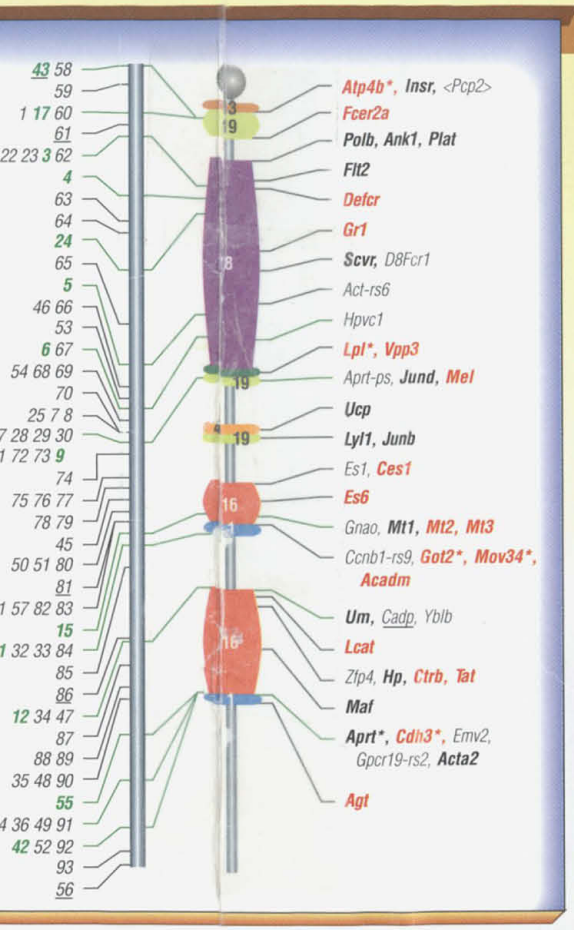
54 73 74 75
8
Nds2
11 9
10 33 34 50 76
35
12
36
24
Mmel, Il1bc
Ldlr
Odc-rs14, Olfr2,
Dnmt, Gpcr13
Penk, Icam1*
Glur4
Appl
Tpi-rs4
Tpi1
Ets1, Fli1
Es17
Ybla, Otf11-rs1
Gpcr6
Cd3d, Cd3e,
Drd2, Apoa1, Ap
Hexa
Crabp1, Cyp1a2
Cyp11a, Cyp1
Csk, Mpi1, Acra
Cal1h, Pk3
Acra3
D9Ag3, Htr1b,
Col12a1, Gsta
Mod1, Pgm3
Rbp1


Pckb
Il4r
Spn*, Fgfr2, Oat
Cyp2e1
Mgmt
Fis1, Ins2, Th, Gtx
Mtv35
H19, Igf2, Hras1,
Fgf3, Fgf4, Ccnd1
Drd4
15
47 Nds4

18 19 20 8
22 Nds2
41

12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Flt2
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Gr1
Scvr, D8Fcr1
Act-rs6
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Lpl*, Vpp3
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Ucp
Lyl1, Junb
Es1, Ces1
Es6
Gnao, Mt1, Mt2, Mt3
Ccnb1-rs9, Got2*, Mov34*,
Acadm
Um, Cadp, Yblb
Lcat
Zfp4, Hp, Ctrb, Tat
Maf
Aprt*, Cdh3*, Emv2,
Gpcr19-rs2, Acta2
Agt
43 58
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35 78

Nid*
Rasl1, Inhba, <Tcrg>,
Rrm2-ps3, Gli3
Tpi-rs8, Hist1
Pl1*, Pl2*, Prl, Fim1
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D13H6S231E
Hbvi, Lamb1-2
Drd1
Il9
Ntrk2
Srd5a1
Nec1
Rasa
As1
Hilda
Crhbp, Dhfr, Hexb, Mtap5
Ccnb-rs13
Htr1a
Ctla3, Itga1, Itga2, Hmgcr
Tel13

ct/ct

13

1 10 11 43 46 47 48 49
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12 Nds1
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61 62
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Wnt5a
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Bmp4
Psp2, Apex, Rib1
Tcra, Tcrd, Np1
Ctla1, Ctsg, Myhca,
Myhcb, Ang
Gjb2, Gja3
Snrpn-ps1
Nfl, Clu
Bmp1, Sftp-2, hc Gnrh
Es10, Htr2, Rb1
Ccnb1-rs12
D14Hi3S26
Tyrp2
Rap2a
Pcca

14

Mmel, Il1bc
Ldlr
Odc-rs14, Olfr2, <Epor>,
Dnmt, Gpcr13
Penk, Icam1*
Glur4
Appl
Tpi-rs4
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Ets1, Fli1
Es17
Ybla, Otf11-rs1
Gpcr6
Cd3d, Cd3e, Ups
Drd2, Apoa1, Apoa4, Ncam
Hexa
Crabp1, Cyp1a2, Cyp1a1,
Cyp11a, Cyp19
Csk, Mpi1, Acra5
Cal1h, Pk3
Acra3
d
D9Ag3, Htr1b, Bmp5
Col12a1, Gsta
Mod1, Pgm3
Rbp1
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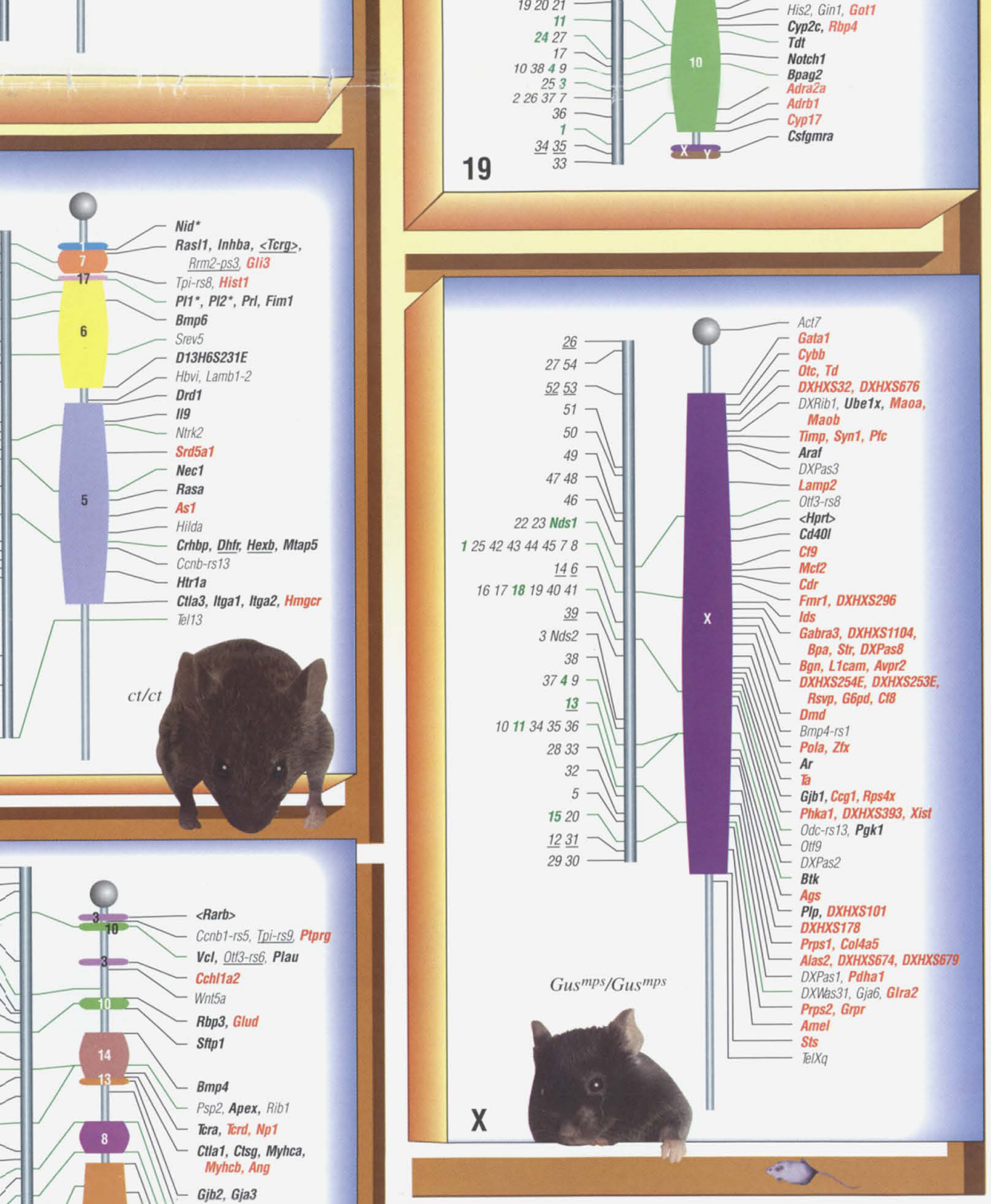
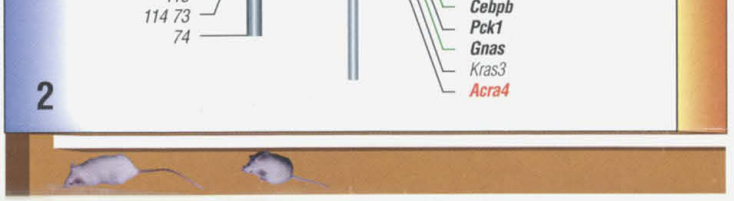


FIGURE LEGEND

The chart shows a current genetic map of the mouse that provides a marker every 0.6 centimorgans (cM) on average, which is roughly equivalent to every 1.1 megabases. The genetic markers are of two types: 1518 simple sequence length polymorphisms (SSLPs), shown on the left, and 1098 gene-based loci, shown on the right.

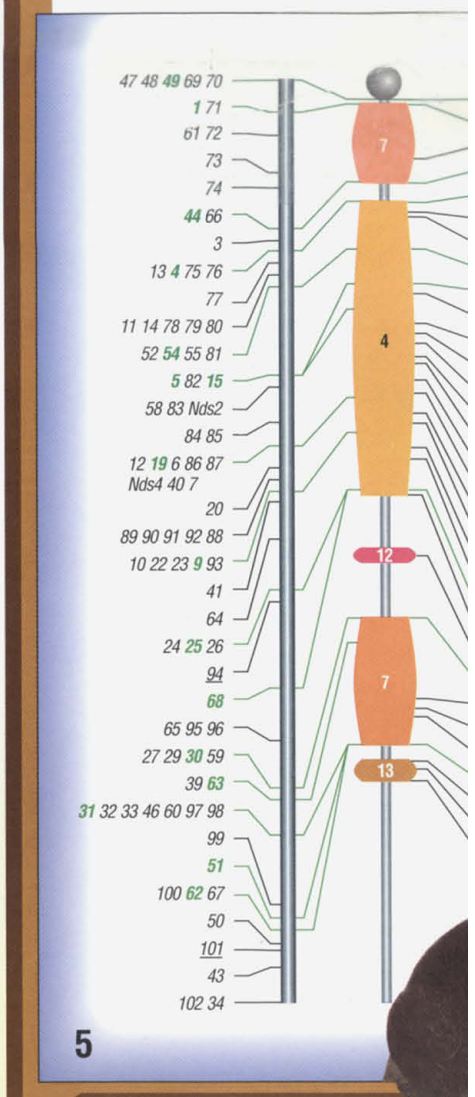
SSLP Map. The SSLP map consists of loci defined by polymerase chain reaction (PCR) assays, each involving a specific pair of PCR primers flanking the site of a short repeat sequence of variable length



REPRESENTATIVE SINGLE-GENE MOUSE MODELS FOR HUMAN GENETIC DISORDERS

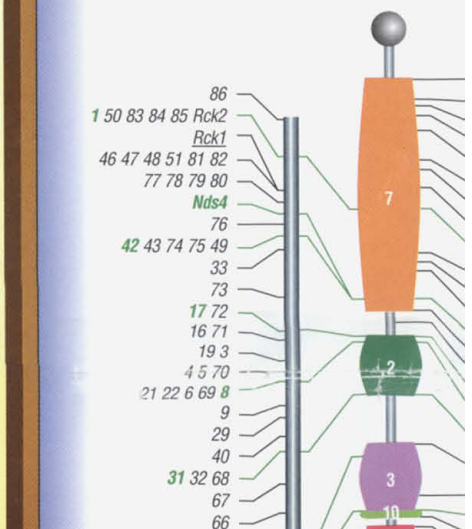
Mouse gene*	Mouse chr	Human condition	Human chr
Apc^{Min}	18	Adenomatous polyposis coli	5q21-q22
Ar^{Tfm}	X	Androgen insensitivity	Xq11.2-q12
Btk^{xid}	X	X-linked agammaglobulinemia	Xq21.33-q22
Dmd^{mdx}	X	Duchenne muscular dystrophy	Xp21.3-p21.2
Gli3^{Xt}	13	Greig cephalopolysyndactyly	7p13
Gus^{mps}	5	Mucopolysaccharidosis type VII	7q22
Hbath	11	Alpha-thalassemia	16p13.3
Hbbth	7	Beta-thalassemia	11p15.5
Kit^{W}	5	Piebaldism	4p11-q22
Otc^{spf}	X	Ornithine transcarbamylase	Xp21.1
p^{p}	7	Tyrosinase-positive (type II) oculocutaneous albinism	15q11-q12
Pah^{enu2}	10	Phenylketonuria	12q22-q24.2
Pax3^{Sp}	1	Waardenburg syndrome type 1	2q35-q37
Pax6^{Sey}	2	Aniridia	11p13
Pit1^{dw}	16	Pituitary hormone deficiency combined	3q
Plp^{jp}	X	Pelizaeus-Merzbacher disease	Xq21.33-q22
Pmp22^{Tr}	11	Charcot-Marie-Tooth disease type 1A	17p12-p11.2
Rd2^{Rd2}	17	Retinitis pigmentosa	6p21.2-cen
Sry^{Sxr}	Y	Gonadal dysgenesis	Yp11.2-pter
Tyr^{c}	7	Tyrosinase-negative (type I) oculocutaneous albinism	11q14-q21

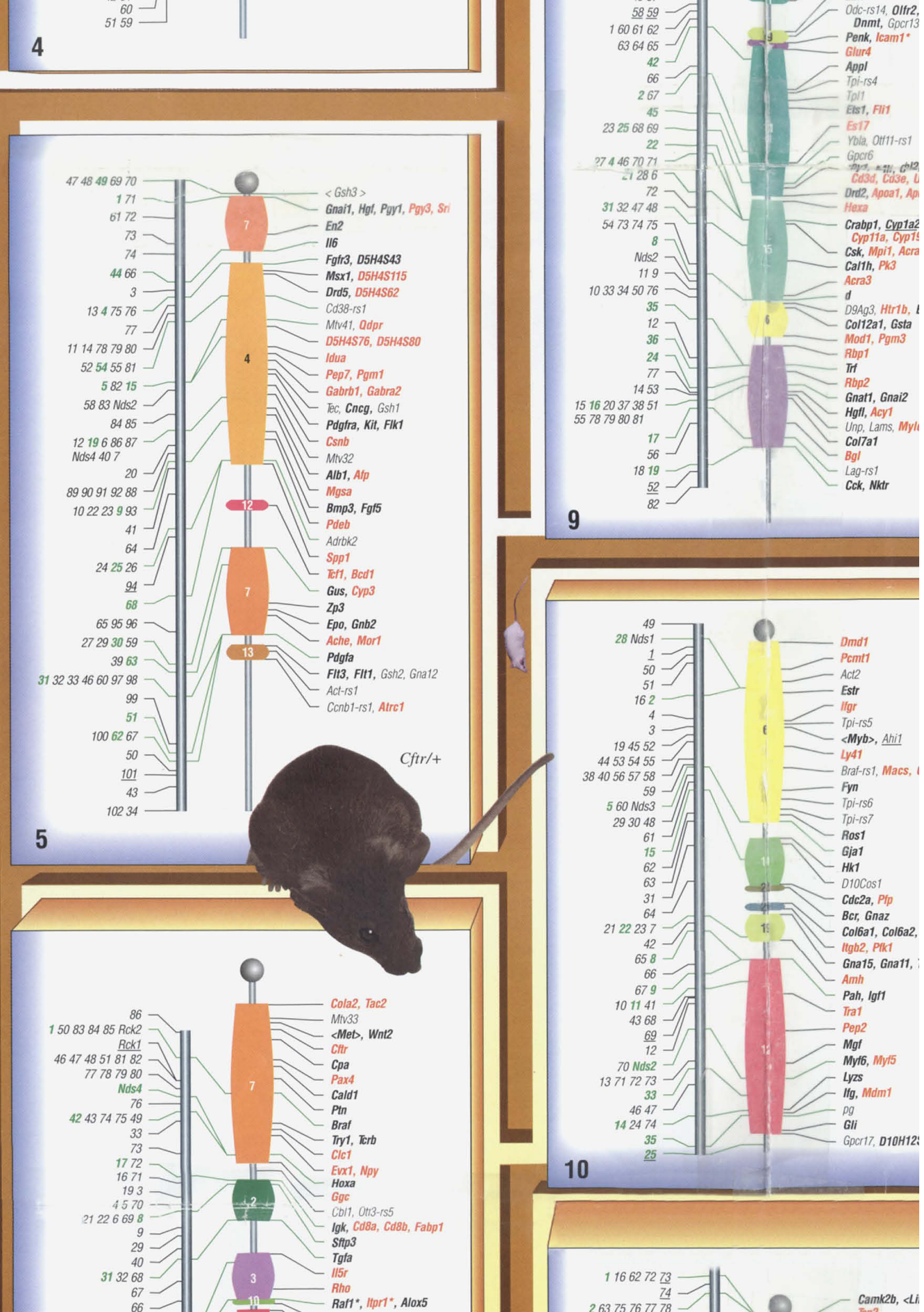
*Superscripts represent alleles



REPRESENTATIVE MOUSE POLYGENIC DISORDERS THAT MAY BE MODELS FOR CERTAIN HUMAN CONDITIONS

Disorder	Strain
Alcoholism/drug addiction (all opiates)	C57BL/6J
Asthma	A/J
Atherosclerosis	C57BL
Audiogenic seizures	DBA
Cleft palate	A
Deafness	LP

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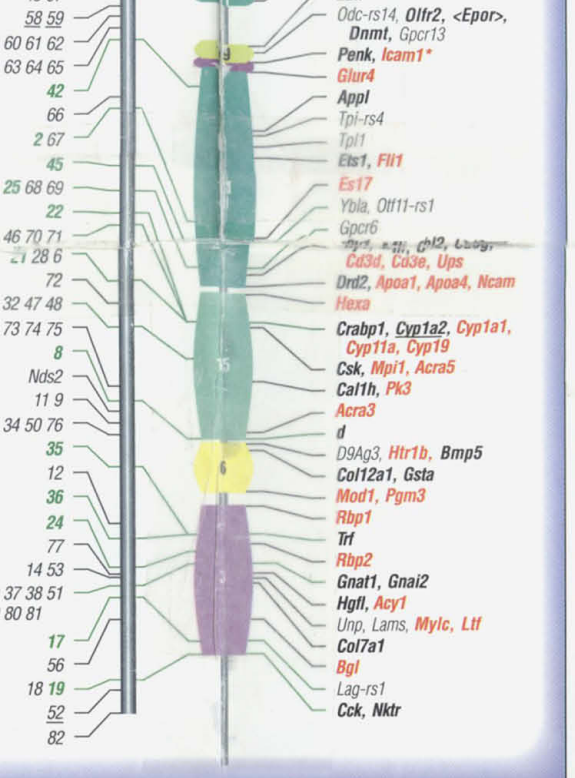
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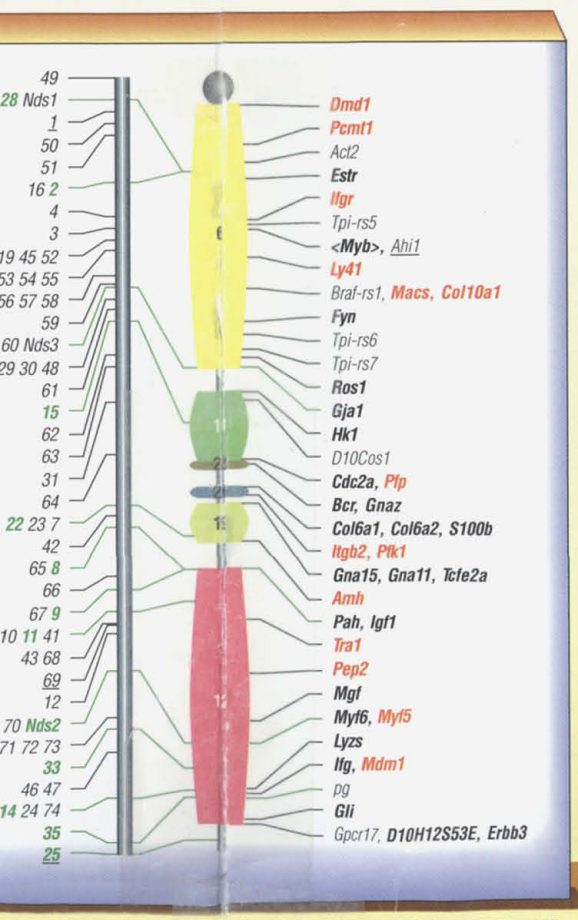
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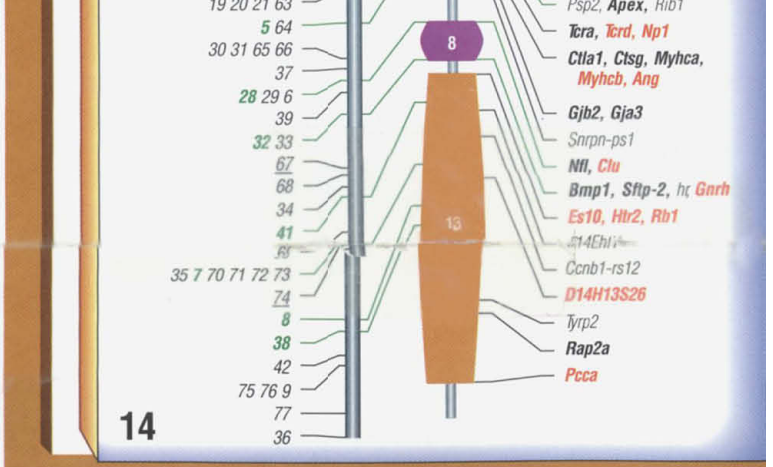
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Bmp1, Sftp-2, hc, Gnrh
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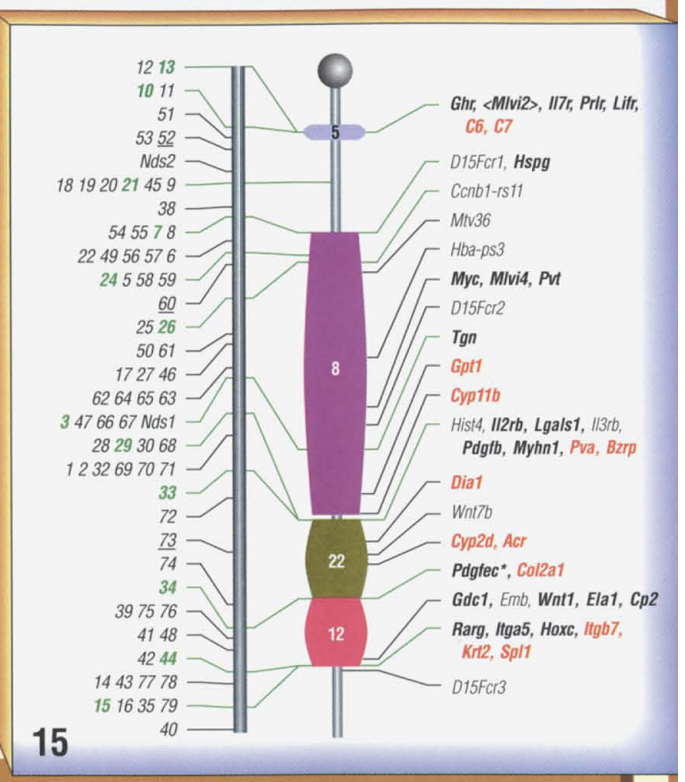
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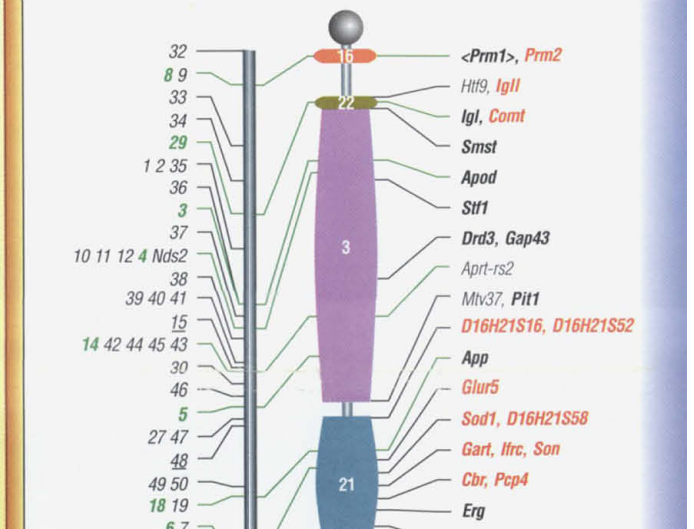
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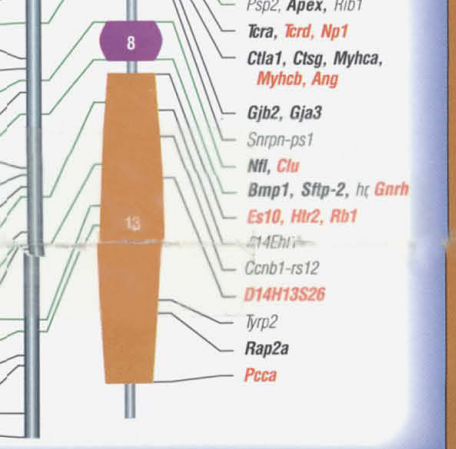
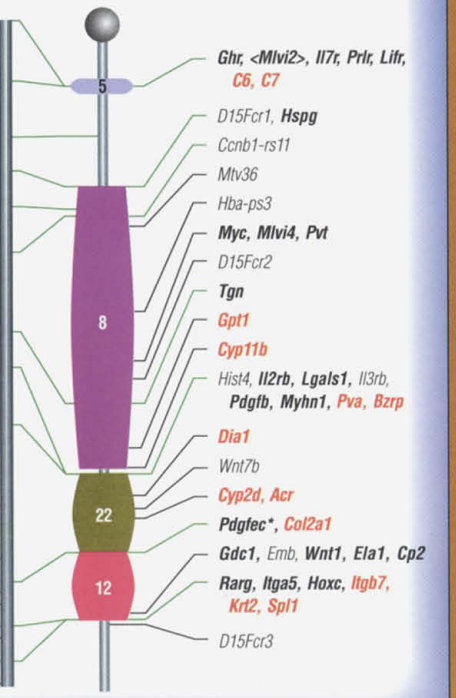




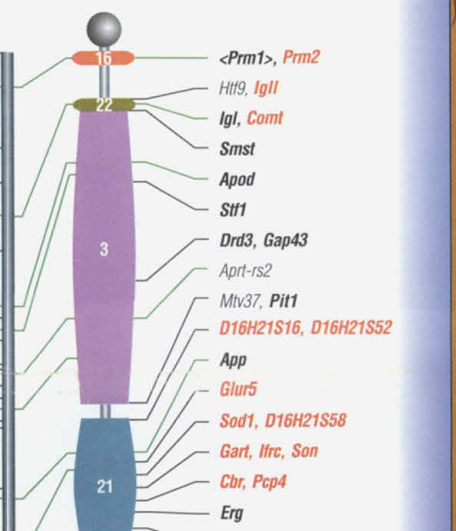


FIGURE LEGEND

The chart shows a current genetic map of the mouse that provides a marker every 0.6 centimorgans (cM) on average, which is roughly equivalent to every 1.1 megabases. The genetic markers are of two types: 1518 simple sequence length polymorphisms (SSLPs), shown on the left, and 1098 gene-based loci, shown on the right.

SSLP Map. The SSLP map consists of loci defined by polymerase chain reaction (PCR) assays, each involving a specific pair of PCR primers flanking the site of a short repeat sequence of variable length — in most cases, $(CA)_n$. These markers tend to be highly polymorphic among inbred laboratory strains, making them especially useful for genotyping intraspecies crosses. Most were based on anonymous (random) sequences, although about one-tenth were taken from repeats found in known genes. The SSLP markers shown were all genotyped in a single interspecies F_2 cross [W. Dietrich *et al.*, *Genetics*, **131**,423 (1992)]. The data were subjected to a mathematical error-checking procedure [(S.E. Lincoln and E.S. Lander, *Genomics* **14**, 604 (1992)], with the result that the relative positions of the loci were supported by a likelihood ratio of 1000:1, except for underlined loci, for which the ratio was >10:1. For nearly 98% of the markers, which were developed at the Whitehead Institute/MIT Center for Genome Research (WI-CGR), Cambridge, MA, formal locus names have been abbreviated. For example, D17Mit3 is shown as 3 on chromosome 17. The remaining SSLPs were developed elsewhere but were also genotyped in the same cross. For these loci, the symbol of the originating laboratory was retained (For example, D4Nds1 is abbreviated Nds1).

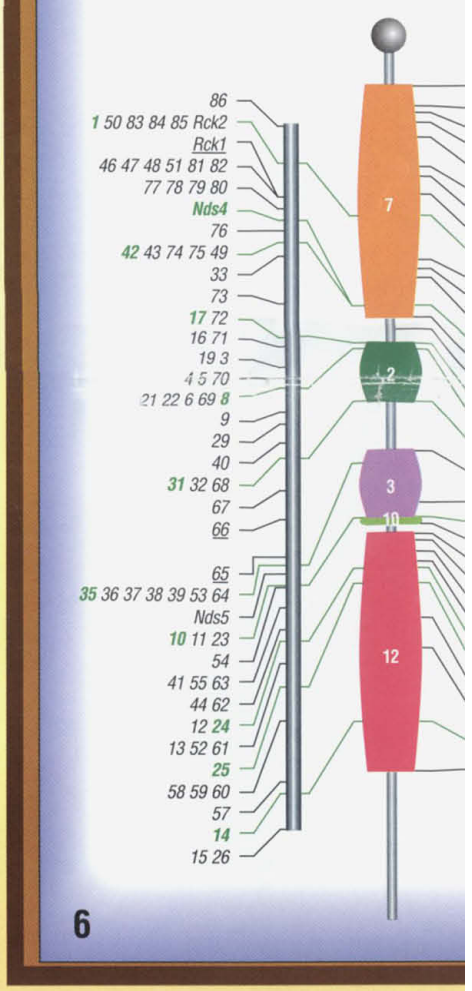
Gene-Based Map. The gene-based map is primarily defined by cloned gene probes that detect restriction fragment length polymorphisms (RFLPs). This map is especially valuable for comparative mapping between the mouse and human genomes. The framework for the map is the Frederick interspecies backcross map [N.G. Copeland and N.A. Jenkins, *Trends Genet.* **7**,113 (1991)], shown in *black type*. Because these loci were all mapped in a single backcross and subjected to mathematical error-checking, the orders of loci were supported by a likelihood ratio of 1000:1, except for underlined loci for which the ratio was >10:1. Loci shown in ***red*** (■) consist of additional genes and anonymous DNA segments mapped in human and mouse that were not mapped in Frederick, but were reported in the 1993 Mouse Chromosome Committee Reports (*Mammalian Genome*, in press); the positions of these genes were inferred on the basis of mapping information in these committee reports. Loci in brackets <> were used to align the maps with respect to the centromere. The position of the centromere is indicated by a circle at the top of each chromosome. Chromosome lengths were drawn to scale on the basis of a 1600-cM genetic length. A break in the distal region of chromosome 11 indicates that this chromosome is slightly longer than predicted by a 1600-cM map.

Integration of Maps. The two genetic maps were integrated by mapping approximately 250 of the SSLPs from the WI-CGR in the Frederick interspecies backcross. These SSLPs are indicated in

*Superscripts represent alleles

REPRESENTATIVE MOUSE POLYGENIC DISORDERS THAT MAY BE MODELS FOR CERTAIN HUMAN CONDITIONS

Disorder	Strain
Alcoholism/drug addiction (all opiates)	C57BL/6J
Asthma	A/J
Atherosclerosis	C57BL
Audiogenic seizures	DBA
Cleft palate	A
Deafness	LP
Dental disease	C57BL, BALB/c
Diabetes	NOD
Epilepsy	EL, SWXL-4
Granulosa cell tumors of the ovary	SWR
Germ cell tumors of the ovary	LT
Germ cell tumors of the testis	129
Hemolytic anemia	NZB
Hepatitis	BALB/c
Hodgkin's disease (pre-B cell lymphoma)	SJL
Hypertension	MA/My
Kidney adenocarcinoma	BALB/cCd
Leprosy *(M. leprae)*	BALB/c
Leukemia	AKR/J, C58/J, P/J
Lung tumors	A, MA/My
Measles	BALB/c
Osteoporosis	DBA
Polygenic obesity	NZB, NZW
Pulmonary tumors	A/J
Rheumatoid arthritis (Sjögren's syndrome)	MRL/Mp
Spina bifida	CT
Systemic lupus erythematosus	NZB, NZW
Whooping cough (pertussis)	BALB/c



GENOME MAPS IV

SCIENCE COORDINATOR: **Barbara R. Jasny**

AUTHORS: **Neal G. Copeland, Debra J. Gilbert, and N**
Research Program, National Cancer Institute, Frederick, M
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C. Miller, William F Dietrich, Robert G. Steen, Stephe
Diane C. Joyce, Mark Merchant, Michael Wessel, Hill
Mary Pat Reeve, Mark J. Daly, Robert D. Dredge, An
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REVIEWERS: **Stephen D.M. Brown**, St. Mary's Hospital
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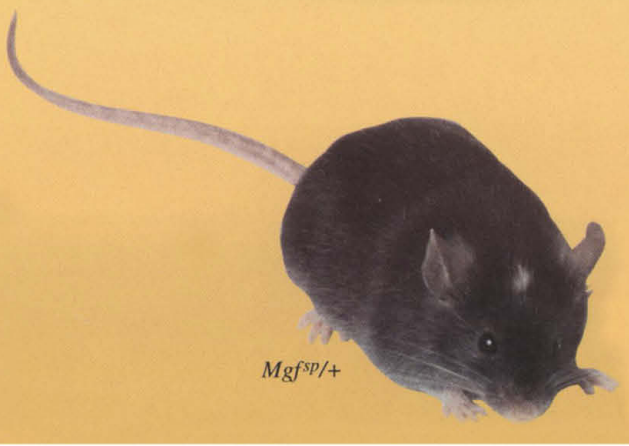
Bethesda, MD. **Mary F. Lyon**, Medical Research Counci
Pearson, Johns Hopkins Medical School, Baltimore, MD
University Medical Center, Durham, NC.

GRAPHIC ARTIST: **Susan Nowoslawski**

PHO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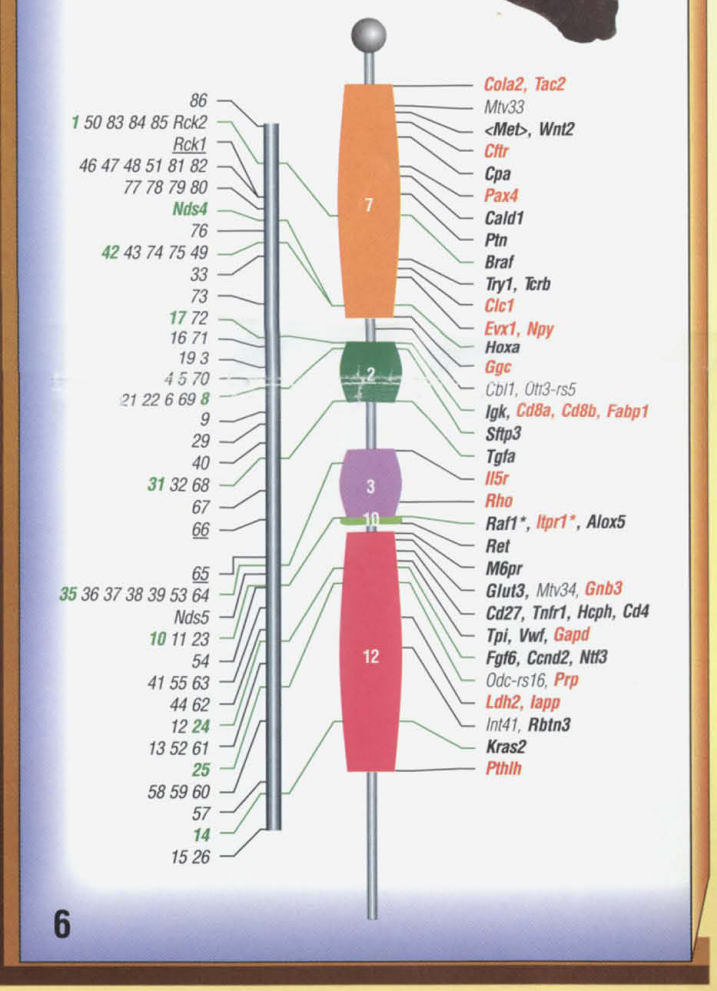
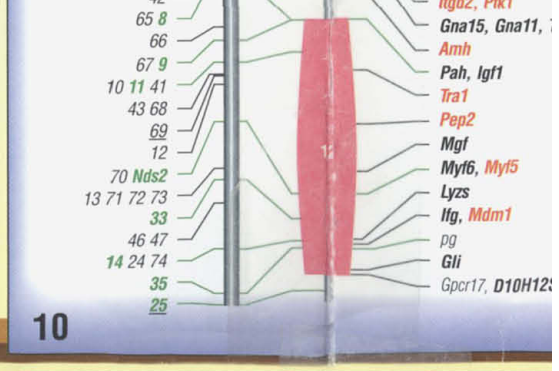
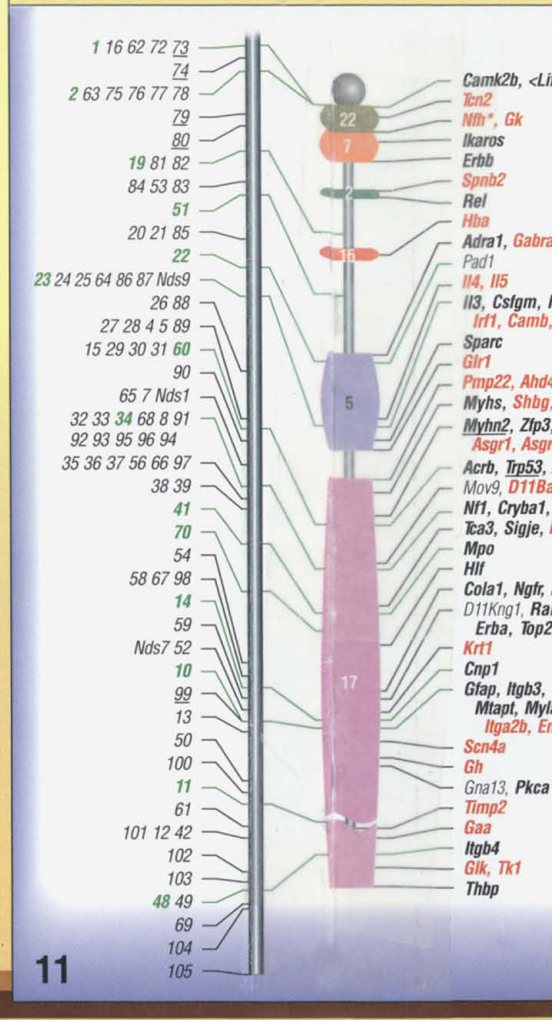






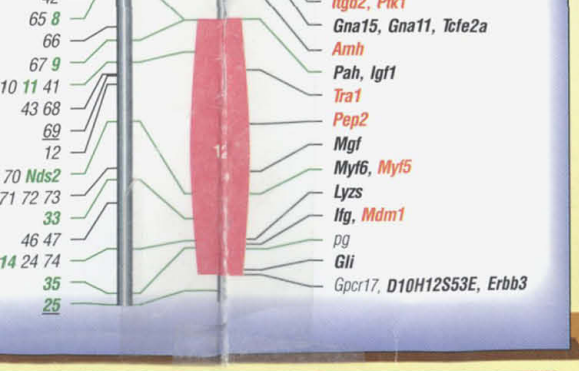
ENOME MAPS IV

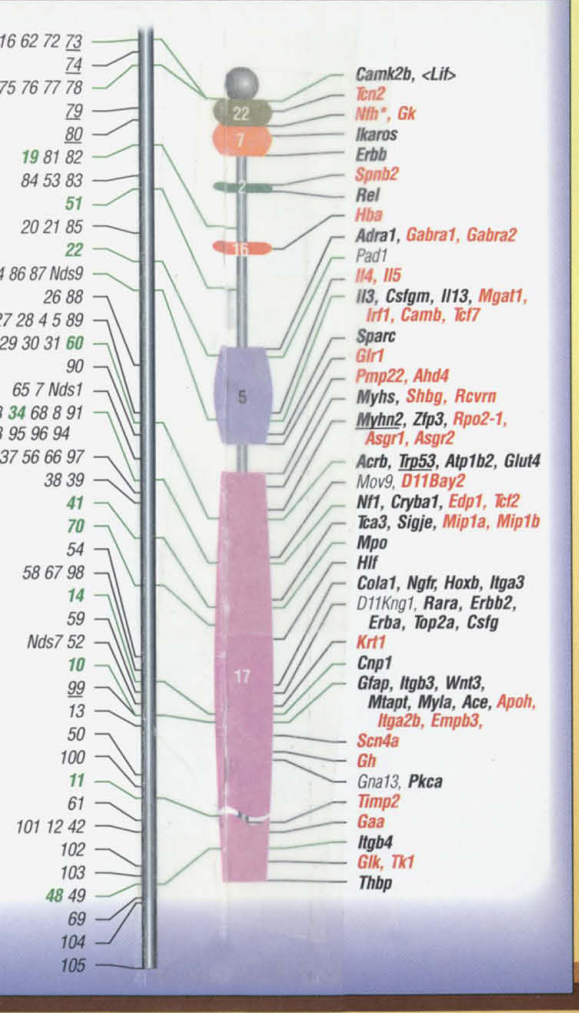
VCE COORDINATOR: **Barbara R. Jasny**

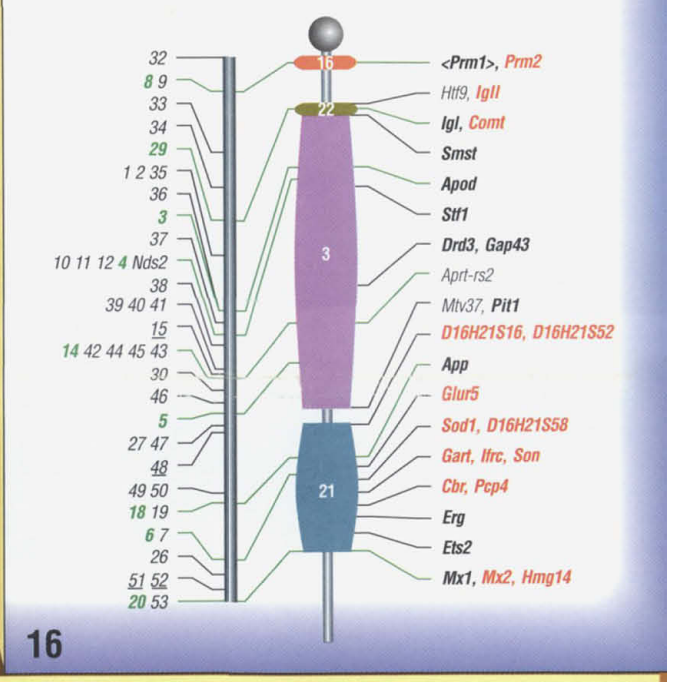
HORS: **Neal G. Copeland, Debra J. Gilbert, and Nancy A. Jenkins,** ABL - Basic arch Program, National Cancer Institute, Frederick, MD. **Joseph H. Nadeau, n T. Eppig, and Lois J. Maltais**, The Jackson Laboratory, Bar Harbor, ME. **Joyce iller, William F Dietrich, Robert G. Steen, Stephen E. Lincoln, Alix Weaver, e C. Joyce, Mark Merchant, Michael Wessel, Hillary Katz, Lincoln D. Stein, y Pat Reeve, Mark J. Daly, Robert D. Dredge, Andre Marquis, Nathan dman, and Eric S. Lander,** Whitehead Institute/MIT Center for Genome arch, Cambridge, MA.

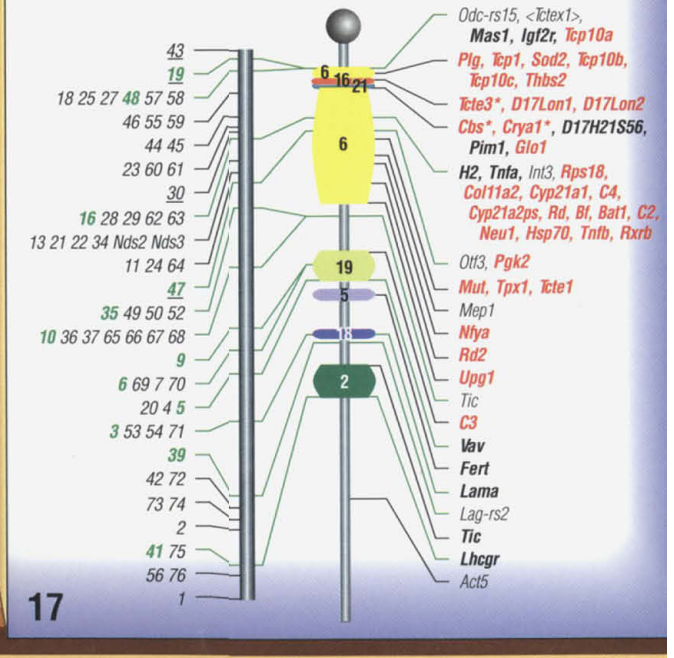
EWERS: **Stephen D.M. Brown**, St. Mary's Hospital, London, U.K. **Jean Louis het**, Institut Pasteur, Paris, France. **Christine Kozak,** National Institutes of Health, esda, MD. **Mary F. Lyon**, Medical Research Council, Oxford, U.K. **Peter L. son**, Johns Hopkins Medical School, Baltimore, MD. **Michael F. Seldin**, Duke ersity Medical Center, Durham, NC.

PHIC ARTIST: **Susan Nowoslawski** PHOTOGRAPHER: **Stan Short**









TECHNOLOGIES

uzzles, mazes: for life scientists, every answered question asks a hundred more. For over 2
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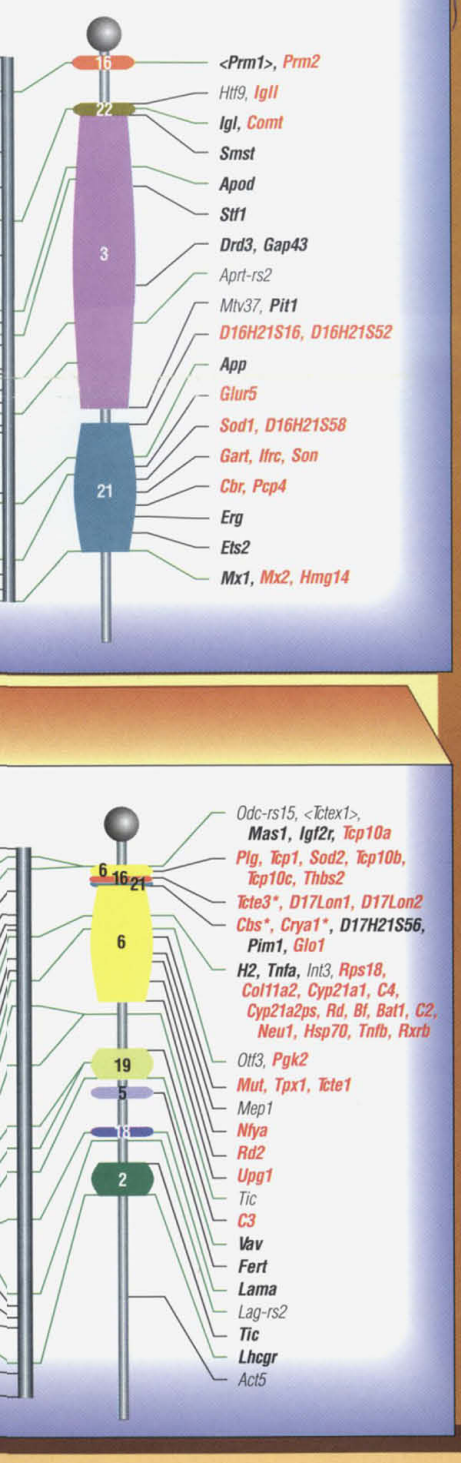

loci were supported by a likelihood ratio of 1000:1, except for underlined loci for which the ratio was >10:1. Loci shown in ***red*** (■) consist of additional genes and anonymous DNA segments mapped in human and mouse that were not mapped in Frederick, but were reported in the 1993 Mouse Chromosome Committee Reports (*Mammalian Genome*, in press); the positions of these genes were inferred on the basis of mapping information in these committee reports. Loci in brackets <> were used to align the maps with respect to the centromere. The position of the centromere is indicated by a circle at the top of each chromosome. Chromosome lengths were drawn to scale on the basis of a 1600-cM genetic length. A break in the distal region of chromosome 11 indicates that this chromosome is slightly longer than predicted by a 1600-cM map.

Integration of Maps. The two genetic maps were integrated by mapping approximately 250 of the SSLPs from the WI-CGR in the Frederick interspecies backcross. These SSLPs are indicated in ***green*** (■), with lines connecting their locations in the two crosses.

Mouse-Human Homology. The colored segments within each chromosome map indicate known regions of synteny or linkage conservation between the mouse and human genomes. The correspondence is based on those mouse genes whose human homologs have been mapped in the human genome; these loci are indicated in ***bold***. For loci mapping near the boundary of two human homology segments, those loci mapping to the proximal segment are marked with an asterisk.

Table. The table included in the chart contains a representative list of single-gene mouse models for human genetic disorders. These mouse genes are altered in the corresponding human hereditary disorders. Also included is a representative list of mouse polygenic disorders that may be models for certain human conditions.

Further Information. Further information about the maps and their construction, the precise locations of the mouse-human homologies, and further sources of information concerning the markers can be found in the accompanying article (N.G. Copeland *et al.*) in the 1 October, 1993 issue of *Science*.

Human homology color key

1	2	3	4	5	6	7	8	9	10	11	12
13	14	15	16	17	18	19	20	21	22	X	Y

5 cM

db/db

ks a hundred more. For over 25 years,
logy, cell biology and immunology.
reliability.