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COMMUNICATIONS

If you want advice on gene nomenclature, or have other queries concerning *Mouse Genome*, contact the editor

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Telephone 071 723 1252; FAX 071 706 3272

DEADLINES

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<td>Dr Festing</td>
</tr>
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MEMBERSHIP OF THE NOMENCLATURE COMMITTEE

The International Committee on Standardized Genetic Nomenclature for Mice has voted to change the terms of its members to five year periods, with the possibility of re-election. Current members will retire, or come up for re-election, at annual intervals in the order in which they were originally elected to the Committee. The Committee welcomes nominations, which should be sent to M.T. Davisson. A list of current Committee members, with addresses, follows.

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ANNOUNCEMENTS

COMMITTEE ON STANDARDIZED GENETIC NOMENCLATURE

Request for Nomination of New Members

The Committee on Standardized Genetic Nomenclature for Mice solicits nominations for new members who will be voted on by the genetic community at the October meeting of the Mouse Genome Mapping workshop and/or by written ballot. Five-year terms will begin and expire in April of each year. Remember that the purpose of the Committee is to establish new nomenclature guidelines as they are needed, to encourage fellow scientists to use good genetic nomenclature and to provide advice to others on mouse genetic nomenclature. Members must be willing to work on Committee business, for example, serving on subcommittees to resolve nomenclature issues.

The Committee was formed in 1939 to ensure standardization of strain and gene symbols. It has been chaired by Drs George Snell, Margaret Green and Mary Lyon. The current chairwoman is Dr Muriel Davisson. The Committee is also the owner of Mouse Genome via Mouse News Letter Limited, a company registered in the UK. We try to have members of the Committee provide a wide representation of different areas of mouse genome mapping, from traditional to molecular techniques, and to provide wide geographic representation. Please keep this in mind when making your nominations. A list of the current members of the Committee appears on p.iii. The three people who will rotate off the Committee in April 1992 are M.F.W. Festing, J.-L. Guénet, and M.F. Lyon. Send nominations to Dr. Muriel T. Davisson, The Jackson Laboratory, 600 Main Street, Bar Harbor, ME 04609, USA.

HUGO - MOUSE GENOME COMMITTEE

Request for Nomination of New Members

The HUGO - Mouse Genome Committee will meet at the forthcoming Mouse Genome Mapping Workshop at Lunteren on 14-18 October 1991. One item for consideration will be the election of three new members. Mouse geneticists are asked to make nominations for new members now, to be elected by ballot of all participants at the Workshop. The Committee exists to further the representation and collaboration of mouse geneticists in the human genome project. Members are chosen to give a wide representation both geographically and in aspects of mouse genetics.

The present members are P. Avner (France), R. Balling (Germany), S. D. M. Brown (UK), V. M. Chapman (USA), M. T. Davisson (USA), J.-L. Guénet (France), I. Jackson (UK), M. F. Lyon (UK), K. Moriwaki (Japan), K. Paigen (USA), J. Peters (UK), T. Roderick (USA).

Please send your nominations to Dr. M. F. Lyon, MRC Radiobiology Unit, Chilton, Didcot, Oxon OX11 0RD, UK.
FIFTH INTERNATIONAL WORKSHOP ON MOUSE GENOME MAPPING

This meeting will take place at Lunteren, The Netherlands, from October 14-18, 1991.

Enquiries should be made to

• Dr. P. Demant,
The Netherlands Cancer Institute,
Plesmanlaan 121,
1066 CX Amsterdam, The Netherlands.

SECOND MAMMALIAN GENETICS AND DEVELOPMENT WORKSHOP

This Workshop will be held in the Geological Society lecture theatre, Burlington House, Piccadilly, London on Tuesday 5th, Wednesday 6th and Thursday 7th November 1991.

Enquiries to :

• Dr. S. Rastan,
Section of Comparative Biology,
Clinical Research Centre, Watford Road,
Harrow, Middlesex HA1 3UJ, UK

or

• Dr. D.B. Whitehouse,
MRC Human Biochemical Genetics Unit,
Wolfson House, 4 Stephenson Way,
London NW1 2HE, UK
NEW INBRED STRAINS OF MICE

CBRB
Inbr. F2+29. Agouti. Origin: Spontaneous translocation Rb(8.17)Iem was found originally by V.S. Baranov in C3HA/Iem mice and carriers were crossed CBA/CaLac mice. After an unknown number of backcrosses, mice were inbred initially by Yu.V.Korogodina (to F11), then by E.V.Moiseeva. About 50% of virgins have mammary tumours at age 15.2 months after a short breeding period, and 90% at age 10.6 months after a longer period (Moiseeva E.V., Kramnik I.B., Slatinova O.V. *Mouse News Letter* (1989) 85:10)

RLRB
Inbr. F25. Black. Origin: Spontaneous translocation Rb(8.17)Iem was found originally by V.S.Baranov in C3HA/Iem mice and carriers were crossed to the stock of black mice of unknown C57BL substrain. Inbreeding of the progenitors was started in 1981 by E.V. Moiseeva. About 70% of virgins have mammary tumours at age 14.1 months, and survival of females with tumours is 15.9 months.

95% mammary cancer in breeding females; mammary tumours develop at about an average of 12.3 months after a short breeding life, and at about 8.4 months when bred for 5 months or more (Moiseeva E.V., Kramnik I.B., Slatinova O.V. *Mouse News Letter* (1989) 85:10).

Correction

In the short paper 'Single day detection of transgenic mice by PCR of toe-clips' by Daniel Pomp and James D. Murray which appeared in *Mouse Genome* (1991) 89:279, the author line should have read Daniel Pomp, James D. Murray and Juan F. Medrano. Our apologies to Dr Medrano for this omission.
<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Gene Symbol</th>
<th>Gene Name</th>
<th>Contributor or Reference</th>
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<tr>
<td>8</td>
<td>Abl&lt;sup&gt;m1&lt;/sup&gt;</td>
<td>Abelson leukaemia oncogene m1</td>
<td>Schwartzberg et al (1991) Cell 65:1165</td>
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<td>4</td>
<td>C8b</td>
<td>complement component 8, beta subunit</td>
<td>Tanaka et al (1991) Immunogenetics 33:18</td>
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<td>DNA segment, Chr 1, Lehrach 1</td>
<td>Youngblood et al (1991) Genomics 10:270</td>
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<td></td>
<td>Es-29</td>
<td>Deimling (1991) Mouse Genome 89:549</td>
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<td>X</td>
<td>Glra2</td>
<td>Derry &amp; Barnard (1991) Genomics 10:593</td>
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</table>
7 Glud-2 glutamate dehydrogenase-2
2 Glvr-1 gibbon ape leukaemia virus receptor-1
10 Hal histidine ammonia lyase
1 Hlx H2.0-like homeobox gene
- Hpd 4-hydroxyphenyl pyruvic acid dioxygenase
- Hpdhy 4-hydroxyphenyl pyruvic acid dioxygenase, hypertyrosinemia
4 Hrs-1 minor satellite DNA-1
4 Hrs-2 minor satellite DNA-2
9 Hsp86-1ps3 heat shock protein pseudogene 3
4 Hsp86-1ps4 heat shock protein pseudogene 4
X Hst-3 hybrid sterility-3
6 Iapp islet amyloid polypeptide
16 Ifgt interferon gamma transducer
7 Il4r interleukin 4 receptor
13 jd juvenile depilation
- Lef-1 lymphoid enhancer-binding factor 1
- ll long-lived
19 Lpc-1 lipocortin-1
8 Mel Mel protooncogene
- Mse-1 esterase modifying locus-1
6 Ntf-3 neurotrophin-3
5 pal-1 proviral insertion pal-1 (provisional)
1 Pdc phosducin
- rac-1 ras-related C3 botulinum substrate 1 (provisional)
- rac-2 ras-related C3 botulinum substrate 2 (provisional)
11 Rara retinoic acid receptor alpha
15 Rarg retinoic acid receptor gamma
2 Rmp-2 resistance to mouse pox-2
17 Rmp-3 resistance to mouse pox-3
- rmy rimy

Prochazka & Leiter(1991) Mouse Genome 89:553
Sweet et al (1991) Mouse Genome 89:574
Travis et al (1991) Genes Dev.5:880
Deimling(1991) Mouse Genome 89:549
Keightley & Hawkins(1991) Mouse Genome 89:410
Rnu3b-1  U3B small nuclear RNA-1
Rnu3b-2  U3B small nuclear RNA-2
Rnu3b-3  U3B small nuclear RNA-3
Rnu3b-4  U3B small nuclear RNA-4
Rnu3b-rs1 U3B small nuclear RNA related sequence-1
Rnu3b-rs2 U3B small nuclear RNA related sequence-2
Rnu3b-rs3 U3B small nuclear RNA related sequence-3
Rnu3b-rs4 U3B small nuclear RNA related sequence-4
Rnu3b-rs5 U3B small nuclear RNA related sequence-5
Rnu3b-rs6 U3B small nuclear RNA related sequence-6
Rpr-1    rod photoreceptor protein-1
Scn1a    sodium channel, type I, alpha polypeptide
Scn2a    sodium channel, type II, alpha polypeptide
Scn3a    sodium channel, type III, alpha polypeptide
Shbg     sex hormone binding protein
stg      stargazer
Tshr     thyroid-stimulating hormone receptor
Ttp      tris-tetraprolin
wag      waggler
Xist     X-linked specific transcripts

Noebels et al (1990) Epilepsy Res. 7:129
Sweet et al (1991) Mouse Genome 89:552
### NEW RESERVED GENE SYMBOLS

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<td>Cnca</td>
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<tr>
<td>Cncg</td>
<td>cyclic nucleotide gated channel, cGMP gated</td>
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<tr>
<td>Eef-1</td>
<td>eukaryotic translation elongation factor</td>
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### NEW CHROMOSOME ANOMALY

T(1;12)52H  
C.Rasberry, Harwell

### CHANGED GENE SYMBOLS

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<td>Hsp86-2</td>
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<td>Hsp86-3</td>
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<td>Locus</td>
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<td>At-3...Spna-1...(Tgfb-2,Ilx)</td>
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<tr>
<td>1</td>
<td>cen...Gls...(Cd28,Ctla-4)...Inha</td>
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<td>Fn-1...Tp-1...(Vil,Lsh)...Des...Inha...Akp-3...Acrn...Sag</td>
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<tr>
<td>1</td>
<td>Ly-5...D1Leh1...At-3</td>
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<td>1</td>
<td>Rnu3b-rs1 has been localized to 1A4 to 1C1 by in situ hybridization</td>
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<tr>
<td>2</td>
<td>cen...Abl...Hc...Ggta-1</td>
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<td>2</td>
<td>Hc...D2Leh1...Gcg...Fshb...D2Leh2...Ltk</td>
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<td>2</td>
<td>Hc...Neb...Pmv-7... (Scn2a,Scn3a)...Scnla...Mpmv-14</td>
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<td>Rnu3b-rs2 has been localized to 2F to 2G by in situ hybridization</td>
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<td>Scn2a and Scn3a are separated by a maximum of 600 kb</td>
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<td>Car-2...Hsp86-1ps2...Gba</td>
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<td>(D3Leh2,Evi-1)...Fgfb...D3Leh1...Fgg</td>
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<td>cen...Mtv-14...Hsp86-1ps4...Mtv-17</td>
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<td>c...Pkcb...Il4r...Int-2</td>
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<td>9</td>
<td>cen...D9Pas1...Thy-1...Mpi-1...Mod-1</td>
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<td>9</td>
<td>d...Hyal-1...Bgl-s</td>
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<td>9</td>
<td>Hsp86-1ps3...Icam-1...Fli</td>
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Hal has been localized to 10C2 to 10D1 by in situ hybridization

Rnu3b-rs4 has been localized to 10A4 to 10B2 by in situ hybridization

Sl...D10Led1

cen...Erbb...Adra-1...D11Sel1...Il-3... (Erba,Erbb-2).

Erbb...Adra-1...Pad-1...df...(Sparc,Csf6g,Il-3)...Myhs... (Rpo2-1,Asg)... Edp-1...Erba...Gh

Myhs...(D11Leh1,Trp53)...Evi-2...Mpo... D11Leh2...Pkca

Rara has been localized to 11D by in situ hybridization

Rnu3b-1,Rnu3b-2,Rnu3b-3 and Rnu3b-4 have been localized to 11C to 11D by in situ hybridization

Shbg...Hsp86-1ps1...Int-4

Tk-1 has been localized to 11E1 to 11E2 by in situ hybridization and by chromosome aberration analysis

Pomc-1...D12Leh1...D12Nyu5

Rnu3b-rs5 has been localized to 12D3 to 12F by in situ hybridization

Tshr...Hsp86-1...Igh

cen...Dhfr...Htrla...Ctla-3

cen...jd...pe

Gldu...Np-1...sys...Es-10

Rarb has been localized to 14A by in situ hybridization

Rarg has been localized to 15E to 15F3 by in situ hybridization

uw...Gpt-1...wag...Gdc-1...Ca

uw...stg...bt

cen...Grl-1...Pdgfr...Adrb-2...Mbp

D18Leh1...Fgfa...Grl-1...Ili...D18Leh2...Mbp

Mbp has been localized to 18E2 to 18E3 by in situ hybridization

cen...Cyp2c...Gin-1...Adra-2...Adrb-1

cen...Ly-1...Ly-10...Ea-4...Lpc-1...Xmv-18

Emv-22...Got-1...Cyp17
19 Ly-1...Fth...Pax-9
19 ocd...ab; ocd...ru
19 Rnu3br-rs6 has been localized to 19A to 19C1 by in situ hybridization

X Ags...Plp...DXWas31...Amg
X (Ccg-1,Phka)...(Xist,DXPas19)...Pgk-1
X Ccg-1...(Phka,DXCrc177,DXCrc318,Xist)...DXCrc13...Pgk-1
X cen...Cybb...Hprt...DXS144Eh...Gabra-3...Rsvp...(DXS253Eh,Cf-8)...Dmd...Pgk-1...DXPas2...Plp...DXPas1...Glr-2
X cen...Cybb...Otc...((Timp,Araf,Syn-1)...Xlr-1)...Hprt
X cen...G6pd...Ta...Xcat
X Otc...Maoa...((Pfc,Timp)...Hprt...Cf-9
X Pfc has been localized to XA3 by in situ hybridization
X Ts2R...Mobo
X (Zfx,DXCrc28,DXCrc57,DXCrc202,DXCrc140,DXCrc190)...(Ar,DXCrc131)...(ta,DXCrc169)...(DXCrc94,DXCrc171,Ccg-1)...(Phka,DXCrc177,DXCrc187,DXCrc318)...DXCrc13...Pgk-1...(DXCrc112,DXCrc323)...(DXCrc47,DXSmh44)...DXCrc98

Sweet & Bronson (1991) J.Hered.82:140
Derry & Barnard (1991) Genomics 10:593
Grant & Chapman (1991) Oncogene 6:397
INTRODUCTION

Inbred mice have been used more extensively than those of any other species of laboratory mammal. A total of about 400 strains are listed here, but some of the more widely used strains have become divided into substrains among which there are detectable genetic differences. Some strains are recorded in the literature but are now extinct. In most cases such strains have been excluded from this list, but there are some strains for which there are no known holders, but which we can not be sure are extinct.

Many of the strains are related, having come from the same outbred colony, or having some other form of common ancestry. On the other hand, there is increasing evidence that laboratory mice have been developed with contributions from more than one species/subspecies of wild mouse. For example, some strains carry the *Mus musculus domesticus* Y-chromosome, while others have the *M.m. musculus* type. Thus, Nishioka (1987) found the following:

**M.m. musculus** type

**M.m. domesticus** type

NOTES ON THE LISTINGS

The number of generations of full-sib mating is given for each strain, but this should be regarded as an approximate figure, as it varies considerably between colonies, and it is very difficult to keep it updated. In any case it is doubtful whether the exact figure has much significance once 30-40 generations have been completed, except possibly in studies of substrain differentiation.

In the case of quantitative characteristics strains have been ranked, and approximately the top and bottom quarter of the strains have been ranked as 'high' and 'low', respectively. Thus, 'low intra strain aggression (13/14)' indicates that in a study of intra-strain aggression the strain in question ranked thirteenth out of fourteen strains being tested. These strain rankings should be treated with some caution, as they depend on exactly which strains happened to be chosen for the study, and the rankings could be altered by environmental influences. In some cases it will be noted that studies by different workers are contradictory. In the case of qualitative characteristics a 'cf' (compare) precedes the number responding out of the number tested. Thus good immune response to X antigen (cf. 4/8) means that the strain was one of four responders out of eight tested.

Where much information is available for a given strain this has been classified into 'Behaviour', 'Life-span and spontaneous disease', etc. The heading 'Drugs'
refers to response to any xenobiotic such as chemicals and drugs, and also includes response to irradiation.

In compilations of this sort, substrain differences present a problem. Where there are major substrains of an inbred strain, an attempt has been made to show which one was involved in each study. However, references have been given, and where necessary the original article should be consulted.

I am aware of a number of inconsistencies and omissions in this listing which I have not had time to correct. For example, many of the references do not list the last page number. However, I would appreciate it if people could send me reprints and other information which describe strain characteristics which are not listed here.

I would like to thank Mouse News Letter Ltd. for financial help in preparing this list.

INBRED STRAINS AND THEIR CHARACTERISTICS

A

Inbr: More than F150. Albino. Genet: a, b, c. Origin: Dr L. C. Strong, 1921, from a cross between the Cold Spring Harbor and Bagg albino random-bred stocks (and therefore related to BALB/c). Internationally distributed, Strain A was the third most widely used strain in cancer and immunology research (Festing, 1969), though its popularity has probably declined recently. Although it may be classified as a general-purpose strain, it is well known for a high susceptibility to induction of congenital cleft palate by cortisone and a high spontaneous incidence of lung adenomas, as well as developing a high incidence of lung tumours in response to carcinogens. Shimkin and Stoner (1975) suggest that this response may be used as a rapid in vivo assay for carcinogenesis. The strain also suffers from a defect in macrophage function somewhat resembling the mutant lps found in C3H/HeJ (Vogel et al 1981).

The following main substrains are recognised, though they have not been defined by genetic markers:

A/St Maintained by Strong.

A/He Strong to Heston, 1938.

A/GrFa Main British substrain, Strong to Gruneberg 1932, and mainly distributed by Falconer.

A/WySn Strong to Bittner 1927, to Wooley, to Snell, 1951.

A/J Strong to Cloudman 1928, to Jackson Laboratory 1947, now widely distributed.

Behaviour

Low intra-strain aggression (13/14) (Southwick and Clark, 1966), low food drive (15/15) and exploratory activity (15/15) (Thompson, 1953). Low spontaneous bar pressing activity (12/14), low open-field activity (13/14 and 14/14 in J and He substrains), low social grooming during aggressive encounters (12/14 in He substrain) and high tail rattling score (3/14 and 5/14 in J and He substrains) during aggressive encounters (Southwick and Clark, 1968). High shock avoidance learning (3/9) (Bovet et al., 1966), high avoidance conditioning (2/9) (Royce, 1972), and (2/6 males, 1/6 females) (Royce et al., 1971), but poor shock avoidance.

Life-span and spontaneous disease

Primary lung tumours 6% in male, 32% in female and 26% in virgin females in J substrain; 44% in males, 23% in females and 30% in virgin females in He substrains (Hoag, 1963). Zero incidence of lymphatic leukaemia in He substrain, 1% in J substrain. Mammary adenocarcinomata zero in males, 1% in virgin females, 28% in breeding females of J substrain and 54% in breeding females of He substrain (Hoag, 1963). Pulmonary tumours 90% in mice at 18 months (Heston, 1963). Leukaemia 3% in HeJ substrain (Myers et al., 1970). A high proportion of the mammary tumours are of the acinar type (3/7) (Tengbergen, 1970). Lung adenomas 53-64% in BrA and A substrains, but mammary tumours zero (Muhlbock and Tengbergen, 1971). Lung tumours 4-31% and lymphatic leukaemia 10-43% (Festing and Blackmore, 1971). Spontaneous lung tumours occur at rate of 0.21 tumours/mouse at 24 weeks (Poirier et al., 1975).

Life-span in conventional conditions intermediate in both sexes (9/22 = 490 days in males, 13/22 = 590 days in females (Storer, 1966). Life-span in SPF fostered conditions intermediate (8/17 = 512 days) in males and short (3/17 = 558 days) in females (Festing and Blackmore, 1971). Life-span 662 days in males and 688 days in females (Goodrick, 1975). Median life-span 400 days in HeJ substrain (Curtis, 1971).

Spontaneous congenital cleft palate 4% and high susceptibility to teratogenic effects of cortisone, which may be associated with the H-2a allele, (Bonner and Slavkin, 1975). Low incidence of virus-like particles in chemically induced sarcomas (6/6) (Liebelt et al., 1970). Congenital malformations in new-born mice 10% (1/9), including cleft lip and palate and polydactyly (Kalter, 1968). Can be made obese by a suitable diet (Fenton and bowling, 1953). High incidence of amyloidosis (Russell and Meier, 1966). No amyloidosis found by Powers et al. (1976) in He and HeJ substrains, in contrast to previous reports. About 4% incidence of congenital open eyelids (Dagg, 1966). High incidence of cannibalism of young restricted to anatomically defined mutilation and amputation, particularly of neck, lower jaw and digits in Ha substrain (Hauschka, 1952).

Normal physiology and biochemistry


Low systolic blood pressure (17/19) (Schlager and Weibust, 1967). Low peripheral nerve conduction velocity (5/6) (Hegmann, 1972). High concentration of prostaglandin F in epididymis (1/6) (Badr, 1975). High glucose-6-phosphate dehydrogenase and nicotinamide-adenine dinucleotide phosphate levels in


Anatomy

Drugs

Immunology
Develops autoimmune phenomena, immunological deficits with ageing and autoimmunity following neonatal thymectomy (Yunis et al., 1972). Low lymphocyte

Infection

Encephalomyocarditis virus causes diabetes mellitus (cp. 7/14) (Boucher et al., 1975). Highly susceptible to infection by measles virus (cf. 3/6) (Rager-Zisman et al., 1976).

Reproduction

Miscellaneous
Recommended host for the following transplantable tumours: anaplastic carcinoma 15091 AK, hepatoma H6, round cell tumour C 1300 and spindle cell sarcoma Sal (Kaliss, 1972).

AA
AB

ABJ

ABP/Le

ACR
Inbr ?. Albino. Origin: AKR substrain to CIBA 1949. Known as AKR/FuA. Genetic contamination in 1953 (see AKR), renamed ACR. Leukaemia only 10-36% and lung adenomas 54-67% in AKR/FuA substrain (Muhlbock and Tengbergen, 1971). Maint. by A.

AE

AEJ
Inbr (Rk) 70. Black. a Origin: Hollander, Iowa. Ames waltzer stock with miscellaneous markers to M.C.Green 1963. Crossed once to C57BL/10 followed by cross to C57BL/6-AwJ, then b x s. To Roderick 1972. Maintained by Rk.

AG

AKR
Albino: a,b,c. Origin: a dealer named Detwiler in Norristown PA. Carried by Furth as a high-leukaemia strain from 1928 to 1936, then random bred at the Rockefeller Inst. for several generations. b x s by Mrs. Rhoades to F9 then C.Lynch to F21. This strain is best known for it's high incidence of lymphatic leukaemia, and for the Thy-1 T-cell antigen, which is only present in this and a few other strains. Early history is obscure, but Acton et al (1973) found substantial substrain differences, which can best be accounted for by genetic contamination. This could have occurred at the time the strain was maintained by random mating. The strain has an international distribution, ranks about eighth in frequency of use and is widely used in cancer research for its high leukaemia
incidence (Lilly and Pincus, 1973) and in immunology as a source of the Thy-1.1 (theta AKR) antigen, although this varies between substrains. Mice of this strain are viraemic from birth and express in all tissues the ecotropic retrovirus AKV, copies of which are integrated into the genome and which is associated with the development of the leukaemia. In some substrains additional copies of the viral genomes may be integrated (Herr and Gilbert 1982).

Major substrains include:

**AKR/J**
Inbr ?. To Jackson Laboratory 1940. Maintained by J.

**AKR/LwN**
Inbr ?. To Law 1940, to N 1956 at F53. Maint. by N.

**AKR/PuA**
Genetically contaminated and renamed ACR/A. Maint. by A.

**AKR/Cum**
Inbr ?. Origin ?. Differs from other substrains in being Mod-la.

**AKR/PuRdA**
Oak Ridge, to ?, to Rudale, to Netherlands Cancer Institute 1953. Carries Thy-1.1 and appears similar to AKR/J.

**AKR/TlAld**
Carries an autosomal translocation and has a low fertility (Leonard and DeKnudt, 1967)

**Behaviour**

**Life-span and spontaneous disease**
Life-span short (1/22 = 326 days in males, 276 days in females) in conventional conditions (Storer, 1966). Life-span short (1/17 = 350 days in males, 312 days in females) in fostered SPF conditions (Festing and Blackmore, 1971).


**Normal physiology and biochemistry**
Anatomy

Drugs

Immunology

Infection

Reproduction
Poor reproductive performance (22/25); output 0.74 young/female/week, litter size low (22/25) at 4.1 weaned (Festing, 1976a). Intermediate breeding performance (14/24) (Hansen et al., 1973). Low post-implantation loss of embryos (1/8), but high pre-implantation losses (8/8) (Leonard et al., 1971).

Miscellaneous

- **AKXL**

- **AL**
  Inbr: F166. Albino. Genet: a, b, c. Believed to have originated from an illegitimate mating of strain A followed by b x s mating, but should not be considered as a strain of strain A. Very low mammary tumour incidence (Staats, 1976).

  Normal physiology
  High erythrocyte catalase level (3/18) (Hoffman and Rechcigle, 1971).

Drugs
Phenobarbital i.p. does not induce hepatic epoxide hydrase (cf. 3/7) (Oesch et al., 1973). Long hexobarbital sleeping time (9/9) and low liver hexobarbital oxidase level (1/9) (Vesell, 1968).

Immunology
Resistant to the induction of liver amyloid, but a high level of spontaneous amyloidosis (10/10) (Ram et al., 1969). Low susceptibility to induction of amyloid (6/6) (Willerson et al., 1969). Good immune response to synthetic double-stranded RNA (1/7) (strain quoted as ALN, but presumed to be AL/N) (Steinberg et al., 1971). Poor immune response to Vi antigen (cf. 3/5) (Gaines et al., 1965). High anti-DNP antibody concentration (1/7) (Paul et al., 1970).

Reproduction
Good breeding performance with 2.3 young per female per month (5/24) (Hansen et al., 1973).

Miscellaneous
AL/N has only 38 chromosomes, including two translocation submetacentrics involving Robertsonian translocations of chromosomes 5 and 19 (White and Tjio, 1975).

- **AM**

- **AS/Wf**

- **AT/Wf**
ATEB
Inbr (Le) 42. Grey: a,d. Also carries at/+ (atrichosis), eb/+ (eye-blebs). Origin: at arose in DBA/1 at F15 in 1964, eb arose in a non-inbred hairless stock maintained by Hummel in 1960. Balanced stock bxs to M.C.Green 1972, to Lane 1975. at/at mice are sterile. eb/eb mice have defects of eyes, kidneys and feet. Maint. by Le

AU

AX

AXB-
Inbr circa 20+. Set of 48 recombinant inbred strains developed by Nesbitt from A/J x C57BL/6 and C57BL/6 x A/J. (Nesbitt and Skamene 1984). Maint. by Ns.

AY

A2G

Behaviour
Whisker chewing 75% in cages of 2-3 mice by 60 days of age. Whisker trimming seems to be associated with social dominance (Strozik and Festing 1981). Low balsa-wood gnawing activity (3/16) in A2G-hrhr, but high activity in A2G (1/16) (Fawdington and Festing 1980)

Life-span and spontaneous disease
Long life-span in males (13/17 = 640 days) but intermediate in females (8/17 = 644 days), and lung tumours 17-65% in SPF fostered conditions (Festing and Blackmore, 1971).

Normal physiology and biochemistry

Anatomy
Absence of third molar in 7% of cases (1/20) (Festing, 1975b). High incidence of absence of the 3rd. molar, which has been used as a threshold character to study the effects of weak teratogens (Berry and Nickols 1979)
Drugs

Immunology
Incidence of serum antinuclear factor high (2/17) at 28% (Barnes and Tuffrey, 1967). Low antibody affinity to HSA (7/9) (Petty et al., 1972).

Infection
Uniquely resistant among twenty strains tested to infection with diverse strains of pneumotropic and neurotropic influenza viruses. Resistance is due to a dominant autosomal gene, and does not depend on the immune system (Fiske and Klein, 1975; Lindenman et al., 1963). Develops a chronic non-healing lesion on infection with Leishmania tropica, the parasite causing cutaneous leishmaniasis (Howard et al. 1980)

Reproduction
Good breeding performance (7/25), colony output 1.2 young per female per week, litter size at weaning 5.7 (12/25) (Festing, 1976a).

B6NXC3N-.
Set of 7 recombinant inbred strains developed from a cross involving C57BL/6N and C3H/HeN. Maintained by Lm.

BA

BALB/c
Albino: A,b,c. Origin: Stock acquired by H.Bagg in 1913, to MacDowell, to Snell in 1932 (who added the /c). Now widely distributed and among the top 2-3 most widely used inbred strains. The strain is particularly well known for the production of plasmacytomas on infection with mineral oil. These tumours form the basis for the production of monoclonal antibodies. Used as a general-purpose strain in many different disciplines. Good breeding performance and long reproductive life-span. Normally has low mammary tumour incidence but can be infected with the mammary tumour virus by fostering to C3H (which carries the virus), and it then gets a high incidence of mammary tumours.

The history and characteristics of the strain have been reviewed by Potter (1985). Three major substrains trace back to before 1940, and are listed separately below. Data on genetic markers suggest that these substrains have diverged largely through mutation or residual heterozygosity rather than genetic contamination. Hilgers et al. (1985) have shown that the substrains differ as a result of mutations at the Raf-1 locus (controlling the expression of alpha-fetoprotein), the Qa-2 locus (governing cell surface antigens), the Gdc-1 locus (governing L-glycerol 3-phosphate dehydrogenas activity in the liver) and the PR1 repetative sequence. There is no evidence for genetic contamination during the early history of the strain. A fourth substrain, BALB/cWt is also listed as it has a high incidence of hermaphroditism.

BALB/cHeAn
Inbr ?.To Snell circa 1932, to He circa 1935. Now widely distributed (including the By, AnN, HeA and AnPt substrains). This substrain is much less aggressive than the J substrain. Maint. by A, N.

431
BALB/cJ
Inbr 150 +?. Snell to Jackson Laboratory after 1940. Males of this substrain are extremely aggressive and will fight if housed together once mature. The Lac substrain separated in 1952 is non-aggressive. Maintained by J, Ola (JLac substrain).

BALB/cRl
Inbr ?. Snell to Green circa 1937, to W.L. and L.B.Russell c1948.

BALB/cWt
Inbr ?. Has about a 3% incidence of true hermaphroditism, which significantly distorts the sex ratio (Eicher et al. 1980)

Behaviour

Life-span and spontaneous disease

Left auricular thrombosis occurs in 66% of older breeding females. This is associated with reduced levels of the prothrombin complex factors such as factor IX (40% of normal), factor XIII (60% of normal), factor X (50% of normal) and prothrombin (about 33% of normal). These deficiencies occur slightly before parturition (Meier and Hoag, 1966). High incidence of epicardial mineralisation (11% in males, 4% in females), which increases slightly with age (Frith et al., 1975). Heart defects, including cardiac calcinosis 17-62% (Festing and Blackmore, 1968).
1971). Spontaneous myocardial lesions of right ventricle found in 60% of females and 30% of males. These macroscopically visible degenerative fibrosclerotic lesions may represent a last phase of myocarditis of the inflammatory type found in apparently normal mice (Bellini et al., 1976).

Zero incidence of spontaneous congenital malformations (cf. 2/9) in GrKt-tk substrain (Kalter, 1968).

**Normal physiology and biochemistry**


High peripheral nerve conduction velocity (1/6) (Regmann, 1972). High brain L-glutamic acid decarboxylase (GAD) and choline acetyltransferase and catechol-U-methyltransferase (1/7 in all cases); low brain acetylcholinesterase (5/7) and monoamine oxidase activity (7/7) (Tunnicliff et al., 1973). High brain tyrosine hydroxylase activity (1/5) (Ciranello et al., 1972). High brain plasmalogen (1/5) (Sampugna et al., 1975). High proportion of time spent sleeping (2/6) and low proportion of paradoxical sleep (6/6) (Valatx and Bugat, 1974).


**Anatomy**


**Drugs**

Susceptible to skin ulceration by DMBA (cf. 13/23) (Thomas et al., 1973). Susceptible to tumour induction by 3-methylcholanthrene (3/12) (Whitmire et al., 1971). Susceptible to induction of leukaemia (1/6) but resistant (6/6) to induction of liver tumours by neonatally administered DMBA (Flaks, 1968). High incidence of interstitial tumours of testis induced by stilboestrol, high incidence of haemangioendotheliomas, particularly in interscapular fat pad and lung in mice treated with O-aminoazotoluene (Heston, 1963). High incidence of lung tumours
after administration of methycholanthrene by gavage (1/5) (Akamatsu and Barton, 1974). Injection of mineral oil i.p. induces a high incidence of transplantable plasmacytomas (myelomas). Bence Jones proteins include kappa and lambda light chains and the two-chain IgA protein. 60% of tumours are of the IgA type (Potter, 1972).

Sensitive to X-irradiation (26/27) (Roderick, 1963), (10/10) (Storer, 1966); low LD50 to X-irradiation (9/9) (Yuhas and Storer, 1969).


Immunology


Resistant to induction of experimental autoimmune thyroiditis (cf. 2/5) (Vladutiu and Rose, 1971a). Resistant to induction of anaphylactic shock by ovalbumin (cf. 6/13) (Tanioka and Esaki, 1971).

Infection

Highly susceptible to infection by Salmonella typhimurium strain C5 (1/7) (Plant and Glynn, 1974), (2/5) (Robson and Vas, 1972). Relatively resistant to a natural intestinal helminth infection (1/10), (Eaton, 1972). High susceptibility to BALB/Tennant leukaemia virus (1/12) (Tennant, 1965). Transmission of murine leukaemia virus (Scripps) through three successive generations 100% (cf. 2/5) (Jenson et al., 1976). Highly susceptible to development of leukaemia on infection with Friend virus (cf. 5/11) (Dietz and Rich, 1972). Susceptible to Mycobacterium
*marinum* (3/9) and good plateau harvest of *M. leprae* 8 months after infection (2/9) (Shepard and Habas, 1967). Susceptible to mouse hepatitis virus type 3 (cf. 5/14) (Le Prevost *et al.*, 1975). Resistant to induction of diabetes mellitus by encephalomyocarditis virus (cf. 7/14) (Boucher *et al.*, 1975). Highly susceptible to the *Leishmania tropica* parasite, with the local disease being uncontrolled and with the development of metastases and fatal visceralization (Howard *et al.* 1980).

**Reproduction**


**Miscellaneous**

Recommended host for transplantable tumours: melanoma HP and pleomorphic sarcoma 5180 (although the latter is not host-specific) (Kaliss, 1972). Low mortality after neonatal thymectomy (2/6) (Law, 1966a).

- **BBT**

- **BDP**

**Behaviour**

Low food drive (14/15) and high emotionality (2/15) (Thompson, 1953). Nervous (Staats, 1976).

**Life-span and spontaneous disease**

Short life-span in conventional conditions (4/22 = 421 days in males, 6/22 = 468 days in females) (Storer, 1966). Frequent mammary tumours, polycystic or granular kidneys (Staats, 1976).

**Normal physiology and biochemistry**


**Anatomy**


**Drugs**


**Immunology**

Erythrocytes have a high agglutinability (cf. 14/25) (Rubinstein *et al.*, 1974).

- **BL**
  Inbr: F108. Albino. Genet: a, b, c strain developed by Lynch from Bagg albino stock via Strong, and maintained at Rockefeller Institute since 1921.
Charac: Low mammary tumours, some lung tumours in older mice (Staats, 1976). Ovaries often appear pale and enlarged and have an infiltration of amyloid. Thought to be due to necrotising arteritis in which there is a thickening of the walls of the vessels to duodenum and ovaries due to a deposition of eosinophilic material within the intima and media (Deringer, 1959b). Aseptic necrosis of bone in 22% of females often resulting in fracture of femoral neck and other complications, so that the animals are severely crippled (Sokoloff and Haberman, 1958).

- **BFM/1**
  Inbr 30 (Mpl). Light agouti colour with a light belly $A^*, B_1 C$, where * indicates a variant allele. Origin: Wild mice trapped at Univ. of Montpellier. It has never been crossed with laboratory mice.

- **BFM/2**
  Inbr 15+24 (Ms). Light agouti colour with a light belly $A^*, B_1 C$, where * indicates a variant allele. Origin: Wild mice trapped at Univ. of Montpellier. It has never been crossed with laboratory mice. BFM/2Ms was branched from the original BFM/2Mpl at F12-15 (Bonhomme et al 1982). Inbred a further 24 generations by Ms. Maint. by A.

- **BIMA**
  Inbr, G10F58. Colour ?. Origin: Muhlbock 1959. A partly congenic strain of C57BL/LiA following an outcross to C3H/HeA with cross-intercross matings with selection for large mammary glands to G10 then further selection with bxs mating to F11. Maint. by A.

- **BIR**
  Inbr, G12F61. Colour ?. Origin: Muhlbock 1958. A partly congenic strain of C57BL/LiA following an outcross to DBA/LiA with cross-intercross matings with selection for resistance to the growth of C57BL/LiA mammary tumour cell line transplanted in vivo. Maint. by A.

- **BLN**

- **BLRB**
  Inbr F20. Black. Origin: EV Moiseeva, 1981. Spontaneous translocation Rb(8.17)Iem was found originally by VS Baranov in C3HA/Iem mice and the carriers were crossed to black mice of an unknown C57BL/Iem strain, followed by b x s mating. $Hb^b$. Mammary tumours in 95% of breeding females at an average age of 11.2 months. Maintained by Moiseeva, Institute of Immunology, Kashirskoe sh., 24-2 Moscow 115478, USSR.

- **BN/a**
  Inbr: 55. Genet: $A, B, c, D, H-2^b$. Origin: Anna Dux, Gliwice, September 1950 from unknown parents. F13 taken to Warsaw June 1956, where it was split into two substrains, inbreeding continued. Charac: 35% lung tumours at 662 days; 7.1% leukaemia at 647 days; 23% chronic nephritis at 562 days. Used for carrying transplantable vaginal epithelioma G93 (Czarnomska and Wezykowa, 1971).

- **BN/b**
  Inbr: 60. Genet and Origin: see BN/a. Charac: Lung tumours 25% at 629 days; 1.8% leukaemia at 512 days in females, 13% chronic nephritis at 493 days; used for carrying transplantable vaginal epithelioma G94.

- **BNT**
Inbr (Le) 73. Black a. Origin: mutation to Bn (bent-tail) discovered by E.D. Garber. From A.B. Griffen to Lane 1960. Inbred to F27, then one outcross to C57BL/6J x CBA/Ca F1, followed by b x s. (now only maintained as frozen embryos). Maint. by Le

■ BPH

■ BPL

■ BRSUNT
Inbr: 130. Genet: a, b, C. Origin: Strong, a branch of BRS (from strain NH) continued without further methylcholanthrene treatment. Charac: Gastric lesions, adiposity; 100% incidence of periodontal disease.

■ BRVR
Inbr: F83+. Albino. Genet: c. Origin: Webster (1933a,b) developed two lines resistant and susceptible to infection by Bacillus enteritidis (Salmonella sp.) by selection on the basis of a progeny test, followed by full-sib mating. Mortality under specified test conditions was 15% and 85%, respectively, and was about 39% in the unselected controls. Later tests (Webster, 1937) with loping ill and St Louis encephalitis viruses led to the development of three lines designated BSVS (bacteria-susceptible, virus-susceptible), BSVR (bacteria-susceptible, virus-resistant) and BRVS (bacteria-resistant, virus-susceptible). Later BRVR (bacteria-resistant, virus-resistant) mice were developed from a cross between BSVR and BRVS, with subsequent selection for resistance to the two agents, and full-sib mating. There may have been some confusion between the lines in the past, and authenticity should not always be assumed.

Charac:
Hydronephrosis in 11% of females and 18% of males (1/13) (Taylor and Fraser, 1973), 34-42% in males and 30-34% in females (Collins et al., 1971). Resistant to Salmonella, some encephalitic viruses and experimental allergic encephalomyelitis (Staats, 1976). Resistant to experimental allergic encephalomyelitis (cf. 6/14) (Levine and Sowinski, 1973). Resistant to induction of experimental autoimmune thyroiditis (cf. 2/5) (Vladutiu and Rose, 1971a). Carries gene for resistance to group B arbovirus infection (cf. strain PRI) (Darnell et al., 1974). Differences between the responses of BRVR and BSVS to infections and various antigens are listed by Boehme(1970).

■ BRX58N-

■ BSC

437

- **BSVR**
  Inbr ?. Albino. See BRVR. Maint. by Db.

- **BSVS**
  Charac:

- **BT/Kt**
  Inbr: F???. Resistant to teratogenic effects (cleft palate) of cortisone acetate (1/6) (Kalter 1981). Not related to BT/Os. Possibly extinct.

- **BT/Os**
  Charac:
  High incidence of spontaneous mammary tumours (70%), high incidence of gastric tumours following methylcholanthrene by gavage (1/5), and highly sensitive to skin carcinogenesis (Akamatsu and Barton, 1974). (NOTE: Strain BT/Kt is unrelated to this strain.)

- **BUA**

- **BUB**

- **BXD-**
  Inbr 30+. Set of 24 recombinant inbred strains developed by B.A.Taylor from C57BL/6J x DBA/2J. Maint. by Ty.

- **BXH-**
  Inbr 30+. Set of 13 recombinant inbred strains developed by B.A.Taylor from C57BL/6J x C3H/HeJ. Maint. by Ty.

- **BXSB**
Inbr (Mp) 49. Agouti; +. Origin: E.D. Murphy from a cross of C57BL/6J x SB, followed by selection of the satin, non-beige phenotype, followed by b x s mating. Develops spontaneous lupus-like autoimmune syndrome which is strikingly accelerated in the males. This also occurs in F1 hybrids provided BXSB is the male parent, and appears to be due to a Y-linked gene (Murphy and Roths 1979). Cell-mediated immune function compares favourably with other strains, though there may be reduced reticuloendothelial function (Creighton et al. 1979). Theofilopoulos et al (1980) have compared immune function in this and other autoimmune strains. Maint. by J, Ola.

- BXVII
Inbr ?. No details. Maint. by Cri.

- BOB

- C

- CAT
Inbr. ?+26 (Lac). Albino c,Catfr. Origin: Catfr arose in strain A. Apparently outcrossed to unknown stock, then inbred by Muggleton-Harris. (Muggleton-Harris et al 1987). All mice have bilateral cataracts. The lens abnormality begins to form before 14 days of interuterine life. Selective loss of a family of gene transcripts for specific crystallin synthesis has been reported (Garber et al 1985). Maint. by Muggleton-Harris.

- CBA
Inbr: F90-170 depending on substrain. Agouti. Genet. +. Developed by Strong in 1920 from a cross of a Bagg albino female and a DBA male. Strain CBA was selected for a low mammary tumour incidence and C3H for a high incidence. Now widely distributed, and used as a general-purpose strain. Differences between substrains are probably too large to be accounted for by mutation, and some degree of genetic contamination in the past is probable. The following major substrains are recognised:

CBA/Ca or CBA/H
Strong, to Jackson Laboratory, to Haldane and Gruneberg in 1932. To Carter 1947 and Harwell 1954. This substrain used in most British research.

CBA/Br
Jackson Laboratory, to Haldane 1932, to Bonser (Leeds) approx. 1933.

CBA/CaN
Harwell, to National Institutes of Health in 1966. Carries sex-linked immunological deficit which prevents it from responding to type III pneumococcal polysaccharide. Deficit is expressed on B cells (Gershon and Kondo, 1976; Scher et al., 1976). Do not carry naturally occurring tumour-reactive antibodies commonly found in other strains (Martin and Martin, 1975).

CBA/J
Strong, to Andervont 1947, to Jackson Laboratoy 1948. Carries gene for retinal degeneration \( (rd) \). Skin grafts between CBA/J and CBA/Ca are rejected (Green and Kaufer, 1965).

CBA/St
Original strain maintained by Strong.

CBA/H-T6
T6 translocation backcrossed to CBA/H by Dr M. F. Lyon. Now homozygous for the marker translocation T(14;15) 6Ca, but otherwise congenic with CBA/H.

**Behaviour**

**Life-span and spontaneous disease**
Life-span intermediate both sexes (J substrain) in conventional conditions (11/22 = 527 days males, 10/22 = 527 days females) (Storer, 1966). Life-span (Ca substrain) short in males (4/17 = 486 days) and long in females (17/17 = 825 days) in SPF fostered conditions. Short life-span of males associated with a high incidence of haemothorax, suggesting a high sensitivity to vitamin K deficiency in SPF conditions (Festing and Blackmore, 1971).

High gross tumour incidence (J) (3/22) (Storer, 1966). Overall tumour incidence 29% in males, 55% in females, including lymphoma 6% in males, 15% in females, hepatoma 24% in males, zero in females and mammary tumours 33% in females and zero in males (Smith et al., 1973). Lung adenomas 2-11% in BrA substrain, leukaemia 4-10% (Muhlbock and Tengbergen, 1971). Resistant to the induction of atherosclerosis by a high-fat and high-cholesterol diet (1/13) (Roberts and Thompson, 1976).

**Normal physiology and biochemistry**

High proportion of the time spent sleeping (2/6), with large percentage of slow-wave sleep (1/6) and low percentage of paradoxical sleep (5/6) (Valatx and Bugat, 1974). Low percentage of paradoxical sleep (7/7) (Pagel et al., 1973). Low metabolic rate (14/18) according to Storer (1967), but high metabolic rate (2/6) according to Pennycuik (1967). High cell turnover in J substrain as estimated by rapid clearance of DNA-bound radioactivity (2/17) (Heiniger et al., 1972).


Anatomy

Drugs

Immunology

Infection
Resistant to infection by Salmonella typhimurium strain C5 (7/7) (Plant and Glynn, 1974). Susceptible to infection by liver fluke Opisthorchis felineus (2/6) (Zelentsov, 1974). Good plateau harvest of Mycobacterium leprae 8 months after infection (1/9) (Shepard and Habas, 1967). Resistant to induction of diabetes mellitus by encephalomyocarditis virus (cf. 7/14) (Boucher et al., 1975). Highly susceptible to measles virus (cf. 3/6) (Rager-Zisman et al., 1976). Ca, H-T6 and N substrains carry no detectable endogenous ecotropic MuLV DNA sequences (Jenkins et al 1982)
Good breeding performance (9/25), colony output 1.15 young/female/week, litter size at weaning 5.8(11/25), T6 substrain about equal (Festing 1976a, original data). Intermediate breeding performance (4/8), litter size 5.4, sterility 5.2% (Nagasawa et al., 1973). Good litter size (1/6 to 3/6, depending on parity), but low proportion of females produce four or more litters (2/6) (Fernandes et al., 1973). Poor breeding performance in N substrain (21/24) (Hansen et al., 1973). Low percentage pre-implantation loss of embryos (2/8) (Leonard et al., 1971).

Females have a high rate of fetal resorption when mated with DBA/2 males, but this can be dramatically reduced by immunization with BALB/c, but not DBA/2J spleen cells. This may provide an animal model for the prevention of fetal death by vaccination (Chaouat et al 1985)

Miscellaneous

CBRB
Inbr N?+F25. Agouti. Origin: EV Moiseeva. Spontaneous translocation Rb(8.17)Iem was found originally by VS Baranov in C3HA/Iem mice and carriers were crossed to CBA/Calac. After an unknown number of backcrosses, mice were maintained by b x s matings and were sent to the Institute of Immunology, Kashirskoe Sh., 24-2, Moscow 115478, USSR, in 1984 at F11. Carries Rb(8.17)Iem translocation. More than 50% mammary tumours in breeding females by 9.3 months of age. Hbbd Maintained by Moiseeva at above address.

CBXC-
Inbr circa F25. Set of 9 recombinant inbred strains developed by Ola from a cross between CBA/Ca and BALB/c (Fernandez et al 1989). Maint. by Ola.

CC57BR
Inbr: F83. Chocolate. Genet: a, b. Origin: Medvedev, 1943 from BALB/cN female x C57BL/N male. Charac: No spontaneous mammary tumours, but about 60% when the milk agent is introduced. Primary lung tumours 22%. Incidence of all other tumours less than 1% (Heston, 1963). Life-span about 18 months (Medvedev, 1969).

CC57W

CE
Inbr: F? + 68. Black-eyed grey. Genet: A\textsuperscript{+}, c\textsuperscript{-}. Originating in 1920 from wild mice trapped by J. E. Knight. The coat colour genetics later studied by Detieffen. However, as the strain closely resembles 'laboratory mice' (Taylor, 1972) and is not wild in behaviour, it seems possible that the original mutant mice were crossed with unidentified laboratory mice before being inbred. The strain is not widely used, and has a poor reproductive performance. However, its unique coat colour ensures authenticity, and it has an interesting range of tumour types, including a high incidence of ovarian tumours. F\textsubscript{1} hybrids with DBA/1, DBA/2 and C3H have a high incidence of hepatomas (Hancock and Dickie, 1969).

Characteristics
Sporadic high incidence of ear chewing of young by mother in Lac substrain (Festing 1976, original observation). Low preference for sweet tasting substances (saccharin, sucrose, dulcin and acesulfame, averaged) (22/26) (Lush 1988).


Resistant to induction of diabetes mellitus by encephalomyocarditis virus (cf. 7/14) (Boucher et al., 1975). Carries no detectable endogenous ecotropic MuLV DNA sequences (Jenkins et al 1982)

Short sleeping time under pentobarbitone anaesthetic (1/23), Lovell (1986).

Poor reproductive performance (23/25), colony output 0.53 young/female/wk, although litter size is large (6/22) at 6.1 (Festing, 1976a). High ratio of males at birth (1/11) (Cook and Vlcek, 1961).

Recommended host for transplantable rhabdomyosarcoma BW10139 (Kaliss, 1972).
Origin: \( ch \) (congenital hydrocephalus) on B6CBA background from Gruneberg to M.C. Green 1961. \( ch/+ \) crossed to \( cr,+/-,cr \) to F9. \( ch/+ \) crossed to \( sa/+ \), maintained \( ch,+/-,sa \) to F20. \( ch/+ \) crossed to \( mu/mu \), maintained by \( ch,+/-,mu \) bxs to present. \( mu \) from M. Lyon to Jax 1976. Crossed to C57BL/6J and bxs to F3. \( ch \) homozygotes die at birth and are recognised by bulging haemorrhagic cerebral hemispheres. Maintained by Le.

- **CKB**
  Inbr. 50. Agouti. +. Origin: C3H.SW \((Ig-I^{b,H-2b}) \times C3H \((Ig-I^a,H-2^k)\), FI inbred to give CKB.

- **CL**
  Inbr: F20 +. Strain developed by Fraser from a heterogeneous stock carrying the \( msl \) gene (migratory spot lesion, a variable white spot on the retina), crossed to A/J and subsequently inbred with selection for high frequency of spontaneous cleft lip. Frequency of the latter now 26% in viable 17-day embryos (Bornstein et al., 1970).

- **CLA**
  Inbr 22. Agouti. Origin: Wild mice trapped on a farm near Centreville, Maryland. Strains WSA and WSB were separated from the same stock at F3.

- **CN**

- **CPB-H**
  Inbr: 42. Genet: \( b, d \). Origin: At Centraal Proefdierenbedrijf TNO, Holland (Cpb), about 1950. Charac: Females often have a pair of non-lactating supernumerary nipples (Staats, 1976).

- **CPB-K**
  Inbr: 54. Genet: \( a, b, c, d \). Origin: As CPB-H. Former designation KC discontinued. Charac: Excitable; small litters; amylase type B (other CPB strains are type A).

- **CPB-MO**

- **CPB-N**
  Inbr: 47. Genet: \( a, b, p \). Origin: As CPB-H. Charac: Normal sex difference in major urinary protein content of serum and in reaction to hexobarbital.

- **CPB-P**
  Inbr: 39. Genet: \( a, b, d, p \). Charac: Reacts to LSD with head twitching. Very light red-eyed grey.

- **CPB-Q**
  Inbr (Cpb): \( ? + 61 \). Genet: \( a, b, c, D \). P. Origin: Hagedoorn, Holland, to Hirschfeld 1937, to Cpb 1949. Charac: More susceptible to tuberculosis than most strains; reacts to LSD with head twitching.

- **CPB-R**

- **CPB-S**
Inbr: ? + 59. Genet: A, B, c, D, P. Origin: Rockefeller Institute, probably same origin as BSVS, BRVR, etc., to Laidlaw (Hampstead), to Hagedoorn, to Cpb 1949. Charac: Rather aggressive, especially males (Cpb), territorial, with a sensitive aggression-flight balance; adapted to surface-living rather than hole-living.

■ CPB-TK

■ CPB-V

■ CPB-WG

■ CPB-WV

■ CS
Inbr: 61. Genet: a, b, c, D, s. Origin: Established in 1956 from hybrids between NBC and SII, both now extinct. Unrelated to IVCS. Charac: Good reproductive performance; quick moving; Japanese crooked tail 16%.

■ CT
Inbr (Lac) ?+8 . Agouti: +. Origin: ct (curly-tail) mutation arose spontaneously in GFF inbred mice. Backcrossed to CBA an unknown number of times, then bxs of presumed ct/ct homozygotes. Differs from CBA at many marker loci. Expression of the ct gene ranges from spina-bifida causing pre-natal death to various degrees of kinky tail, to complete normality. The neural tube defects can be modified by environmental factors. Excess vitamin A increases the incidence, but low doses given at a particular stage of gestation can prevent the abnormalities (Seller and Adinolfi 1981). The exencephaly and spina bifida are manifested independently of the maternal environment (Seller et al 1981) Maint. by Lac.

■ CTA

■ CWD

■ CXB-
Inbr (By) 91. Set of seven Recombinant Inbred Strains (CXBD,E,G,H,I,J,K) developed by Bailey from a cross of BALB/c x C57BL/6J. Maint. by By.

■ CXS-
Set of 14 recombinant inbred strains developed by A from a cross of BALB/cA x STS/A (Hilgers and Arends (1985). Maint. by A.

■ C17/Icrc
Inbr: F25 +. Light brown strain developed in 1956 from a cross of male C57BL/6 and female XVII. Life-span 26 months. Litter size 7.2-7.5 at birth and 4.4-6.2 at
weaning. Low incidence of tumours of all types. Sensitive to weak skin carcinogens. (Ranadive et al., 1969).

### C3H

Inbr: F130 to F170 depending on substrain. Agouti. Genet: +, rd. Developed by Strong 1920 from a cross of Bagg albino with DBA male (see CBA) with selection for a high incidence of mammary tumours. Now among the most widely used of all mouse strains. Most substrains have a good reproductive performance. Unfostered substrains (which are now relatively rare since 'SPF' animals have become popular) have a high incidence of mammary tumours (usually > 90% at one year) caused by a virus which is passed from mother to offspring through the milk. Fostering of the young or transfer of fertilised ova to a mammary tumour virus-free strain eliminates the virus, and substantially reduces the incidence of mammary tumours. Note that all 'SPF' stock will be free of this virus.

The unfostered substrains are widely used in cancer research for the sake of their mammary tumours. Fostered stock are widely used as a general-purpose strain which is readily available and well known. The strain should be used with care in behavioural studies, since it carries the rd (retinal degeneration) gene and is blind after about 6 weeks.

Some substrain differences are large, and can not be accounted for solely on the basis of mutation, and must be ascribed either to substantial residual heterozygosity or genetic contamination (McLaren and Tait, 1969), though C3H/HeJ is known to differ from C3H/He as a result of a mutation at the lps (lipopolysaccharide) locus.

The following major substrains are recognised:

**C3H/Bi**
Strong to Bittner 1931, to Kirschbaum 1952. Has 83% mammary tumours in unfostered breeders. Low leukaemia.

**C3H/Fg**
Origin not known, but has a very high incidence of lymphatic leukaemia (over 90%) (Fuchs, 1962).

**C3H/He**
This strain was passed to Heston in 1941, and is now the most widely distributed of all. Non-fostered substrains have more than 90% mammary tumours by about 11 months. Fostered substrains have a high incidence of hepatomas (Festing and Blackmore, 1971).

**C3H/HeJ**
Heston, to Jackson Laboratory in 1947, and now widely distributed. Has poor immune response to endotoxic lipopolysaccharide due to a B-cell deficit (Rosenstreich and Glode, 1975; Coutinho, 1976).

**C3HeB/De**
A substrain developed by transfer of fertilised ova to strain C57BL by Deringer. This strain lacks the mammary tumour virus and therefore has a lower incidence of mammary tumours (4% in virgin females and 55% in breeding females and 74% in force-bred females) (Deringer, 1959a).

**C3HeB/Fe** (syn: TC3H)
Developed by Fekete in 1948 by transfer of fertilized ova of C3H/HeJ to C57BL/6. Lacks mammary tumour virus.
C3H/He-mg
'Mahogany' coat colour mutation occurred spontaneously in C3H/He stock held at Laboratory Animals Centre, Carshalton, in 1967. The strain has been propagated because authenticity can be guaranteed by the colour of the coat.

C3H/He-A^v
Congenic line developed by backcrossing the A^v to the C3H background. Has an exceptionally high mammary tumour incidence, virtually 100% at 7-8 months. The fostered substrain C3H-A^vF.B has a 90% incidence of mammary tumours transmitted by either parent (Vlahakis et al., 1970).

C3H.RV
Congenic line resistant to arbovirus infection, developed by Groschel and Koprowski (1965) by backcrossing the resistance gene from PRI to C3H.

Behaviour


Carries the retinal degeneration gene and is capable of pattern discrimination up to 40 days, and brightness discrimination to at least 100 days (Nagy and Misanin, 1970).

Life-span and spontaneous disease
Almost 100% of mammary tumours in females of unfostered substrains (Heston, 1963). Mammary adenocarcinomas in unfostered substrains less than 1% in males, 95% in breeding and 88% in virgin females. Lymphatic leukaemia zero incidence (Hoag, 1963). Mammary tumours 100% at 6.8 months in C3H-A^v, 90% in C3H-A^vTC57BL at 15.3 months. Mammary tumours 40% at 18.8 months in C3HFC57BL, but 99% at 7.2 months in unfostered C3H (Heston and Vlahakis, 1971). Mammary tumours 37% at 2 years in fostered substrain (Bentvelzen et al., 1970). Median latent period to develop mammary tumours in unfostered substrains ranged from 276 to 566 days, depending on breeding status and environmental stress (Riley, 1975). A high proportion of the mammary tumours are of the acinar type (2/7) (Tengbergen, 1970). Hepatomas 72-91% in males at 14 months, 59% in virgin females, 30-38% in breeding females (Heston, 1963). Hepatomas have eosinophilic cytoplasmic inclusion bodies (Liebelt et al., 1971). Lung adenomas 2-10% in fostered A substrain, leukaemia 6-30% (Muhlbock and Tengbergen, 1971). Occasional Harderian gland tumours (Heston, 1963).

Life-span in SPF fostered conditions intermediate in both sexes (11/17 = 590 days in males, 12/17 = 676 days in females). Liver tumours 9-23%, lung tumours 2-10% and mammary tumours 21-36%. Heart defects 13-25% and cystic ovaries 13-26% (Festing and Blackmore, 1971). Tail lesions similar in appearance to bit wounds were found in grouped C3H/HeJ by Les (1972).

Can be made obese by a suitable diet (Fenton and Dowling, 1953).
C3HeB/FeJ

Primary lung tumours 8% in males, 4% in breeding females and 10% in Virgin females. Lymphatic leukaemia zero. Mammary adenocarcinomas zero in males, 12% in breeding females, 2% in virgin females (Hoag, 1963). Ovarian tumours 47% in Virgin and 37% in breeding females, 29% in force-bred females (Heston, 1963). Hepatomas 91% in breeding males, 58% in Virgin and 30% in breeding females (Murphy, 1966). Life-span above average in both sexes (16/22 = 652 days in males, 17/22 = 657 days in females). High gross tumour incidence in males (5/22) (Storer, 1966).

Normal physiology and biochemistry


C3HeB/FeJ


Anatomy


C3HeB/FeJ


Drugs

Susceptible to skin ulceration to DMBA (cf. 13/22) (Thomas et al., 1973). Susceptible to induction of subcutaneous tumours by 3-methylcholanthrene (1/14 to 4/14, depending on substrain) (Kouri et al., 1973). Susceptible to tumour induction by 3-methylcholanthrene in fostered and unfostered substrains (1/8 to


Susceptible to skin ulceration by DMBA (cf. 13/22) (Thomas et al., 1973). Sensitive to X-irradiation (23/27) (Roderick, 1963). Good ovulatory response (94%) to 3 I.U. PMS (1/6), but poor response (33%) to 7 I.U. PMS. Response facilitated by exposure to males (Zarrow et al., 1971).

Immunology

Good immune response to Pro-Gly-Pro-ovalbumin (1/7) and (Pro-Gly-Pro)$_n$ (2/7) (Fuchs et al., 1974). High susceptibility (3/12) to IgE-mediated passive cutaneous anaphylaxis (De Souza et al., 1974). Good immune response to Salmonella strasbourg lipopolysaccharide (1/7) (Di Pauli, 1972). Low PHA- stimulated lymphocyte blastogenic response in Ent substrain (6/6) (Hellman and Fowler, 1972). Erythrocytes of C3HeB/FeJ have a high agglutinability (cf. 14/25).

**Infection**

HeJ substrain (which carries the *lps* mutation) resistant to LCM virus (76% survival) prior to 1970, but has now become susceptible (3% survival) (Oldstone and Dixon, 1973). Resistant to LCM virus infection (1/5) (Oldstone and Dixon, 1968). Resistant to induction of diabetes mellitus by encephalomyocarditis virus (cf. 7/14) (Boucher et al., 1975). Susceptible to lethal infection with *Rickettsia akari* strain Kaplan, in contrast with seven other substrains of C3H and 24 other strains (Anderson and Osterman 1980a,b).

C3HeB/FeJ
Highly susceptible to mammary tumour virus, but believed to be free of the virus (Murray and Little, 1967). Low susceptibility to BALB/Tennant leukaemia virus (11/12) (Tennant, 1965). Resistant to induction of diabetes mellitus by encephalomyocarditis virus (cf. 7/14) (Boucher et al., 1975).

**Reproduction**
Breeding performance intermediate/good (5/25 He substrain, 10/25 He-mg sub-line). Colony output 1.1 to 1.4 young/female/week. Litter size at weaning 5.9 (8/25) (Festing, 1976a). Good reproductive performance (2/8), litter size 6.4, sterility 10% (Nagasawa et al., 1973). Large litter size (1/6 to 3/6), high proportion of females produce four or more litters (1/6) and high proportion of fertile matings (1/6) (Fernandes et al., 1973). Good breeding performance, 2.0 to 2.2 young per female per month (9/24 to 7/24) in fostered and unfostered substrains, respectively (Hansen et al., 1973).

C3HeB/FeJ
High reproductive performance (1/8). Litter size 6.4 ± 0.2, sterility 4% (Nagasawa et al., 1973).

**Miscellaneous**

High rate of spontaneous mutations (1/21) and total deviants (4/21) (Schlager and Dickie, 1967).

C3HeB/FeJ

C3HA
Inbr (Y) 102. Agouti: + Origin; Pogosianz, C3H female x A male, followed by sib mating. Originally 30% mammary tumours, but has declined. Susceptible to hepatic carcinogens. Maint. by Y.

C57BL Black, a. Origin: Little 1921 from the mating of female 57 with male 52 from Miss Abbie Lathrop’s stock. The same cross gave rise to strains C57L and C57BR. Female 58 mated with the same male gave rise to strain C58. C57BL is probably the most widely used of all inbred strains, (substrain C57BL/6 alone accounts for over 14% of occasions on which an inbred strain is used) though in many ways it seems to be atypical of inbred strains of laboratory mice. However, it has a good breeding performance, and has been used as the genetic background for a large number of congenic strains covering both polymorphic and mutant loci. Four major substrains A, GrFa, 6 and 10 appear to be quite similar, and any differences are consistent with what might be expected from the accumulation of new mutations and a small amount of residual heterozygosity. However, the Ks substrain differs at several loci and may be the result of genetic contamination. The seven major substrains existing in 1935 are listed below.

C57BL/A
Inbr(A) ?+142. Origin. Little to A c1932. Maint. by A.

C57BL/An
Little to Andervont 1932. Differs from B6 and B10 at the Ce-1 locus.

C57BL/GrFa.
Origin: Little to Gruneberg 1932, to Falconer 1947. Most British substrains derived from this stock, though 6 and 10 substrains have been imported more recently. This substrain seems to resemble the 6 rather than the 10 substrain. Maint. by Ola

C57BL/KaLwN.
To N 1965 from Lw at F35. Maint. by N.

C57BL/Ks.
Origin: C57BL/6J to Biesele in 1947, then pen bred, to Kaliss in 1948. Ks resumed inbreeding. To J 1948. Differs from 6 and 10 substrains at the Bgl-s, Bgl-t, cdm and H-2 loci. Resembles B6 at the Lv locus (at which B6 and B10 differ). Maint. by J.

C57BL/6
Inbr (J) 150. Origin: substrains 6 and 10 were separated prior to 1937. This substrain is now probably the most widely used of all inbred strains. Substrain 6 and 10 differ at the H-9, Igh-2 and Lv loci. Maint. by J,N, Ola.

C57BL/10
Inbr (J) 158. Origin: see C57BL/6. Maint. by J.

C57BL/10ScSn.

C57BL/10Cr
Carries spontaneous lipopolysaccharide mutation /ps which appears to resemble that found in C3H/HeJ (Vogel et al 1981)
**Behaviour**

Substrain unspecified:
High incidence of tail rattling (1/5) (St. John, 1973). Short latency to attack and eat crickets (2/7) (Butler, 1973). High alcohol preference ratio (1/5) (McClearn, 1965). Short latency to emerge from home cage (1/7), short latency to cross barrier in open-field (1/7), low number of stairs climbed (7/7), low urination (6/7) and defaecation (7/7) (McClearn et al., 1970).

C57BL/6


C57BL/10


**Life-span and spontaneous disease**

Substrain unspecified:
Mammary tumours less than 1% (Heston and Vlahakis, 1971). Lung adenomas 0-9% in LiA substrain (Mühlbock and Tengbergen, 1971). Zero incidence of mammary tumours at 2 years (cf. 3/7) (Bentvelzen et al., 1970).

Mean life-span 800 days in males and 750 days in females according to Rowlatt et al. (1976), who also give details of pathology in a large aging colony of C57BL/1crf-a mice. Hyperphalangy and polydactyly occur with a low incidence in all C57BL strains and substrains (Dagg, 1966). Hydrocephalus 4.1% (Mori, 1968). Type B reticulum cell neoplasms 75% at about 20 weeks in HeDe substrain (Dunn and Deringer, 1968).

C57BL/Ka

Median life-span 23 months in males. Main autopsy findings include reticulum cell sarcoma type B (29%), testes interstitial tumour (13%), thyroid follicular adenoma (9%), unclassified lymphoreticular tumours (9%). Nine other tumour types found. Non-neoplastic lesions include amyloid (83%), Sendai virus pneumonia (20%), periarteritis nodosa (16%), mesenteric disease (10%). Several other lesions noted. (Zurcher et al., 1975). About 50% of mice develop homogeneous immunoglobulins resembling idiopathic paraproteinaemia in man by 24 months (Radl and Hollander, 1974).

C57BL/Fa

Long life-span in males (14/17 = 645 days), but intermediate in females (5/17 =
580 days) in SPF fostered conditions (Festing and Blackmore, 1971). Hydronephrosis 0.5% in females, 1.5% in males (Taylor and Fraser, 1973).

C57BL/6
Primary lung tumours 1% in males, 3% in breeding females and zero in virgin females. Lymphatic leukaemia less than 2%, mammary adenocarcinomas less than 1% (Hoag, 1963). Leukaemia 7% (Myers et al., 1974). Susceptible to the development of atheromatous lesions on wall of aorta after 20 weeks on a high-fat diet (Thompson, 1968; Roberts and Thompson, 1976). Congenital abnormalities 10%, including eye defects, polydactyly and otocephaly (Kalter, 1968). Microphthalmia and anophthalmia 8-20% and hydrocephalus 1-3% (Dagg, 1966).

Life-span above average in both sexes in conventional conditions (17/22 = 676 days in males, 18/22 = 692 days in females) (Storer, 1966). Life-span 827 ± 34 days in males, 818 ± 21 days in females (Goodrick, 1975). Life-span 878 ± 10 days in males and 794 ± 6 days in females (Kunstyr and Leuenberger, 1975). Median life-span 600 days (Curtis, 1971). Gross tumour incidence 70%, maximum life-span about 1200 days in SPF conditions (Mewissen, 1971).

Dermatitis with intense pruritis leading to self-mutilation and death, and sometimes associated with the mite Myobia musculi appears to be more severe in this strain than others (Csiza and McMartin, 1976).

C57BL/10
Long life-span (826±29 days in males, 693±31 days in females). Overall tumour incidence 33% in males and 31% in females, most of which is due to lymphoma (31% in males, 29% in females) (Smith et al., 1973). Microphthalmia and anophthalmia 8-20% and hydrocephalus 1-3% (Dagg, 1966). Dermatitis leading to self-mutilation as described in C57BL/6 is also common in this substrain. Incidence may reach 4% (Sparrow, personal communication).

Normal physiology and biochemistry
Substrain unspecified:


C57BL/6

Low susceptibility to audiogenic seizures (6/6) (Deckard et al., 1976).

C57BL/10

Anatomy
Low proportion of basophilic cells in adenohypophysis (5/5) (Keramidas and Symeonidis, 1973).

C57BL/6

C57BL/10
Small kidney/body weight ratio (21/21) (Schlager, 1968). High number of bristles on foot pad (1/8 to 3/8 in three congenic lines) (Festing, 1976a). High yield of peritoneal exudate cells (1/5) with a high percentage of macrophages (1/5), low percentage of lymphocytes (5/5) and high granulocytes (1/5) (Schwartz et al., 1975).

Drugs

Resistant to chloroform toxicity (cf. 5/9) (Deringer et al., 1953). Resistant to induction of cleft palate by cortisone (4/5) (Kalter, 1965).

Resistant to lethal effects of ozone (22/22) (Goldstein et al., 1973). Resistant to colon carcinogenesis by 1,2-dimethylhydrazine (cf. 4/7) (Evans et al., 1977).

C57BL/Fa
Resistant to induction of lung tumours by urethane (6/6) (Falconer and Bloom, 1962). Insensitive to insulin (8/9), sensitive to histamine (2/9) (Brown, 1965).

C57BL/6

Sensitive to teratogenic effects of acetazolamide (2/6) (Green et al., 1973). Resistant to teratogenic effect (cleft palate) by cortisone acetate (2/6) (Kalter 1981). Hepatic epoxide hydrase activity induced by pentobarbital i.p. (cf. 4/7) (Oesch et al., 1973). Resistant to teratogenic effects of cortisone acetate (4/4) (Dostal and Jelinek, 1973). Resistant to lethal effects of ozone (16/21) (Goldstein et al., 1973). High incidence of convulsions induced by fluranyl (1/5) (Davis and King, 1967). Susceptible to hyperbaric oxygen (4/18) (Hill et al., 1968). Resistant to chloroform toxicity (cf. 5/9) (Hill et al., 1975; Deringer et al., 1953). Resistant to toxic effects of isoniazid (2/10) (Taylor 1976b). Sensitive, as judged by eosinophil response, to cortisone acetate (cf. 3/6) (Wragg and Speirs, 1952). High (89%) ovulatory response to 3 L.U. of PMS in immature mice (2/6), but only a 56% response to 7 L.U. No facilitation by exposure to males at these doses (Zarrow et al., 1971). High locomotor activity after treatment with \( \alpha \)-amphetamine (1/6) (Babbini et al., 1974). Nicotine increases learning ability (1/9) (Bovet et al., 1966). Resistant to colon carcinogenesis by 1,2-dimethylhydrazine (cf. 4/7) (Evans et al., 1977).

C57BL/10

Immunology
Poor immune response to low levels of bovine gamma-globulin (cf. 4/8) (Levine and Vaz, 1970). Poor primary immune response to bovine serum albumin (6/6) (James and Milne, 1972). Poor primary immune response to sheep erythrocytes (3/6 to 6/6, depending on test and dose) (Ghaffar and James, 1973). Poor immune response to Vi antigen (cf. 3/5) (Gaines et al., 1965). Low antibody affinity (7/7) and small quantity of antibody production (6/7) (Alpers et al., 1972). Low antibody affinity to HSA (9/9) (Petty et al., 1972).

C57BL/Fa
Serum anti-nuclear factor in 12% of animals tested (5/17) (Barnes and Tuffrey, 1967). Good primary immune response to bacteriophage \( \alpha \)d (2/7) (Kolsch et al., 1971).

C57BL/6
High susceptibility to induction of amyloid by casein (1/6) (Willerson et al., 1969). Poor immune response to type III pneumococcal polysaccharide (4/5) (Braley and Freeman, 1971). Poor immune response to synthetic double- stranded RNA (6/7) (Steinberg et al., 1971). Good immune response to cholera A and B antigens (2/8) (Cerny et al., 1971). Resistant to induction of anaphylactic shock by ovalbumin (cf. 6/13) (Tanioka and Esaki, 1971). Rapid rejection of about 76% of male skin isografts by females by 25 days (1/10) (Gasser and Silvers, 1971). Poor immune response to GAT (random terpolymer of Glu\(^6\), Ala\(^3\), Tyr\(^2\)) (9/10) (Dorf et al., 1974). Good immune response to Salmonella senftenberg (1/5) and \( S. \) anatum (2/5) lipopolysaccharide (Di Pauli, 1972). Non-responder to synthetic polypeptide Glu\(^5\), Lys\(^3\), Ala\(^5\) (cf. 4/7) (Pinchuck and Maurer, 1965). High sporadic occurrence of

C57BL/10

Infection
High susceptibility to BALB/Tennant leukaemia virus in Ks substrain (3/12) (Tennant, 1965). Resistant to \(\text{Herpes simplex}\) virus (2/11) in Ks substrain (Lopez, 1975). Resistant to oncogenic effects of polyoma virus given at birth (Law, 1966a). Resistant to \(\text{Mycobacterium marinum}\) (2/9) and poor plateau harvest of \(\text{M. leprae}\) 8 months after infection (9/9) (Shepard and Habas, 1967).

C57BL/Fa
Highly susceptible to infection by \(\text{Salmonella typhimurium}\) strain C5 (2/7) (Plant and Glynn, 1974).

C57BL/6
Develops a slowly progressing parasitosis ("low responder") after infection with the Cornell strain of \(\text{Toxoplasma gondii}\) (Macario et al 1980). Susceptible to \(\text{Salmonella typhimurium}\) strain C5 (1/5) (Robson and Vas, 1972). 100-fold more resistant to \(\text{Listeria monocytogenes}\) than A/J when measured by median lethal dose (Sadarakangi et al 1980). Resistant to \(\text{Mycoplasma fermentens}\) (6/6) (Gabridge et al., 1972). Resistant to infection by liver fluke \(\text{Opisthorchis felineus}\) (6/6) (Zeletsov, 1974). Highly resistant to the mammary tumour virus which is thought not to be carried by the strain (Murray and Little, 1967). Resistant to \(\text{Mycoplasma fermentens}\) (6/6) (Gabridge et al., 1972). Resistant to infection by liver fluke \(\text{Opisthorchis felineus}\) (6/6) (Zeletsov, 1974). Highly resistant to the mammary tumour virus which is thought not to be carried by the strain (Murray and Little, 1967). Resistant to \(\text{Herpes simplex}\) virus (2/11) (Lopez, 1975). Susceptible to mouse hepatitis virus type 3 infection (cf. 5/14) (Le Prevost et al., 1975). Develops antibodies to mouse hepatitis virus which can be reliably detected by the complement fixation test, in contrast to five other strains (Kagiyma et al 1991). Low mortality in a natural epizootic of ectromelia (7/8) (Briody, 1966). High expression of RNA tumour virus group-specific antigen in some substrains (1/8) but low in others (7/8) (Whitmire and Salerno, 1972). Resistant to development of leukaemia on infection by Friend virus (cf. 2/11) (Dietz and Rich, 1972).

C57BL/10

**Reproduction**

C57BL/Fa
Poor reproductive performance (25/25) with only 3 young weaned per litter and 0.4 young per female/week (Festing, 1976a).

C57BL/Ka
Good breeding performance, 2.2 young/female/month (6/24) (Hansen et al., 1973).

C57BL/6

C57BL/10

**Miscellaneous**

C57BL/Ka

C57BL/6

High rate of spontaneous mutations at the agouti and W loci (1/21) (Schlager and Dickie, 1967).

C57BL/10
High incidence of spontaneous 'deviants' (possible mutants) (2/21) (Schlager and Dickie, 1967).

C57BR/cd
Inbr (J) 178. Brown: a,b. Origin: Little in 1921 from the same cross that gave rise to C57BL, C57BR/a and C57L. Black and brown substrains were separated in the first generation. Substrain cd was established at F13 from a cross between two brown substrains, one of which had previously given rise to C57BR/a. To Heston 1938, to J 1947 at F66. Maint. by J.

**Behaviour**

Life-span and spontaneous disease
Primary lung tumours 3% in males, 1%, in breeding females and zero in virgin females, lymphatic leukaemia less than 1% (Hoag, 1963). Pituitary tumours 33% in old breeding females (Murphy, 1966).

Long life-span in conventional conditions (20/22 = 703 days in males and 19/22 = 694 days in females), hepatomas 25% in males (Storer, 1966). Life-span intermediate in both sexes in SPF fostered conditions (10/17 = 577 days in males, 9/17 = 660 days in females) (Festing and Blackmore, 1971).

Normal physiology and biochemistry

Low percentage of the time spent sleeping (6/6), with low percentage of slow-wave sleep (6/6) and high percentage of paradoxical sleep (1/6) (Valatx and Bugat, 1974). High proportion of paradoxical (REM) sleep (1/7) (Page et al., 1973).


Anatomy
Low brain weight (24/25) (Roderick et al., 1973). High total leukocyte count (5/18), high haematocrit (2/18), high mean corpuscular volume (3/18) and high haemoglobin (3/18) (Russell et al., 1951).

Drugs

Immunology

Reproduction
Intermediate breeding performance (12/25), colony output 0.98 young/female/week, litter size at weaning high at 6.5 (5/25) (Festing 1976a). Poor

Miscellaneous

C57L
Inbr: F 130 +. Grey (colour very similar to DBA). Genet: a, b, ln. Origin: Murray 1933 from a mutation in F22 of a C57BR substrain which is now extinct. Maintained by Cloudman, to Heston 1938, then to Jackson Laboratory 1947 at F45. Differs from C57BR/cd at the H-2, Igh-1, Pgk-2, Qa-2 and Qa-3 loci. Maint. by J, N.

Behaviour

Life-span and spontaneous disease
Low incidence of RNA tumour virus group-specific antigen expression (5/5) (Diwan et al., 1973). Primary lung tumours less than 1%; lymphatic leukaemia less than 1% in males and breeding females, but about 4% in virgin females; mammary adenocarcinomas 3% in breeding females, zero in males and virgin females (Hoag, 1963). 25% incidence of Hodgkin’s-like lesions, reticulum cell neoplasm type B at 18 months (Heston, 1963) (55% according to Dunn and Deringer, 1968). Pituitary tumours 33% in old breeding females (Murphy, 1966).


Normal physiology and biochemistry


Anatomy
Low brain weight (23/25) (Roderick et al., 1973). High total leukocyte count (3/18), high haematocrit (1/18), high mean corpuscular volume (1/18), high haemoglobin (2/18) (Russell et al., 1951). Adrenal gland has a small X zone (7/8) with a low incidence of vacuolisation (5/6) (Delost and Chirvan-Nia, 1958). About 38% of mice have accessory spleens (1/9) (Hummel et al., 1966).
Drugs


Immunology

Infection

Reproduction
Intermediate breeding performance (15/25), colony output 0.9 young/female/wk, litter size at weaning high at 6.0 (7/25) (Festing, 1976a). Good litter size, mean 5.6 (2/6) (Verley et al., 1967). Intermediate breeding performance (16/24) (Hansen et al., 1973).

Miscellaneous

C57P/A
Inbr (A): 115. Origin: R.Korteweg, 1934. Cross of DBA x C57BL then N20 backcrossing to C57BL, followed by b x s mating. (formerly listed as P/A). Maint. by A.

C58
Inbr: F166 +. Black. Genet: a. Strain developed by MacDowell in 1921 from litter mates 58 and 52 of Miss Lathrop’s stock (see also C57BL). A mating of the same male but a different female gave rise to the C57 group of strains. Used largely as a high-leukaemia strain, the genetics of which has been reviewed by Lilly and Pincus (1973).

Behaviour
Life-span and spontaneous disease

Normal physiology and biochemistry

Anatomy
Small brain/body weight (18/20). Small brain weight (Roderick et al., 1973). Low number of granule cells in right area dentata of brain (5/5) (Wimer and Wimer, 1982). A significant number of acinar cells of the pancreas are multinucleated (cf. AKR), in contrast to seven other strains (Pollard, 1973). Accessory spleens in about 21% of mice. One or both kidneys reduced or missing in 10-12% of individuals. Polyovular follicles common (Hummel et al., 1966).

Drugs

Immunology
Discriminator between 'H' and 'L' sheep erythrocytes (cf. 12/18) (McCarthy and Dutton, 1975). Develop immune polioencephalomyelitis, a paralytic central nervous system syndrome characterised by mononuclear cellular infiltration of the spinal cord and brain stem when aged mice are immunised with formalin-inactivated line 1b malignant lymphocytes (Sager et al., 1973). Erythrocytes have low agglutinability (cf. 11/25) (Rubinstein et al., 1974). High responder to dextran (cf. 4/10) (Blomberg et al., 1972).

Infection
Resistant to Mycoplasma fermentens (5/6) (Gabridge et al., 1972). Resistant to Plasmodium berghei infection (7/8) (Most et al., 1966). Encephalomyocarditis virus causes diabetes mellitus (cf. 7/14) (Boucher et al., 1975).

Reproduction
Poor breeding performance (22/24) (Hansen et al., 1973).

DA/Hu

DBA
Grey: a,b,d. Origin: Little 1909 from stock segregating for coat colour. Oldest of all inbred strains of mice. In 1929-30 crosses were made between substrains, and several new substrains established, including the widely used substrains /1 and /2. Differences between the substrains are probably too large to be accounted
for by mutation, and are probably due to substantial residual heterozygosity following the crosses between substrains. Thus DBA/1 and DBA/2 differ at least at the following loci: Car-2, Ce-2, Hc, H-2, If-1, Lsh, Tla, and Qa-3. With such large differences, they should probably be regarded as different strains rather than substrains of the same strain. DBA/LiA differs from /1 and /2 at the Gpd-1 locus, and is similar to DBA/2 at the Tla locus. Note that unfostered substrains carry the mammary tumour virus and have a high incidence of mammary tumours.

Main substrains are:

DBA/LiA
Inbr(A) ?+126. Origin: Little to Amsterdam circa 1932. Maint. by A.

DBA/1

DBA/2

Characteristics of substrains other than DBA/1 and DBA/2:
Ehling (1964) reported sensitivity to X-irradiation (1/5). Lung adenomas 1-11% in DBAf/A, and leukaemia 0-% in DBA/LiA and 5-8% in DBAf/A (Muhlbock and Tengbergen, 1971). DBA/Li is resistant to colon carcinogenesis by 1,2-dimethylhydrazine (cf. 4/7) (Evans et al., 1977).

DBA/1

Behaviour

Life-span and spontaneous disease
Primary lung tumours 3% in males, 1% in breeding females and zero in virgin females; lymphatic leukaemia less than 1%. Mammary adenocarcinomas zero in males, 90% in breeding females and 61% in virgin females in unfostered substrain (Hoag, 1963). A high proportion of the mammary tumours are of the acinar type (1/7) (Tengbergen, 1970). Lung tumours 2-27% (Festing and Blackmore, 1971). Low gross tumour incidence in males (19/22) (Storer, 1966).

Life-span of males short in conventional conditions (6/22 = 433 days) but long in females (21/22 = 750 days) (Storer, 1966). Life-span in SPF fostered conditions also short in males (5/17 = 487 days) and long in females (13/17 = 686 days) (Festing and Blackmore, 1971).

Normal physiology and biochemistry

Anatomy
Low brain weight (15/18 males, 18/18 females) (Storer, 1967). High erythrocyte count (1/18), low mean corpuscular volume (17/18) (Russell et al., 1951). Large number of Peyer's patches (1/7) (Hummel et al., 1966).

Drugs
Resistant to skin ulceration by DMBA (cf. 9/22) (Thomas et al., 1973). Resistant to induction of subcutaneous tumours by 3-methylcholanthrene (14/14) (Kouri et al., 1973), (12/12) (Whitmire et al., 1971).

Sensitive to X-irradiation (21/27) (Roderick, 1963). Males have a long sleeping time under hexobarbital (15/15) (Lovell, 1976), long sleeping time under pentobarbitone anaesthetic (23/23), Lovell (1986). Insensitive (eosinophil response) to cortisone acetate (cf. 3/6) (Wragg and Speirs, 1952). Sensitive to teratogenic effect (cleft palate) by cortisone acetate (2/6) (Kalter 1981)

Immunology
Low lymphocyte phytohaemagglutinin response (42/43) (Heiniger et al., 1975). Poor immune response to ovomucoid, but good response to ovalbumin (cf. 6/12) (Vaz et al., 1971). Good primary immune response to bovine serum albumin (2/6) (Lawrence and Milne, 1972). Good primary immune response to sheep erythrocytes (2/6 for haemagglutinin response at 3 x 10^7, 3 x 10^6 and 3 x 10^9 dose levels, 1/6 for haemagglutinin response at 3 x 10^8 dose only) (Gaffar and James, 1973). Non-discriminator between 'H' and 'L' sheep erythrocytes (cf. 6/18) (McCarthy and Dutton, 1975). Poor immune response to (Pro-Gly-Pro)_n (cf. 6/7) (Fuchs et al., 1974). High susceptibility to IgG-mediated (2/12) but low susceptibility to IgE-mediated (10/12) passive cutaneous anaphylaxis (De Souza et al., 1974). Good immune response to Salmonella strasbourg lipopolysaccharide (2/7) (Di Pauli, 1972). Erythrocytes have a high agglutinability (cf. 14/25) (Rubinstein et al., 1974).

Infection

Reproduction
Poor breeding performance (20/22), colony output 0.77 young/female/week, litter size 4.4 weaned (19/25) (Festing, 1976a).

Miscellaneous
Recommended host for the following transplantable tumours: anaplastic carcinoma dbRB, mammary adenocarcinomas CaD1 and T1703, melanoma S91 and pleomorphic sarcoma S37 (which is not host-specific) (Kaliss, 1972).


DBA/2

Behaviour

Life-span and spontaneous disease
Primary lung tumours 1% in males, 2% in females. Lymphatic leukaemia zero in males, 2% in females and 3% in virgin females. Mammary adenocarcinomas in unfostered substrains 1% in males, 72% in breeding females and 48% in virgin females (Hoag, 1963). A high proportion of mammary tumours are of the acinar type (1/7) (Tengbergen, 1970). Overall tumour incidence 15% in males, 49% in females, including lymphomas 10% in males and 12% in females; mammary tumours zero in males and 31% in virgin females (Smith et al., 1973). Leukaemia 3% (Myers et al., 1970).

Long life-span in SPF fostered conditions (12/17 = 629 days in males, 15/17 = 719 days in females) with 6-35% liver and 1-23% lung tumours (Pesting and Blackmore, 1971). Long life-span in conventional conditions (21/22 = 707 days in males, 20/22 = 714 days in females) (Storer, 1966). Life-span 722±30 days in males and 683±26 days in females (Goodrick, 1975).

High incidence of expression of RNA tumour virus group-specific antigen (2/5) (Diwan et al., 1973). Type B reticulum cell neoplasms 18% at about 20 weeks (Dunn and Deringer, 1968).

Spontaneous calcified heart lesions progress with age. 90% of individuals affected by 1 year (Rings and Wagner, 1971). Incidence of calcareous heart lesions high (1/5) among some related strains (Di Paola et al., 1964). Chronic hypertropic gastritis, duodenal polyps and calcareous pericarditis frequently observed. Other lesions include malignant lymphoma and degenerative processes in the myocardium, skeletal muscle, subcutaneous adipose tissue, cornea and blood vessels. Lesions partly depend on diet (Hare and Stewart, 1956).

Normal physiology and biochemistry

High basal level of growth hormone at 78 days (1/6) and low basal level of serum prolactin (6/6) (Sinha et al., 1975). High brain L-glutamic acid decarboxylase (2/7), choline acetyltransferase (2/7) and acetylcholinesterase (1/7) activity (Tunnicliff et al., 1973). Low brain sulphatide (5/5) and plasmalogen (5/5) and high brain sterol (1/5) (Sampugna et al., 1975). Low brain cholinesterase (5/5) (Pryor et al., 1966).

**Anatomy**

Small forebrain (9/9), neocortex (9/9) and hippocampus volume (8/9) (Wimer et al., 1969). Large heart/body weight (1/5) (Mokler and Iturrian, 1973). High proportion of acidophilic (1/5) and low proportion of chromophobe (5/5) cells in adenohypophysis of DBA/Sy substrain (Keramidas and Symeonidis, 1973).

**Drugs**
Resistant to skin ulceration by DMBA (cf. 9/22) (Thomas et al., 1973). Resistant to induction of subcutaneous tumours by 3-methylcholanthrene (12/14) (Kouri et al., 1973), (11/12) (Whitmire et al., 1971). Resistant to induction of adenocarcinomas of the colon by 1,2-dimethylhydrazine (cf. 2/4) (Evans et al., 1974).


**Immunology**
Infection
Resistant to infection by *Salmonella typhimurium* strain C5 (4/7) (Plant and Glynn, 1974). Susceptible to liver fluke *Opisthorchis felineus* (1/6) (Zelentsov, 1974). Susceptible to natural intestinal helminth infection (9/10) (Eaton, 1972). Develops a chronic non-healing lesion on infection with *Leishmania tropica*, the parasite causing cutaneous leishmaniasis (Howard et al. 1980).

Low susceptibility to BALB/Tennant leukaemia virus (10/12) (Tennant, 1965). Hyperglycaemia can be induced by encephalomyocarditis virus (cf. 2/6), which also causes diabetes mellitus (cf. 7/14) (Boucher and Notkins, 1973; Boucher et al., 1975). High susceptibility to develop leukaemia on infection with Friend virus (cf. 5/11) (Dietz and Rich, 1972).

Reproduction

Miscellaneous
Recommended host for the following transplantable tumours: fibrosarcoma SaD2, lymphatic leukaemia P1534 and mammary adenocarcinoma CaD2 (Kaliss, 1972). Hybrids involving DBA/2 are recommended host for transplantable leukaemia L1210, melanoma S91 and MOPC myeloma used as models in screening potential anticancer drugs (E.O.R.T.C. Screening Group, 1972).

The Fv-2r allele appears to be lethal on the DBA/2 genetic background (Blank and Lilly, 1976). High mortality after neonatal thymectomy (5/6) (Law, 1966a).

DC
Inbr (Le) 100. Agouti, +. Origin: mutation to dancer Dc arose in an obese stock outcrossed to a BALB/c x C3H/He hybrid in 1956. One cross to C3H/HeJ, then inbred. Heterozygotes exhibit circling and head tossing behaviour but are not deaf. Homozygotes die at birth with cleft lip and cleft palate. Maint. by Le.

DD

DDD
Inbr: F 50 +. Albino. Genet: c. Origin: Outbred dd stock from Germany to Kitasato Institute before 1920. To Manchuria, and back to Japan, with inbreeding started in 1962 by Suzuki. Charac: Mammary tumours 8% at 14 months in virgin and 14% at 14 months in force-bred females. Mammary tumour virus carried by the strain is probably of plaque-inducing group found in DD, RIII and BR6 but not in C3H (Matsuzawa et al., 1974). Develop a high incidence of heritable hydronephrosis, with a higher incidence observed in males and females. This appears to be...

**DDI**

**DDK**
Inbr: F105. Albino. Genet: \(A, B, c, D, S\). Developed by K. Kondo from outbred dd stock from Institute of Infectious Diseases, University of Tokyo, in 1944.

Charac: DDK females mated to C57BL males are semi-sterile, but the reciprocal cross is fully fertile. The low 'fertility' is caused by embryonic death at the morula-blastocyst or pre-egg cylinder stage 3-5 days after fertilisation. A deficit of trophoblast formation was observed. Transplantation experiments show that the defect is the property of the embryos, not the uterine environment. The DDK karyotype appears normal (Wakasugi, 1973). Tendency to diabetes (Nishimura, 1969).

**DDN**
Inbr. 27. Albino: \(A, b, c\). Origin: Outbred Jcl:ddN inbred in 1972 by R. Shoji. Selected for expression of spontaneous forefoot post-axial polydactyly found in the ddN mice. Maintained by Idr

**DDP**
Inbr. 52. Albino: \(A, B, c\). Origin: Outbred Jcl:ddN mice inbred in 1972 by Shoji. Selected for the expression of postaxial polydactyly, which is now almost 100% in both forefeet of these mice. Maintained by Idr.

**DDY**
Inbr: 72. Genet: \(a, B, c\). Origin: From non-inbred dd of Institute of Infectious Diseases, University of Tokyo, 1953 (Tajima, 1968).

**DE**
Inbr: 60 +. Off-white. Genet: \(c^e\). A. Origin: Eaton 1940, from cross of CE/Wy x E/Gw, selected for \(c^e\) phenotype. Charac: High incidence of amyloidosis; have polydipsia (30–50 ml/day) and polyuria (25–45 ml/day), more noticeable in females; specific gravity of urine is 1.005.

**DF/Wf**
Inbr: 26. Genet: \(a^e, Df/df\) (\(df\) is Ames dwarf.) Origin: + \(df\) from A. Bartke 1965, crossed with C57BL/6JNclr, then sib-mated. Charac: Large, vigorous; adults as well as young are jumpers.

**DHS**

**DK**
Inbr (Lm) 28. Brown, non-agouti, yellow \(A^p/a, b\). Yellow mice turn dark (sable) at first molt. Descended from mice of TOE, By, RW and JUN stocks (Harwell). (Lamoreaux and Galbraith 1986). Maintained by Lm.
DKI
Inbr: F61. Albino. Genet: a, c. Strain developed from dd outbred stock of Central Laboratory of Experimental Animals. Inbreeding started by B. Kitasato in 1953. Charac: Susceptible to Salmonella enteritidis. A congenic strain in which the gene for resistance found in C3H/He has been backcrossed to DKI has been developed (Kishimoto, 1972).

DKI-R

DL

DLS

DM/Mk

DM
Inbr. F70+10 (Shi). Albino: a,B,c,D,S. Origin: One pair of inbred dd mice (F69) from Ms to Shionogi Research Labs. in 1971. Coat colour genes of DM/Ms are reported to be A,b,c (Exp. Anim. 17:31, 1968), but this substrain is a,B. Maintained by Shi.

DMC

DOPG

DRC
Inbr(Hok) ?+48. Albino (?). Origin: DDD mated to C57BL/6.F1 backcrossed 8 times to DDD then b x s. To Hok 1972 from Univ. Tokyo. Maint. by Hok.

DSD
D103/Ms

EBT

EL

F/St

FB

FL/1
Inbr (Brk) 93. Black a, f, rd. Origin: f, flexed-tail gene (causing microcytic anaemia) originally obtained from Jay came from Snell's WA linkage testing stock. Seven rounds of cross-intercross to C3H by Jay and E.S. Russell. Outcross to WB/Re by Re, with b x s mating of f/f progeny started in 1956. Charac. Foetuses and new-born f/f mice show a transient siderocytic anaemia which is cured by 14 days. Variable belly spotting and tail flexure. Good breeding performance when young, later impaired by obesity. Maint. by Brk.
FL/4

FM

FRG

FS

FSB
Inbr (Dn) 98. Black a, fs. Origin: furless mutation fs arose in unpedigreed stock at Ohio State University in 1951. Mutant females mated with C57BL/10 males. Charac. Low fecundity and viability. fs/fs mice grow a normal coat, but this thins at about 19 days. A new coat grows but persists only a short time. Mature mice are partly devoid of hair at all times. All types of hairs are shorter than normal (Green, 1954). Maint. by Dn.

FTC

FVB

G/Gw
Inbr: 75 +. Origin: Goodale, selected for body weight, to Gowen at Iowa State; b x s inbreeding; to Nash at Colorado State 1967. Charac: Hyperglycaemic, with mean plasma glucose levels of 230 mg/100 ml at 60 days, at which time males are about 41 g and females 35 g. Normal response curve following i.p. glucose tolerance test and no gross pathological changes with aging (Nash and Logsdon, 1974).

GL
Inbr (Le) 50. Agouti: *. Origin: mutation to grey-lethal (gl) discovered in a stock segregating for c^e by Gruneberg in 1935. From G to Jay to M.Dickie to P.Lane who inbred to F25, then one outcross to dI/dJ^2 (downless, closely linked to gl and used as a linked marker), and b x s mating as a balanced stock. Maint. by Le

GLF
Inbr (Y): F65. Agouti, *. Origin: From Jax as bearers of grey lethal mutation; selected for the normal genotype by progeny testing. Maint. by Y.

GRS
Inbr (A) 101. Albino a,c. Origin: Formerly called GR/A. Muhlbock 1965 from outbred mice obtained from Grumbach in Zurich.
Breeding females have a high incidence of mammary tumours which are highly hormone-responsive. Carries a mammary tumour agent different from MTI, transmitted by milk and gametes, which is not eliminated by foster nursing. Hormone-dependent tumours are also produced in GRS x RIII F1 hybrids (Muhlbock, 1965; van Nie and Thung, 1965; Brcand and Daehnfeldt, 1973). High incidence of hepatomas in mice treated with normal horse serum or horse-anti-mouse antilymphocyte serum (Den Engelese et al., 1976). Leukosis 11% (Hilgers and Galesloot, 1973). Susceptibility to mammary tumour induction with progesterone and oestrone in ovariectomised mice may be due to a single gene, although linkage with eighteen marker loci could not be established (van Nie and Hilgers, 1976).

**GT**

Inbr (Le) 28. Breeders are agouti: A,gt/+;gt/gt are light grey with white belly spot and have tremors like myelin deficient mice. Origin: Developed by Lane. gt mutation arose in strain HYIII/Le in 1977 at F55. One outcross to C3HeB/FJ x C57BL/6-A<sup>w</sup>-F1, then bxs. Has a spontaneous spongiform encephalopathy whose expression is determined by the interaction of genetic factors and an unconventional unrecognised transmissible agent (Sidman et al. 1985) The stock is maintained by heterozygous breeders that are kept in isolation. Maint. by Le.

**HC**


**HLC**

Inbr 47. Colour ?. Origin: from hybrid stock derived from crosses involving C57BL/6J, C57BR/cd, A/J, BALB/c, LG, and SM. Selected for high leukocyte count (strain LLC was selected for low leukocyte count). Mean total leukocyte count 36,000 in males to 38,000 in females. Thymus and spleen weights much higher than in strain LLC. Maint. by Harrison (J).

**HLS**

Inbr. F75. Albino. Origin: Stock from Univ. of California (Dr. Epstein) to Yokohama Univ. to Hok in Jan 1967 at F40. Crossed several times to SWJ/Hok, then sib mating. Hairless. Maintained by Hok.

**HPG**

Inbr (Bm) 21. Agouti. Origin: WG Beamer, 1988 following hysterectomy and fostering on B6D2 of a stock carrying hpg (hypogonadal) mutation obtained from Bruce Cattanach. Maintained by Bm.

**HPT**

Inbr (Le) 26. Agouti, segregating patchy coat; A, Hpt/+. Origin: Priscilla Lane 1988 from a spontaneous mutation (Hpt, hair patches, MNL 65:29) which arose in a segregating hybrid (C57BL/6JxC3HeB/FJLe-a/a) background at N3 in 1979. Hpt was backcrossed three times to this hybrid, then inbred. Maintained by Le.

**HR/De**

Inbr: F78 +. Pink-eyed dilute strain carrying hairless mutation. Genet: p, hr with forced heterozygosity. Inbred by Deringer from hr stock received from Carnochan in 1948. Charac: Haemangioendotheliomas 19-33% (Heston, 1963). Skin papillomas in 3-9% of hairless animals at 18-22 weeks (Murphy, 1966). High incidence of haemangioendotheliomas (54-76%) in mice treated with 4-0-tolylazo-Otoluidine (Heston, 1963). Sensitive to chloroform toxicity (cf. 4/9) (Deringer et al., 1953). Type B reticulum-cell neoplasms at 20 weeks 12% (haired) and 8% (hairless) (Dunn and Deringer, 1968).
HRA.HRII-c/+  
Inbr (Skh) F30N11. Albino and pigmented (thought to be a,b,c or C,hr). Origin: PD Forbes by backcrossing outbred pigmented mice to the HRA/Skh strain (F?+30, homozygous for hr) with selection for both pigmented and albino hairless offspring. Strain homozygous for the hr gene. Comparative immune responses of HRA/Skh described by Smith et al (1982).

HRS  
Inbr. F79 (J). Albino, b,c,d. Origin: Hairless (hr) stock from Crew to Carnochan to Heston to Chase, to E.L.Green 1952, to Les 1956, to M.C.Green 1959, to J 1964. Maintained by mating +/hr females with hr/hr males.

Charac: Homozygous hairless mice lose their hair at about 10 days. The complete hair is lost from the follicle. After a time a few thin fuzzy hairs grow again, but are soon lost. These are exclusively guard hairs. There is hyperkeratosis of the stratified epithelium and the upper part of the hair canals. Hair cell formation is abnormal and the lower part of the follicles tends to separate from the upper part. The isolated lower parts develop into cysts, which may become large and numerous (M. C. Green, 1966). About 45% of hrhr mice develop leukaemia by 8-10 months compared with only 1% in hr/+ mice. Graft versus host assay shows that hrhr mice are immunologically hyporesponsive, which may be associated with the high leukaemia incidence (I’Anson and Gasser, 1973). Similarly, Heiniger et al., (1974) found 70% leukaemia at 8 months in hrhr mice but only 20% in hr/+.


HSFR  

HSFS  
Inbr. ?. Albino c. Origin: As HSFR, but selected for sensitivity. Maint. by N.

HTG  

HTH  

HTI  

HYIII  
Inbr (Le) 83. Agouti: *. Origin: Mutation to hydrocephalus-3 discovered in heterogeneous stock by H.Gruneberg. To M.C.Green 1963, then b x s. To Lane 1975. Homozygous hy-3/hy-3 mice die with frank hydrocephalus by 4-5 weeks. Maint. by Le.

I  
Inbr:(N) F143. Pink-eyed fawn with variable white patches. Genet: a, b, d, p, s, with some substrains carrying ln and/or c^h. Origin: Strong 1926 from unpedigreed mice. Carries the b allele at the Phk locus, a sex-linked locus that controls level of activity of the enzyme phosphorylase kinase. This activity is
virtually absent in muscle and reduced in the brain, kidney and heart in this strain.

**Behaviour**
Short latency to attack crickets (1/7) (Butler, 1973). High hole-in-the-wall entries (2/7), high Y-maze exploration (1/7), high number of stairs climbed (1/7), high urination (1/7) and defaecation (2/7) (McClearn et al., 1970). Low alcohol preference (Rodgers, 1966).

**Life-span and spontaneous disease**
Spontaneous adenomatous stomach lesion occurs in nearly all mice (Heston, 1963).

**Normal physiology and biochemistry**
Mammary gland sensitive to oestradiol and progesterone (2/7) (Singh et al., 1970). Poor growth rate, and no response to fat in diet (4/4) (Fenton and Carr, 1951). Carries Phk, a sex-linked phosphorylase kinase deficiency leading to a 3-4-fold elevation of skeletal muscle glycogen content (Gross et al., 1975). Acutely sensitive to vitamin B₆ depletion. Brief depletion which causes only moderate weight loss in other strains results in hyperactivity and convulsions followed by death. Tissue stores of B₆ are not different from normal. Sensitivity not due to malabsorption, rapid excretion or failure to form a cofactor at normal rate (Bell and Haskell, 1971; Bell et al., 1971). C3HF x I F₁ hybrid used as a model of obesity and diabetes. Characterised by moderate obesity at 3-4 months, glycosuria in 50% of males but only 5% of females. Islets of Langerhans enlarged, with increased insulin levels. Abnormalities associated with hyperphagia and may be prevented by food restriction (Bray and York, 1971; Stauffacher et al., 1971). Pure-line strain I resistant to dietary induction of obesity (Fenton and Dowling, 1953). Thyroid epithelial cells contain crystals in membrane-bounded dense bodies, which may be lysosomes (Neve and Wollman, 1973). Complement undetectable (Staats, 1976).

**Anatomy**
High leukocyte count (1/18), high red blood cell count (4/18), high haematocrit (4/18) (Russell et al., 1951).

**Drugs**
Resistant to skin ulceration by DMBA (cf. 9/22) (Thomas et al., 1973). Susceptible to papilloma induction by methylcholanthrene (1/5) (Andervont and Edgcomb, 1956), but resistant to fibrosarcoma induction by methylcholanthrene (15/15 in males, 14/15 in females) (Strong, 1952).

**Immunology**
Low lymphocyte phytohaemagglutinin response (34/43) (Heiniger et al., 1975). Low immune response to ferritin (13/16) (Young et al., 1976).

**Reproduction**
Poor reproductive performance, with a high incidence of maternal neglect (Andervont and Edgcomb, 1956).

IC

ICRC
Inbr ?. No details. Maint. by Cri.

ICFW

ICR/Ha (NB. Duplicate name)

ICR/Bc (NB. Duplicate name)

IF

ITIES
Inbr: F76 (Nga). Genet: a, b, C, d, s. Origin: Crossbred among CS, DBA/2, NBC and ITIES. Charac: Useful for testing colour genes.

IM

IOR (Hab) 65. Albino: c. Origin: R. Castillo Menendez from 1970 from outbred albino mice. High fertility, low mortality and rapid growth in tropical conditions. Tumour incidence 32%. Amyloidosis and chronic nephritis seen in old animals. 95% of old animals also develop cataracts which may be caused by a recessive mutation Menendez and Abдрашихитова (1990). Maintained by Hab.
IQI

IS
Inbred (Dn) 42+13. Agouti: +. Origin: Mus musculus praetextus male caught in Israeli port x M.m. musculus female from laboratory stock carrying bt, m, b, and a. To Roderick, to Eicher 1971 at F42, to Dn 1983. Maint. by Dn.

ITES

IVCE
Inbred: 48. Origin: Separated during inbreeding and selection of IVCS. Charac: Docile; regular 4 day oestrus cycle; litter size 9; mean body weight at 21 days, females 9.2, males 9.4; at 70 days, females 25.1, males 29.9 (Nobunga, 1973).

IVCS

IXBL

J/Glw

JBT

JE

JGBF
Inbred (Ty) F55. Khaki: a,jg,+/+,bf. Origin: mutation to jagged-tail occurred in C3H/HeJ in 1960. Crossed once to C57BL/10 and b x s, then crossed once to C57BL/6J-bf and inbred as a balanced stock. Homozygous jg/jg mice are usually born dead. Ty maintains it as an RI line, Le as a balanced mutant strain. Maint. by Ty.

JIGR
Inbred (Dn) F65. Agouti: +. Segregates for gr (grizzled) and ji (jittery). Origin: ji arose in waltzing (v) stock of Snell before 1957. bxs to F15. To M.C.Green (1963) and outcrossed to gr/gr stock (F30). Balanced stock bxs. To Eicher 1972, then
Davisson 1980. gr from Falconer to Snell 1950. Two crosses to CBA then bxs to F30. Cross to ji/+ to make the balanced stock. ji/ji die before weaning. gr/gr often poor breeders. Maint. by Dn.

**JU**
Inbr: F41×60 (Ct). Albino. Genet: a, c. Origin: Falconer 1952, from crosses involving Goodale's and MacArthur's large strains, Bateman's high-lactation strain and various mutant stocks with about 50% of C57BL/Fa ancestry. It was the only survivor from an inbreeding experiment involving twenty lines (Falconer, 1960b). Falconer to Cattanach in 1966. Charac: Average first litters 9; high prenatal mortality (50%) in second litters when gestation is concurrent with suckling first litter; low penetrance of nil. (Staats, 1976). Lm maintains a number of substrains with different pigment mutations including a non-albino +c. Maint. by Ct.

**JU/Ct-C.**
Inbr. N5F59 (Ct). Black, a.C. Derived from a cross between Jt female (F41) with a CBA male, five backcrosses to JU, then selection for the CC genotype upon sib mating.

**JU/Ct-C,A.**

**K**
Inbr ?. Albino a,b,c. Origin: Probably from the Rockefeller Inst. in the 1930's. To Gowen, Ames in about 1937. One of four strains from Rockefeller characterised for bacterial and/or viral resistance. This strain is listed as resistant to both (though this is relative). To D.Grahn, Aregonne in 1963. Moderate resistance to S. typhimurium. Sensitive to irradiation. Long life expectancy. No special neoplasia.

**KC**
Now designated CPB-K.

**KE**
Inbr:F94 (Kw). Genet: a, b, c, P. Origin: Krzanowska 1952, from mice of unknown origin; cross also produced KP. Charac: Mean litter size 6.1; low percentage of fertilised ova. 17.6% of spermatozoa have abnormal heads, the inheritance of this character being connected to the Y chromosome and may be due to a rel. deficiency of androgens (Bartke and Krzanowska, 1972).

**KF**

**KI**

**KK**

Characteristics
Strain develops diabetes mellitus associated with insensitivity to insulin and intolerance to glucose without hyperglycaemia. When obesity is induced by nutrition, the yellow obese gene Ay or gold thioglycosulose treatment, the mice
develop hyperglycaemia accompanying marked insensitivity of adipose tissue to insulin (Taketomi et al., 1973). Mode of inheritance of glycosuria depends on both genetic and environmental factors with at least two major genes which are dominant in crosses with C57BL (Butler and Gerritsen, 1970; Butler, 1972). Moderate obesity (mature weight about 45 g) occurs by about 2 months and stabilises by 4-5 months. Carcase fat is about 33% of total. Bray and York (1971) state that the diabetes is characterised by hyperglycaemia, hyper-insulinaemia, glucose intolerance and hyperphagia, although the hyperglycaemia abates by about 1 year. Food restriction makes the animals more normal. There is an elevated pituitary growth hormone level, and significant glomerular lesions. Opperman et al (1975) found that fasting results in impaired glucose tolerance.

Corneal degeneration starts early in life, is progressive with age, tends to be bilateral, and is confined largely to the anterior part of the corneal centre (Huang and Sery, 1971). The obese syndrome is also described by Stauffacher et al (1971).

Resistant to development of anaphylactic shock from ovalbumin (cf. 6/13) (Tanioka and Esaki, 1971).

■ KP
Inbr: F91 (Kw). Fawn (?). Genet: a, b, p. Origin: see KE. Charac: Mean litter size 5.0; high embryonal and postembryonal mortality; frequent sterile matings; low sperm production and low libido of males; testis abnormalities: degeneration of some tubules and cells, large amounts of interstitial tissue (Staats, 1976). Maint. by Kw.

■ KR

■ KSB

■ KYF
Inbr. 83. Brown or chocolate with white spotting: a,b,C,s. Origin: KYF/Ms mice to Shoji. Maintained by Idr.

■ LCS
Inbr. ?. No details. Maint. by Cri

■ LDJ
Inbr (Le) 72. Black or very black: a,mg/*. Also carries ld^*/+. Origin: ld^+ arose spontaneously in CBA/Ca-se stock (M.C.Green) 1959. One cross to C57BL/10Gn, bxs to F3, outcrossed to an inbred mg/mg stock (F27), and bxs as balanced stock. To Lane 1975. The mg mutation arose in a cross of C3H female x Swiss stock in 1950, with bxs until 1950. Maint. by Le.

■ LG
Inbr (J) 104. Albino. Genet. a,c, rd. Origin: Developed by Goodale with selection for large body size begining in 1931 (Chai 1961). Two substrains, J and Ckc were separated at F27. LG/J is H-2^d, Pgk-2^b while LG/Ckc is H-2^ari. Pgk-2^a. LG/J should not be confused with MacArthur's large strain which has never been established as an inbred strain and is now extinct.

LIS

LLC

LM

LMM (was LM)
Inbr (Bc) 49. Agouti. Origin: "C3H" mice from Rockland Farms in 1966, inbred by J.R.Miller, then outcrossed to SWV at F27, followed by sib mating. Resulting colony 1/8th. SWV, and the rest from the original stock. Homozygous for the Ig^ld mutation resulting in 94% open-eyes at birth. (Note. Name changed by Editor in order to avoid duplicate name. LM above appears to have priority). Maint. by Bc.

LP

Characteristics


High lymphocyte phytohaemagglutinin response (9/43) (Heiniger et al., 1975). Poor immune response to ovomucoid, but good response to ovalbumin (cf. 6/12) (Vaz et

Resistant to induction of diabetes mellitus by encephalomyocarditis virus (cf. 7/14) (Boucher et al., 1975).

**LPT**
Inbr (Le) 89. Colour: agouti. Origin: Mutation to loop-tail (*Lp*) arose in strain A in 1949. From W.Holland to Snell 1950. Crossed 7 times to C57BL/6J, once to C3H/He, then b x s. To Lane 1969. Loop-tail is a semi-dominant mutation with *Lp/+* mice having crooked or looped tails and some head wobbling. Homozygotes die at birth. Maint. by Le.

**LS**
Inbr (Le) F80. Black-and-tan, or black and tan with white spots: *a5*/*ls/*a5+. Origin: Mutation to lethal spotting (*ls*) occurred in C57BL-*a5* mice at Harwell. Phillips to Lane as balanced stock in 1961. Homozygous *ls/*ls develop megacolon, but some survive and breed. Maint. by Le.

**LST**

**LT**

Charac: Ovarian teratomas occur spontaneously in about half of the females. Ultrastructurally the stem cells do not differ from those of testicular or embryo-derived teratomas (Damjanov et al., 1975). Some tumours begin to develop at about 30 days and the incidence rises to 50% at 90 days. These resemble normal embryos until blastocyst stage and then become disorganised. A small percentage of ovulated eggs also develop parthenogenetically, but die at 5-7 days (Stevens and Varnum, 1974).

**LTS**

Charac: High mammary tumour incidence in both virgin and breeding females; closely related to LIS/A and STS/A, as deduced from isoenzyme patterns. A fostered low-tumour line, LTSfA, is also kept. Leukosis 6% in unfostered substrain and 18% in fostered substrain (Hilgers and Galesloot, 1973).

**LSXSS**-
Set of 27 recombinant inbred strains developed from a cross between outbred stocks SS and LS, selected for short and long sleeping times, respectively, under ethanol anaesthetic. Mean sleeping time varies from 36 to 171 minutes in the 27 strains (DeFries et al 1989).

**MA**
females, with gross kidney disease in males. Highest blood pressure among 21 strains tested. Maint. by J.

Characteristics
Primary lung tumours 37% in males, 42% in females. Lymphatic leukaemia 1% in males and breeding females, zero in virgin females. Mammary tumours zero (Hoag, 1963). Low gross tumour incidence (22/22) (Storer, 1966). Polydipsia-polyuria (Bernstein, 1966), which may be associated with cysts pressing on posterior lobe of pituitary (Russell and Meier, 1966). Life-span intermediate in both sexes (7/22 = 459 days in males, 12/22 = 585 days in females) (Storer, 1966).


Highly susceptible to the mammary tumour virus, which is not normally carried by the strain (Murray and Little, 1967).

MAS
Inbr (A) 98. Albino. Genet. c. Origin: Muhibock from same stock as GRS, and is similar to it at 48/50 biochemical loci. Maint. by A.

Charac: Low mammary tumour incidence in females, high lung tumour in both sexes. As is BALB/c, this strain is susceptible to both C3H mammary tumour virus and GRS mammary tumour virus. Very similar to GRS/A: of over 50 genetic markers, only two are different. Leukosis 22% (Hilgers and Galesloot, 1973).

MB

MH/Re
Inbr: 40. Genet: mk/+ . Origin: Jax mutant No. 63-36, B6D2F1-mk from M. M. Dickie to E. S. Russell 1963. Charac: Segregates for compensating microcytic anaemia. All mk/mk are born alive; one-third to one-half die before 3 weeks, developing skin lesions and tail amputation, but surviving mk/mk are fertile and appear normal except for supernormal numbers of very small erythrocytes. Haematology well characterised (Staats, 1976).

MIG

MIW

MK
Inbr (Ty) 78. Colour ?. Origin: Mutation to mk (microcytic anaemia) in descendents of a cross between C57BL/6J and DBA/2J. From M.M.Dickie to Re in 1963. Segregates for a gene, mk, causing microcytic anaemia in homozygotes apparently due to a generalised impairment of cellular iron uptake. Maint. by Ty.

MM

MO/Ko
Inbr: 75. Origin: Kobozieff 1949, from local mice of unknown ancestry which had corneal opacity, since shown not to be hereditary. Charac: Used for the study of longitudinal hemimelia, alopecia, periodic hypotrichosis.

MOA

MOC

MOL/3.

MOLD.
Inbr 50. Developed from wild Mus musculus molussinus without any intercrossing with laboratory mice. Differs from laboratory mice at many biochemical loci and also in skeletal morphology (Festing and Roderick 1989). Maint. by Rk.

MOR

MOM

MRL
Inbr (J) 65. Albino: a,c. Origin: Murphy from crosses started in about 1960 involving a number of standard inbred strains. Now estimated to have a composite genome of LG (75%), AKR/J (12.6%), C3H (12.1%) and C57BL/6 (0.3%). A mutation lpr (lymphoproliferation) was found in the 12th generation of b x s. Homozygotes develop massive generalised enlargement of the lymph nodes and autoimmunity, and usually die at 14-16 weeks of age. Origin and characteristics

- MS

- MSM
  Inbr. 20. White-bellied agouti A^w,b,c. Origin: Wild mice trapped in Mishima City, Shizuoka Prefecture, Japan and inbred in 1979 by Moriwaki. Has not been crossed with laboratory mice. Previously called M.MOL-MSM.

- MT

- MTH

- MWT

- MY
  Inbr (Le) 98. Chocolate: a,b. Origin: C57BR/cd female x line 85 F71 male from MacDowell in 1948. F8 male outcrossed to C3HeB/HuJ; selected for aa,bb in F2 generation. Carries my/my, but the only visible effect is missing eyes, though kidneys may also be missing. Poor breeders. Maint. by Le.

- MYD
  Inbr (Le) MYD is N7F12, MYD-+/+ is F71. Agouti: +. Origin Mutation to myd (myodystrophy) occurred in 1963 in is (lethal spotting) stock from R.Phillips in 1961. Crossed to C57BL/6J-^A^+/, then b x s. Os from ROP crossed to +/+ and to myd/+/ (N7). Maintained as Os,+/+myd balanced stock. A +/+ substrain was derived at F35. myd homozygotes have a progressive and diffuse myopathy of all skeletal muscles and die between 5 wks. and 5 months. Maint. by Le.

- N

- NAKED

- NBL
NBR

NC

NCU/CpbU

ND2/Rij

NFR

NFS
Inbr (N) 57. Albino a,c. As for NFR, but selectively bred for sensitivity to the action of histamine after treatment with Bordetella pertussis. Maint. by A,N.

NGP

NH
Inbr: F109 (Ao). Dilute spotted. Genet: a, d, p, s. Origin: Strong, from crosses involving CBA, N and JK. Charac: Low tumour incidence; resistant to induction of leukaemia by Moloney virus (1/9) (Law, 1966b). Nodular hyperplasia and adrenal adenomas in at least 10% of old mice; gonadectomy at 6 weeks enhances adenomas; some obesity; breeding ceases at approximately 9 months.

NIH

NLC

NMRI

■ NRH

■ NOD
Inbr (Komeda) 22. Albino: c. Origin: A substrain developed by selection for diabetes from F6 of the CTS strain, which was derived from JCL:ICR (Makino et al 1980). About 80% of females and 20% of males develop insulin-dependent diabetes by the age of 30 weeks. Genetic analysis suggests that the diabetes is dependent on multiple recessive loci including one associated with the H-2, and another with the Thy-1/Apoa-1 loci (Prochazka et al 1989, Wicker et al 1989). Maint. by Jic, Wak.

■ NON
Inbr (Komeda) 27. Albino: c. Origin: A non-diabetic substrain with the same origin as NOD. Maint. by Wak.

■ NOR (Dn) Reserved symbol.

■ NXSM-
Inbr circa 20. Set of 17 recombinant inbred strains developed by Eva M. Eicher from NZB/BINJ x SM/J. Typed at 58 loci on 16 autosomes and the X chromosome (Eicher and Lee 1990). Maint. by Ei.

■ NX129-
Inbr circa 20. Set of 8 recombinant inbred strains developed by B.A.Taylor from NZB/BINJ X 129/J. Maint. by Ty.

■ NYLR

■ NZB
Inbr: F121 (J). Black. Genet: a. Origin: Outbred mice from Imp. Cancer Research Fund, London, to Univ. of Otago Med. School 1930. Inbred by Bielschowsky 1948. A number of other strains, including NZO, NZC, NZX and NZY, were developed from the same stock. (Bielschowsky and Goodall, 1970). Strain NZW was derived from the same outbred stock, but was inbred independently by Hall (Hall and Simpson, 1975).

Life-span, spontaneous disease and immunology
Develops autoimmune haemolytic anaemia of the Coombs-positive, warm antibody type (Simpson, 1976; Howie and Simpson, 1974) as well as a nephropathy which is variable in expression and unpredictable in progress, but is probably an
immune-complex-induced glomerulonephritis. Burnet (1972a, b) considered that at least two genes are involved, one of which is also present in NZC. A virus may also be involved, although Simpson (1976) considered that: '...the case for a viral aetiology is unproven, although the possibility exists that virus may be present in incomplete form'. According to Burnet, NZB mice have an abnormally high immunological vigour and resistance to induction of immunological tolerance or paralysis, which is manifested before the animals become Coombs-positive. The condition may be transferred to young isogenic mice by cells from the spleen, but not from other lymphoid organs. Thus, the condition appears to depend on stem cells of immunocyte lines. Autoimmune plaque-forming cells, active against mouse erythrocytes, are present in old mice. Onset and severity of the condition can be influenced by diet (Fernandes et al., 1972). Theofilopoulos et al (1980) have compared immune function in this and other autoimmune strains. Only NZB splenic lymphocytes from autoimmune donors inoculated into pre-autoimmune NZB or in BALB/c mice could evoke a positive Coombs test (Jenkinson and East (1980).

In hybrids with C57Bl there is a late-appearing positive direct Coombs test. Hybrids with NZW develop an autoimmune disease resembling human systemic lupus erythematosus (Talal et al., 1972), with high titres of natural thymcyto-toxic autoantibody in many animals (Shirai and Mellors, 1972). Pure-line mice have a high level of natural thymocytotoxic autoantibodies (Auer et al., 1974), a low immune response to Dextran (cf. 6/10) (Blomberg et al., 1972), a low lymphocyte phytohaemagglutinin response (30/43) (Heiniger et al., 1975), a high 25% incidence of serum antinuclear factor (4/17) (Barnes and Tuffrey, 1967) and a poor immune response to DNP-keyhole limpet haemo-cyanin (9/11) (Borel and Kilham, 1974), and are discriminators between 'H' and 'L' sheep erythrocytes (cf. 12/18) (McCarthy and Dutton, 1975). Mean life-span short (2/17 = 459 days in males, 441 days in females) in SPF fostered conditions (Festing and Blackmore, 1971). Median life-span short (4/4 = 280 days males, 4/4 = 270 days females) (Stutman, 1974).

Hypertrophy of the pituitary in 80% of survivors to 1 year and pituitary tumours in 25% of aged breeders (Russfield, 1966).

Other characteristics

Susceptible to mouse hepatitis virus type 3 infection (cf. 5/14) (Le Prevost et al., 1975). No transmission of murine leukaemia virus (Scripps) to succeeding generations (Jenson et al., 1976). Carries no detectable endogenous ecotropic MuLV DNA sequences (Jenkins et al 1982). In contrast to ten other strains, it does not carry type I and II endogenous type-c viruses (cf. SWR) (Stephenson et al., 1975). Totally refractory to infection by Leishmania tropica parasite (Howard et al 1980)

Poor reproductive performance (24/25). Litter size 3.8 at weaning, colony output 0.5 young/female/week (Festing, 1976a). First litter size high (1/6) but fourth litter low (6/6). Low proportion of females produce four or more litters (6/6) and low percentage of fertile matings (6/6) (Fernandes et al., 1973). Intermediate breeding performance (17/24) (Hansen et al., 1973).
Inbr ?. Chocolate brown a,b. Origin: Presumed mutation to brown (b) in NZB/Bi at F73. Develops autoimmune anaemia like NZB. Maint. by Wehi.

■ NZC
Inbr: F137 (Wehi). Chocolate-brown. Genet: a, b. Origin: see NZB. Charac: High incidence of ovarian granulosa cell tumours (Bielschowsky and D'Ath, 1973). High incidence of spontaneous hydronephrosis (56% in males, 81% in females) (Warner, 1971). Has excessively high endogenous spleen colony forming activity with a marked reduction in spleen haematopoietic colony formation in isogenic transplantation experiments. In in vitro studies it has reduced immunological response to sheep red blood cells, phytohaemagglutinin and pokeweed. This may be due to a defect in the number of progenitor cells for the immune system, possibly involving only one component cell population (Herrod and Warner, 1972; Burnet, 1972a, b).

■ NZM-38-4479.
Set of 26 inbred strains, at F24-F30. All the mice originated in NZB x NZW mice, either F2 or backcrosses to NZW, in the Wadsworth Center for Laboratories and Research, Albany, NY (WCLR). The stocks came from UMC (University of Minnesota) in 1973 and had been strictly inbred at WCLR. For 12-15 months the mice were pen-bred for coat colour: chocolate brown, tan and grey. Inbreeding was started from single pairs in 1981 by CT Olsen. At F4-F5 AE Gabrielsen took over and abandoned the colour criterion for the grey mice and selected for continuation lines-to-be in which lupus nephritis deaths had already been documented. Inbreeding continued in WCLR and at All Children's Hospital (ACH), St. Petersburg, Florida. All are maintained at WCLR since 1990. Since March 1989 the colony has been maintained by UH Rudofsky and BD Evans. There are 26 strains, 24 descended from grey coated mice and one each from tan and chocolate. Many lines at F13-F18 resembled NZBxNZWFl in lifespan and pathology. One had accelerated disease, and others had delayed disease. In a few, males sickened almost as early as females. 3-4 strains had delayed or no disease. (AE Gabrielsen and UH Rudofsky, personal communication 2nd. July 1991).

■ NZO
Intermediate to low incidence of ovarian granulosa cell tumours (Bielschowsky and D'Ath, 1973). Median life-span about 460 days in males and 530 days in females. High incidence (15-20%) of malignant lymphomas of Peyer's patches and high incidence of duodenal and lung tumours (Goodall et al., 1972, 1973; Rappaport et al., 1971).

Very obese. Fat collects mainly in the abdomen, starting about 4 weeks, although divergence of growth curves is not detectable before about 2-4 months. At maturity 50-74% of body weight is fat. Animals are hyperglycaemic but not hyperinsulinaemic (Bray and York, 1971). Blood glucose levels, plasma insulin levels, body weight and glucose tolerance return to normal after implantation of pancreatic islets from normal albino mice. The genetic lesion therefore appears to be situated within the islets of Langerhans (Gates et al., 1972). Obesity is largely caused by an increase in adipose cell numbers, although cell size is slightly increased (Johnson and Hirsch, 1972). Obesity may be at least partly due to an abnormality in the cyclic AMP system which controls lipolysis in adipose tissue (Lovell-Smith and Sneyd, 1973). Obese syndrome also reviewed by Stauffacher et al. (1971).

■ NZW
Characteristics


Strain widely used as the NZB x NZW F1 hybrid (also known as the B x W hybrid), giving a model of systemic lupus erythematosus. Syndrome includes typical lupus erythematosus cells, antinuclear antibody, haemolytic anaemia, proteinuria with casts and terminal nephrosis with renal failure before 8 months (see Milich and Gershwin 1981). Incidence and severity of the disease is greater in females than males (Dubois et al., 1966).

NZX
Inbr: F90 (Wehi). Origin: see NZB. From an NZC female (F33) and an NZY male (F27), offspring b x s mated. Charac: From F13, some females have shown congenital imperforate vagina, and both sexes have a low incidence of megacolon.

NZY
Inbr: F120 (Wehi). Cinnamon piebald (?). Genet: b, s. Origin: see NZB; a piebald male appeared at F2 which was mated with a tan sister; at F4 a piebald brother-sister appeared; b x s mating with selection for piebald gene, s led to fixation of the coat pattern at F7. Charac: High incidence of mammary tumours and pituitary adenomas. Megacolon 10%, associated with the s gene (Bieschowsky and Schofield, 1962). Highly susceptible to induction of connective tissue tumours by 4-nitroquinone N-oxide (1/5) (Searle and Spencer, 1966). A subline has been developed: NZYf/Bu Inbr: 65 since fostering. Origin: NZY at F26 were fostered on NZC. Charac: Marked reduction in mammary cancer, pituitary tumour incidence unchanged.

O20
Inbr (A) 194. Albino: a,c. Origin: R.Kortweg, 1931, from Amsterdam petshop mice. Charac. Mammary tumours 0% in virgins, 5% in breeders, 13% in force-bred; believed not to carry the milk agent, but susceptible to it and can transmit it to offspring (Muhlbock and Rijssel, 1945). Leukosis 8% (Hilgers and Galesloot, 1973). Maint. by A.

OIR
Inbr. G12F90. Colour ?. Origin: Muhlbock 1959. A strain partly congenic with O20 developed by cross-intercross matings with selection for resistance to transplanted mammary tumour of O20 following outcross to DBA/LiA. Maint. by A.

OUBCr
Inbr: F10 since mutation. Origin: In Oct. 1969 a mutation occurred in NZB/Bi to creamy ventral surface; mutants b x s mated. Charac: Similar to NZB, but survival time about 100 days longer; mice develop strongly positive direct Coombs tests but do not become severely anaemic (Hall and Simpson, 1975).
OUBW
Inbr: F10 +. Origin: Varicoloured outbred mice taken to Dunedin from California by R. Ortman in 1957. At F6 a pair selected for black and white coats were mated. Charac: A small number have shown minor kidney pathology (Hall and Simpson, 1975).

OUCW Syn: NZCW.
Inbr: F45 +. Origin: As OUBW; at F6 a chocolate and white male was mated with a black and white littermate. From this pair a pair of chocolate and white siblings was selected. Charac: Average life-span: males 640, females 540 days. Average litter size 6; increase in amount of lymphoid tissue, thymic enlargement common; hybrids with NZB have enlarged abdominal lymph nodes (Hall and Simpson, 1975).

OUF
Inbr: F60 +. Origin: Random-bred fawn-coloured mice, selected for coat colour, 1953. Charac: Average survival times about 580 days for females and 700 days for males; nephritis and liver tumours (Hall and Simpson, 1975).

OUGW
Inbr: F20 +. Origin: As OUBW; a grey and white male was mated to a black and white female. In the F2 a grey and white pair was obtained. Charac: Lacks a characteristic pathology (Hall and Simpson, 1975).

OUW Syn: NZW. See NZW.

OUYW
Inbr: 20 +. Origin: Varicoloured random-bred mice from UK in 1939; a pair of ginger and white mice produced yellow and white offspring, which were inbred; dwarfism appeared at F4 (Hall and Simpson, 1975).

P/A
Inbr: 91. Origin: P. Kortweg 1934; DBA female x C57BL male, then N20 to C57BL, then b x s inbred.

P/J

Characteristics


PAA

- **PAB**
  Inbr (42) Colour: Agouti, but 91% white head spot. Origin, Maint.: see PAA.

- **PAC**
  Inbr (42) Colour: Agouti (?). Origin, Maint.: See PAA

- **PAD**
  Inbr (45) Colour: Agouti (?). Origin, Maint.: see PAA.

- **PBA**
  Inbr: F20 +. Albino. Genet: c. Origin: P. C. Bailey from a pair of pet shop mice purchased in Birmingham, Alabama, in 1958. Charac: Litter size 6.1; 6 week weight 21 g (both sexes); mean life-span 317 days in males, 268 days in females. Spontaneous lymphomas 100%, mammary tumours 74%, pulmonary adenomas 77% in animals over 1 year (Bailey et al., 1970). Development of spontaneous adenomas associated with increased serum concentration of IgG1 and IgG2 immunoglobulins (Schrohenloher and Bailey, 1972).

- **PBB**
  Inbr: F20+. Black. Genet: a. Origin: P. C. Bailey from multicoloured pet shop stock, in about 1972. Charac: Become obese on a standard diet. Maximum body weight of about 65-75g is achieved at about 12 months. Blood glucose normal, but glucose tolerance abnormal. Serum insulin elevated. No histological abnormalities of pancreatic islets seen, although mild fatty infiltration of the liver is common. Breeding performance good and life-span normal (18-22 months). Obesity may be analogous to mature onset obesity in man (Hunt et al., 1972), and equivalent in many respects to the mutants ob (obese) and A\textsuperscript{y} (yellow) and the NZO inbred strain. The diabetes in this case has a polygenic mode of inheritance (Hunt et al., 1976).

- **PE**

- **PERU-COPPOCK W4.**

- **PET**

- **PF**

- **PH/Re**
Inbr: 77. Genet: Ph/+ . Origin: Spontaneous mutation Ph (patch) in C57BL/6J. Truslove to E. S. Russell 1957, as non-inbred stock. Charac: White- spotted agouti; Ph/Ph is lethal.

- PHH
  Inbr: f95 (wE). Grey. Genet: a, In. Origin: Weir, from MacArthur outbred stock obtained from Butler 1949; b x s inbreeding with selection for high blood pH (Weir and Clark, 1955; Wolf, 1959). Charac: Blood pH 7.48 ± 0.004, blood lactic acid 3.6 ± 0.2 umol/ml, CO₂ output 5.90 ± 0.01 mg h⁻¹ g⁻¹ body weight, sex ratio 52.8 ± 1.00% males (Weir, 1962). Overall fertility low.

- PHL
  Inbr: F97 (We). Genet: a¹, b, In. Origin: Same as PHH, selected for low blood pH. Charac: Blood pH 7.43 ± 0.004, blood lactic acid 5.4 ± 0.3 umol/ml, CO₂ output 5.90 ± 0.03 mg h⁻¹ g⁻¹ body weight, sex ratio 41.8 ± 0.93% males. Overall fertility high. (Weir, 1962).

- PIC

- PL

Characteristics
Leukaemia 50% in females and 19% in males (Staats, 1976), 80-90% (Heston, 1968). Life-span intermediate in males (10/22 = 517 days) and short in females (4/22 = 448 days) in conventional conditions (Storer, 1966). High incidence of leukaemia (Albert et al., 1965). High gross tumour incidence in females (5/22) (Storer, 1966).

- PM

- PN
  Inbr 52. Albino c. Origin: Albino mice from pet shop in New Zealand in 1948. From Palmerston North Hosp. to Massey Univ. as 'NOS outbred'. To Glaxo Labs of New Zealand as 'GW outbred'. Back to Palmerston North as 'PN'. Maintained by Wigley in Med. Res Dept. and inbred starting in 1964, with selection for positive ANA (anti nuclear antibody) in first three generations. Antinuclear antibodies appear at about 5 months, and 80% are positive by 10 months. Develops lupus erythematosus with glomerular deposits of IgG, IgM and IgA (Wigley et al., 1975). Maint. by Hm, Mac.
PRO
Inbr: F61 (Brk). Genet: a, c\textsuperscript{ch}, p. Origin: E. S. Russell, 129/ReJ x C57BL/6J. Sib-mating with selection for a, c\textsuperscript{ch} and p. Mutation to Pro-1\textsuperscript{b} (proline oxidase-1) which controls activity level of proline oxidase in liver, kidney, and brain, which is about 20% of normal in this strain. 7 and 50 fold elevation of proline in blood and urine, respectively.

Charac: hyperprolinaemia and prolinuria with increased taurine excretion (Blake et al., 1974); sluggish movements; 50% generalised hair loss (Kanwar et al., 1975). Hyperprolinaemia due to a deficiency in the activity of component 1 of mitochondrial proline dehydrogenase (Blake et al., 1976), and associated with a decreased liver proline oxidase activity (Blake, 1972). High lymphocyte phyto-haemagglutinin response (8/43) (Heiniger et al., 1975).

PT
Inbr (Y) 45. Near-white: a,b,c\textsuperscript{ch},d,p,s. Origin: Seven-locus stock from Dr. Mary Lyon, Harwell in 1969. Inbred since then. Also carries se and Ty/+. Maint. by Y.

PUC

PUH
Inbr (Cam) 50. Agouti. Origin: wild mouse from Rimach valley in Peru (Peru-Harland) x CBA. Inbred by M.E. Wallace. Many mutations present in this strain. Maint. by Cam (?).

PWD

QF-5604
Inbr: 51. Genet: A\textsuperscript{w}, b, In. Charac: Selected for early sexual maturity of males. Mean age of first fertile mating 51.7 days; mean body weight at 100 days 39.6 g.

QF-5612
Inbr: 51. Genet: a. Charac: Selected for early sexual maturity of males. Mean age of first fertile mating 49.9 days; mean body weight at 100 days, 35.9 g.

QC

RAP/Ko
Inbr: 70 +. Origin: Kobozieff, 1951, from a pair of Rockland (USA) mice. Charac: Used for the study of longitudinal hemimelia and periodic hypo-trichosis; the mutation cataract appeared in this line.

RB/1
Inbr (Bg) 45. Albino; c. Origin: Swiss albino stock from dealer selected for high incidence of audiogenic seizures (Frings and Frings 1953). Susceptible to audiogenic seizures. Inbred since 1959. Maintained by Bg.

RB/2
Inbr (Bg) 55. Albino; c. Origin: as for RB/1, but selected for resistance to audiogenic seizures. Maintained by Bg.
RBA

RBB
Inbr (Dn) ? +35. Agouti. Origin: wild mice captured near Bondo, Val Bregaglia, Grisons, S.E. Switzerland by A.Gropp and H.Winking. To Davisson and Roderick 1977, to Davisson 1981. Homozygous for Robertsonian translocation Rb(1.10)10Bnr. Probably has never been crossed to a laboratory strain, but is not as wild as RBA. Maint. by Dn.

RBC

RBD
Inbr (Dn) 60. Albino: A,E tob,c. Origin: derived from crosses of Swiss mice (probably Han:NMRI, A.Gropp personal communication to Dn) with wild mice captured in the Valle di Poschiavo in S.E. Switzerland. The wild population was originally known as the 'tobacco' mouse because of their dark coat colour (Etob). They also appear in the literature as Mus poschiavius (Gropp et al, 1970). To Fls from Gropp (probably via F.Ruddle) to T.Roderick in 1970, b x s ever since. To M. Davisson in 1981. Homozygous for the Robertsonian chromosomes Rb(5.15)3Bnr, Rb(11.13)4Bnr and Rb(16.17)7Bnr. Maint. by Dn.

RBE
Inbr (Dn) 36. Albino. A,c. Origin: constructed by Davisson by crossing together two stocks originally derived from F1 hybrids between wild mice captured in the Valle di Poschiavo in SE Switzerland and Swiss laboratory mice (probably Han:NMRI, A.Gropp, personal communication to Dn) or a B6D2F1 female. Fls from A.Gropp to F.H.Ruddle to T.H.Roderick in 1970. Stocks to M.T.Davisson in 1974. Homozygous for the Robertsonian chromosomes Rb(1.3)1Bnr, Rb(4.6)2Bnr, Rb(16.12)5Bnr and Rb(11.13)6Bnr. Maint. by Dn (now extinct).

RBF
Inbr (Dn) 60. Albino c,E tob. Origin: Swiss mice (probably Han:NMRI) crossed with wild mice captured in Valle di Poschiavo in S.E. Switzerland. The wild mice originally known as 'tobacco mouse' because of the coat colour. They also appear in the literature as Mus poschiavius Fatio and more recently as Mus musculus poschiavius. Gropp to Roderick 1970 at Fl. To Davisson 1981. Homozygous for Robertsonian translocation Rb(1.3)1Bnr, Rb(8.12)5Bnr and Rb(9.14)6Bnr. Maint. by Dn.

RBI (reserved symbol)
Inbr (Dn) 13. Tobacco A,E tob. Origin: Muriel Davisson 1984 from cross between 1) two stocks of Swiss mice (NMRI ?) crossed with wild "tobacco" mice from the Valle di Poschiavo in S.E. Switzerland and 2) a stock derived from wild "Orobie."

492
(captured near Bergamo, N. Italy) mice × a 129/- female mice. Genetic background also includes a contribution from C57BL/6J, DBA/2J and AEJ/Gn strains. Strain is homozygous for Rb(1.3)1Bnr, Rb(4.6)2Bnr, Rb(5.15)3Bnr and RB(9.14)6Bnr and has the Y chromosome from wild M.m. domesticus from the Poschiavo valley. Maintained by Dn.

**RBJ** (reserved symbol)
Inbr (Dn) 14. Tobacco: A,Etob. Origin: Muriel Davisson 1984 from crosses between 1) a stock derived from an "Orobie" male captured near Bergamo, N. Italy × a 129/- female (note that the Orobie stock was developed with crosses involving C57BL/6J, DBA/2J and AEJ/Gn) and 2) an inbred strain RBD (see above). Homozygous for Rb(2.8)2Lub, Rb(5.15)3Bnr, Rb(11.13)4Bnr, Rb(16.17)7Bnr and has a Y chromosome from the Orobie wild population. Maintained by Dn.

**RC**

**RF**

**Behaviour**
High spontaneous bar pressing (1/14), high open-field activity (2/14) and high social grooming during aggressive encounters (1/14), but low tail rattling during such encounters (14/14) (Southwick and Clark, 1968).

**Life-span and spontaneous disease**
Intermediate life-span in males (15/22 = 651 days) but short in females (5/22 = 452 days) in conventional conditions. High gross tumour incidence in males (4/22) (Storer, 1966). Necrotising arteritis involving the aorta, its major branches and other arteries and arterioles seen in 10-20% of aged mice. Disease may involve an autoimmune mechanism (Upton et al., 1967). Mean life-span 619±7 days. Leukaemia 66%, glomerulosclerosis 63% and reticulum cell sarcoma 52% (Yuhas and Clapp, 1972). Spontaneous glomerular hyalinisation and glomerrnlosclerosis develops at 8-20 months (Russell and Meier, 1966). Leukaemia 46% (Myers et al., 1970)

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Immunology
Resistant to experimental allergic encephalomyelitis (cf. 7/18) (Levine and Sowinski, 1973). Erythrocytes have a low agglutinability (cf. 11/25) (Rubinstein et al., 1974).

Infection
Encephalomyocarditis virus causes diabetes mellitus (cf. 7/14) (Boucher et al., 1975).

Reproduction
High post-implantation loss of embryos (2/8) (Leonard et al., 1971).

RFM
Inbr 120 +. Albino a,c. Origin: Furth, to Oak Ridge 1949. Strain has been found to be segregating for $H-2^f$ and $H-2^k$ and substrains also differ in other respects, so genetic contamination of some substrains must be suspected. Maint. by N.

Characteristics
Leukaemia low (5%), but highly susceptible to induction of both myeloid and lymphoid leukaemia by X-irradiation and chemical carcinogens (Walburg and Cosgrove, 1971). Spalding and Brooks (1971) found that the strain was segregating for $H2^k$ and $H-2^f$. Two substrains were developed with a mean life-span of 655 ± 8 days in the $H-2^k$ substrain and 604 ± 10 days in the $H-2^f$ substrain, implying that the $H-2$ locus may affect survival. The substrains also differed in radiation resistance and spontaneous activity. The presence of genetic variation within this strain may imply that some substrains have become genetically contaminated by a non-strain mating. Develops a spontaneous myelogenous leukaemia that is transplantable into non-leukaemic mice of the same strain (Huseini et al., 1976). Reticulum cell sarcomas 57% in males and 70% in females. Lung tumours 24% in males and 19% in females, and non-neoplastic moderate to severe glomerular lesions in more than 50% of animals (Zurcher et al., 1976).

RHJ
Inbr (Le) 72. Albino: a,b,c. Origin: Mutation to rhino-J ($hr^{th-h}$) in a Carworth Farms stock received by E.P. Nagler, then back to Carworth, then to M.M. Dickie 1951. To Lane 1960. Crosses to BALB/cHu 6 times, b x s to F16, then outcrossed to albino stock followed by b x s. Homozygotes begin to lose hair about 10 days. Skin becomes thickened and more wrinkled than $hr/hr$ animals, and females do not nurse young. Maint. by Le.

RIII
Inbr 80+. Albino: A,c. Origin: Dobrovolskaia-Zavadskaia, Inst. du Radium, Paris 1928, then see below. High mammary tumour incidence in unfostered substrains. The following substrains are recognised:

RIII.
Origin as above.
RIII/An. Dobrovolskaia-Zavadskaia to Andervont.

RIIIS.
In 1967 both RIII and RIII/An maintained at The Jackson Laboratory failed to produce viable young. RIII/2J was developed from a cross between the two substrains. Name later changed to RIIIS.

RIII/SeA.
From Severi (Perugia) to Muhlbock (Amsterdam) 1964. Differs from the other substrains at the Hbb and Mup loci

**Behaviour**
Short latency to emerge from home cage (2/7), high rearing in Y-maze (2/7), high hole-in-the-wall entries (1/7), high exploration in Y-maze (2/7), high number of stairs climbed (2/7) and low urination score (7/7) (McClearn *et al.*, 1970).

**Life-span and spontaneous disease**
Long life-span in conventional conditions (19/22 = 685 days in males, 16/22 = 655 days in females) (Storer, 1966). High incidence (88%) of mammary tumours in breeding females (Heston, 1963), but a low proportion are of the acinar type (7/7) (Tengbergen, 1970). Ovarian tumours 60% in breeding females, 50% in virgin females (Murphy, 1966). Mammary tumours 96% at 9 months (Schlom *et al.*, 1973), 70% at 12 months (Seman and Dmochowski, 1973). Has been known to loose the mammary tumour virus spontaneously (Andervont and Dunn, 1962).

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**Infection**
RIIS carries no detectable endogenous ecotropic MuLV DNA sequences (Jenkins *et al* 1982)

**Reproduction**
Poor reproductive performance (7/8), litter size 6.1 ± 0.2 and sterility 31% (Nagasawa *et al.*, 1973).

- **RNC**
  Inbr 50+. Colour ?. Origin: R.C.Roberts, Edinburgh 1968. Three pairs carrying *Re* (Rex), *Tr* (trembler) and *nu* (nude). *Tr* was lost. Recombinant *Re,nu/+,*+ were mated b x s. Homozygous *nu/nu* (nude) mice are athymic and have defective cell-mediated immune response.

- **ROP**
RR

RSV
Inbr (Le) 66. Agouti: +. Origin: Carter's Stock 1 (Edinburgh linkage testing stock El) to Woodworth 1950. Outcrossed to C57BL/6, CBA and C3H. To Lane 1967. b x s thereafter. Carries Re/Re (Rex) Sd/+ (Danforth's short-tail) and Va/+ (varitint-waddler). Maint. by Le.

RW
Inbr (W) 82. Albino: a,b,c,D. Origin: Albino mice from the University of Wroclaw, Poland. Inbred since 1960. No known crosses with other strains. Mammary tumours 41% in virgin and 74% in breeding females at 300-400 days. Haematopoetic tumours 41-49% at same age. Maint. by W.

S

SB
Inbr (Le) 101. Pale grey. sa,bg,Aw. Origin: mutations to sa and bg, possibly radiation-induced, occurred independently in stocks used for mutation rate studies at Oak Ridge. RI to Le 1961, then sib-mated.

Charac: Develops progressive infectious pneumonitis associated with the beige gene. Also has a high incidence of lymphadenopathy, including reticulum cell neoplasms and atypical lympho-proliferative lesions. Beige mice have giant lysosomal granules in the leukocytes and pigment dilution closely analogous to the Chediak-Higashi syndrome in man and similar disorders in mink and cattle. These mice appear to be a suitable model for the study of the increased susceptibility to infection seen with this syndrome (Lane and Murphy, 1972).

SC

SD

SEA
SEC Pale brown: \(a,b,c^{ch}\). Origin: Green prior to 1946 from a cross between NB and BALB/c. Carries \(se\) (short-ear) gene causing short ears and skeletal abnormalities. Substrains as follows:

**SEC/R1**
- Inbr (R1) 100 +. Colour ?: \(a,b,c^{ch},d,se\). Origin: E.L.Green 1948 to R1 at F20–F21. Maint. by J.

**SEC/1Gn**
- Inbr (Le) 149. Gets multiple lung cysts, especially in \(se/se\).

**SEC/1Re**
- Inbr (J) 138. From SEC/1Gn, but short-ear eliminated by E.S.Russell.


**SF/Cam**

**SEN**
- Inbr (Or) 20. Albino +. Origin: From outbred "SENCAR" mice produced by Boutwell (1964) using Rockland-derived mice from A.Sutter, Springfield Mo. Selected for skin tumors following initiation by DMBA and promotion with croton oil. Later crossed with outbred CD-1 mice. To McArdle Lab. for Cancer Research, to Oak Ridge. Inbred by Sloga in 1979 with further selection to F9 for skin tumors following treatment with DMBA and TPA. Maint. by Or.

**SF**
- Inbr (Dn) 42+32. Agouti +. Origin: wild mice trapped in coal mine and sib-mated by M.Barnawell (Berkeley CA) as 'Corte Madera' to Cambridge 1959. Maint. by Dn.

**SF-5613**
- Inbr: 45. Genet: a. Charac: Selected for late sexual maturity of males. Mean age of first fertile mating formerly 58.8 days, now 52 days; mean body weight at 100 days, 28.9 g.

**SF-5621**
- Inbr: 36. Genet: A, b. Charac: Selected for late sexual maturity in males. Mean age of first fertile mating. 58.2 days; mean weight body at 100 days, 32.3 g.

**SHI**
- Inbr (Le) 78. Agouti chinchilla: \(c^{ch}\). Origin: Snell's FS stock to M.C.Green. Outcrossed to C57BL/10Gn then b x s. To Lane 1975. Carries \(sh-1\) (shaker-1) gene. Homozygotes show circling, head tossing, deafness and hyperactivity. Maint. by Le.

**SHM/2Gn**
- Inbr: 35. Genet: + /shm. Origin: \(shm\) (shambling) mutation arose in random-bred stock in March 1960. This strain descended from (SHM/Gn x C57BL/6J) x C3HeB/FeJ (SHM/Gn discontinued at F35 in 1970). Charac: Homozygotes have
abnormal gait, small body size, phospholipidosis, sterility, low viability (Green, 1967).

**SHN**
Inbr: F51 (Mei). Genet: c, A, b. Origin: Swiss albino mice from the City of Hope Medical Centre, California. Inbred as SWM/Ms by Natl. Inst. Genet. Misima. Selected for high early mammary tumour incidence with full-sib mating starting in 1964, and F20 in 1972. Have mammary tumour virus locus Mtv-4 which is genetically transmitted rather than being passed through the milk (Imai et al. 1983). Tumour incidence 90-100% in breeders and virgins at 6.6 months. Litter size 7.6, weaning rate 89% (Nagasawa et al., 1976a, b). See also SLN.

**SHR**
Inbr (Dn) 59. Colour ?. Origin: E.L.Green from shm (shambling) mutation which arose in outbred stock in 1960. Crossed with C57BL/6J, C3HeB/FeJ and finally to a Re (rex) stock. Strains SHM and SHM/2 from same cross discontinued in 1970 and 1977. Homozygotes have abnormal gait, small body size, are infertile, and have phospholipid-like material deposited in the central nervous system. Maint. by Dn.

**SIIT**

**SIM** [Sandos Inbred Mice]
Inbr: F 20 + (?). Albino. Origin: A strain of Swiss mice highly susceptible to Friend virus. Congenic line SIM.R has been developed by backcrossing resistance gene from C57BL/6 (Ware and Axelrad, 1972). (NOTE: This is not the SIMPSON strain.)

**SJL**
Inbr: F104 (J). Albino. Genet: c, p, rd. Origin: Swiss Webster outbred stock from three sources that were brought to The Jackson Laboratory between 1938 and 1943, and pen-bred until 1955, when sib-mating was started. Although the strain has been developed relatively recently, it has rapidly become widely used owing to the high incidence of reticulum cell sarcomas resembling Hodgkin's disease. General biological data on the strain have been reviewed by Crispens (1973).

**Behaviour**
High spontaneous fighting (Page and Glenner, 1972). Severe fighting among males housed together, beginning at about 8 weeks. Most males will be killed by 4-5 months unless caged separately (Crispens, 1973).

**Life-span and spontaneous disease**
Short life-span in conventional conditions (8/22 = 472 days in males, 3/22 = 395 days in females). High gross tumour incidence (4/22 females, 6/22 males) (Storer, 1966). Reticulum cell sarcomas appear in about 90% of animals at an average age of about 13 months (Murphy, 1963; Crispens, 1973; Fujinaga et al., 1970). These first appear in the Peyer's patches and mesenteric lymph nodes and later in the spleen, liver, thymus and other lymph nodes (Crispens, 1973). Most of the tumours are pleomorphic or mixed-cell types commonly called type-B reticulum cell neoplasms by Dunn, but a few are type-A histiocytomas. The unusual feature of the SJL reticulum cell tumours is their regular and early appearance, with the preneoplastic lesion detectable as early as 22 days (Potter, 1972). Tumour development as well as autoimmunity may result from an effective amplification of the immune response (Owens and Bonavida, 1976). Leukaemia 83% (Myers et al., 1970). High incidence of spontaneous amyloidosis, possibly associated with

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Infection
Encephalomyocarditis virus causes diabetes mellitus (cf. 7/14) (Boucher et al., 1975). High susceptibility to develop leukaemia on infection with Friend virus (cf. 5/11) (Dietz and Rich, 1972). Resistant to measles virus (1/6) (Rager-Zisman et al., 1976), develop flaccid paralysis and survivors develop a distinct neurological disorder associated with marked mononuclear cell infiltration and active demyelination in spinal cord after intracerebral inoculation with Thielier's encephalomyelitis virus. Incubation period may be 2-3 months (Lipton and Dal Canto, 1976).
SK
Inbr: F?+38 (Dn). Origin: From three mice trapped on SkoKholm Island off Pembrokeshire in 1962 by R. J. Berry; inbred by M. Wallace (Wallace, 1970). Then to Rk, Dn, and Ei. Charac: fairly large litters, the size of which dropped little on inbreeding.

SL
Inbr(A) ?+90. Albino: A,B,c. Origin: derived from SMA as a high-leukaemia strain by K. Tsuchikawa in Misima. Currently, there are four distinct substrains (Ni-Eco-, Ni-Eco+, Kh, QDJ) which differ in leukaemia incidence, ecotropic virus expression, as well as for a number of genetic markers (Hiai et al 1987). In Misima leukaemia incidence is low (Tajima, 1968).

SLN
Inbr (Mei) 45. Colour ?. Origin: see SHN. This is the high-late mammary tumour line. Mammary tumour incidence is about 50-60% in breeders, 9-10% in virgins at 10 months. Litter size 5.8, weaning rate 60%. Maint. by Mei.

SM/J
Inbr (J) 112. White-bellied agouti or black A*w/a or a/a. Origin: MacArthur, 1939 by crossing seven stocks including DBA and selecting for small body size. To Runner 1948, who began b x s mating. Small body size at birth and weaning, but this relatively small size tends to disappear as the animals mature. Very low tumour incidence. Carries a number of relatively rare polymorphic alleles. Maint. by A,J.

Characteristics

SM/JH
Inbr ? +16 (H). White-bellied agouti substrain A*w/A*w. Chai to Bateman (Edinburgh) 1958, to Dickinson 1961, to Lush in early 1970's, to Peters (Harwell), to Nash, to Peters again in 1979. Identical wth SM/J at seventeen loci, but differs in being Gpi-1b while SM/J is Gpi-1a. This is believed to be due to residual heterozygosity or mutation rather than genetic contamination (Peters and Lyon 1986).

SRH
Inbr: F63 (Crusio). Dilute brown: a, b, d. Origin: van Abeelen, C57BL/6J x DBA/2J, backcrossed to DBA/2 five generations, followed by b x s (from 1966) with selection for behavioural traits. Charac: High frequency of exploratory rearing responses and high locomotor activity (Abeelen, 1975). Maint. Crusio, Nmg.

SRL
Inbreeding, genetics and Origin: As SRH. Charac: Differs from SRH in developmental-age dependent behavioural characters, and in hippocampal anatomy. Low frequency of exploratory rearing and low locomotor activity.
SS

SSIN
Inbr (Utsp) 22+. Albino. Origin: 1970's, from outbred SENCAR (sensitive to carcinogens) selected for high skin tumour number following challenge for 10 generations with a topical application of dimethylbenz(a)anthracene followed by repetitive application of the tumour promoter 12-O-tetradecanoylphorbol-13-acetate (TPA). Inbred in 1986. Hardy animals with low spontaneous tumour incidence; useful in chemical carcinogenesis studies because of extreme sensitivity to TPA (Fischer et al 1987)

SSL
Inbr (Le) 60. White spotted, heavy spotting or black-eyed white depending on genotype: a, s/s or s/s¹, or s¹/s¹. Origin: Developed by Le. s¹ arose in the F2 from a cross of C3H/HeSn x C57BL/6J. b x s to F18. In 1968 outcrossed to a/a, s/s stock (F19) and maintained by sib mating of s/s¹ to present. The s gene came from a multiple recessive stock of Holman to Runner before 1955. Outcrossed to C57BL/6J and b x s to F19. In 1968 crossed to a/a hr/+s¹ to make s/s¹ s¹/s¹ get megacolon and generally die though a few live to breed. Maint. by Le.

ST
Inbr.(J) 143. Albino a,b,c. Origin: Englebreth-Holm from outbred Danish white mice in about 1940. To Heston in 1947 at F23. Two major substrains are known which differ at the H-2 locus. These were separated after more than eight generations of sib-mating.

ST/a
See above. This is the H-2ᵇ substrain which is not so widely used.

ST/b
See above. H-2ᵏ substrain. Maint. by J,N.

Characteristics
Life-span in conventional conditions short (5/22 = 433 days in males, 9/22 = 511 days in females), but low gross tumour incidence (21/22 in females, 19/22 in males) (Storer, 1966).


STAR

STR
Inbr (N) 145. Brown a,b. Origin: Strong from a stock treated with methylcholanthrene between generations F4 and F27. Susceptible to periodontal disease. Charac: Susceptible to periodontal disease (Baer et al., 1961); polydipsic.

STR/1
Inbr (N) 138. Piebald brown. a,b,s. Origin: piebald mutation in STR at F29 in 1961. Develops osteoarthropathy of the knee joints, and obstructive uropathy in males before 16 months. Maint. by N.

Charac: Osteoarthropathy of the knee joints (Russell and Meier, 1966). The condition has been considered in detail by Walton 1977a, b, 1979 a, b, c. By around 12 months of age 60% of males and 30% of females have osteoarthrosis as assessed radiologically. Medial patella dislocation is a common feature of the condition, which can be prevented by surgical stabilization of the patella. Eventually, the conditions becomes so severe as to impair joint movement.

High level of plasma phospholipids, cholesterol, cholesterol ester and total lipids, but no excessive obesity (Yamamoto et al., 1963). Sokoloff et al (1962) considered that the mice become obese, but Walton (1979a) found that males were not obese when compared with CBA, though females were, and did not consider that obesity was associated with the osteoarthropathy. Polydipsia (Bernstein, 1966) and obstructive uropathy constantly seen at less than 16 months (Russell and Meier, 1966). Urine has low osmolality (7/7) (Silverstein, 1961).

STS

Charac.
In contrast with five other strains, STS is resistant to mammary tumour induction by hypophysial isografts (van der Gugten et al 1985). No mammary tumours but lung tumours in both sexes; closely related to LIS/A and LTS/A. Leukosis 8% (Hilgers and Galesloot, 1973). High preference for sweet tasting substances (saccharin, sucrose, dulcin and acesulfame, averaged) (3/26) (Lush 1988).

STU

STX
Inbr (Le) 59. Black: E^oo. Origin: sombre (E^oo) mutation in C3H held by N.Bateman before 1961, to M.Foster, to M.C.Green 1966. Crossed with Tw (twirler) from M.Lyon in 1967, then b x s. Also carries Xr' (extra-toes). Has been well characterised at many polymorphic loci and is useful for linkage testing. Maint. by Le.

SUMS
autosomal recessive unnamed mutation causing hydrocephalus in homozygotes which is lethal by 2–6 weeks of age. It is probably not obstructive hydrocephalus. Maintained by T.Richards, Southampton General Hospital, U.K.

- Swiss. Various lines from mainly commercial sources, inbred in different laboratories, as SWJ/Mk, SWM/Ms, etc. Characteristics may vary widely between strains.

- SWJ

- SWM/Ms

- SWR

**Behaviour**

**Life-span and spontaneous disease**

**Normal physiology and biochemistry**

**Drugs**
Immunology

Infection
Susceptible to herpes simplex virus (10/11) (Lopez, 1975). Susceptible to LCM virus infection (5/5) (Oldstone and Dixon, 1968). Encephalomyocarditis virus causes diabetes mellitus (cf. 7/14) (Boucher et al., 1975). In contrast to ten other strains, it does not carry type I and II endogenous type C viruses (cf. NZB) (Stephenson et al., 1975). Carries no detectable endogenous ecotropic MuLV DNA sequences (Jenkins et al. 1982), Rapid immunological expulsion of Trichinella spiralis worms (Wakelin and Donachie 1980).

Miscellaneous

■ SWV

Charac: Females over 8 months have a hereditary polydipsia-polyuria defect with a severe increase in water turnover and with hypotonic urine that contains no glucose, blood or protein: i.e. nephrogenic diabetes insipidus. They are vasopressin-resistant. Females also have a progressive kidney defect which, although it resembles nephronophthisis in some aspects and hypokalaemia in others, is unique. Males have a milder form of the defect at an older age and show no signs of histopathology (Virgo and Miller, 1974). Resistant to acetazolamide-induced teratogenesis (Hackman and Hurley 1983).

■ SWXJ-
Inbr (28-32). Set of 14 recombinant inbred strains developed by Beamer from a cross of SWR x SJL (MNL 75:34,1986). Maint. by Bm.

■ SZA
Inbr 16-26 (Wim). albino a,B,c,D. Origin: R and C Wimer, 1981 from a cross between NZB/BINJ and RF/J. Spontaneous seizures appeared at F6, and the strain has been maintained with selection for seizures. Exhibit apparently spontaneous non-fatal convulsions from the age of 8-12 weeks, possibly triggered by olfactory stimuli. No reduction in fertility or viability. Maintained by Wim.

■ SZB
Inbr 16-26. As for SZA, but separated at F13.

■ SZC
Inbr 16-26. As for SZA, but separated from SZA at F9, and has lost the spontaneous convulsions.
TA1
Inbr. 90. Albino: a,b,c. Origin: Outbred mice from Tianjin (China) in June 1956. Inbred since then. Well characterised at polymorphic loci. Maint. by A.

TA2
Inbr. 66. Albino: a,B,c,d. Origin: Outbred 'Kun Ming' mice from Bioproducts Institute, Peking in 1962. Inbred since then. Mammary tumours 81% in breeding females, 41% in virgins. Well characterised at polymorphic loci. Maint. by A.

TB

TF
Inbr. (Le) 77. Black: a. Origin: M.C.Green from T tf/tf stock received from M.F.Lyon in 1961, then outcrossed to C57BL/10Gn to obtain +tf chromosome in 1962, followed by b x s. To Lane 1975. Has good chromosome 17 markers. Maint. by Le.

TFH

TFM
Inbr F24. Agouti: Ta (carried in balance with Tfm). Origin: Carol M Wilson 1979 from a 1971 cross between Tfm/+ females from S. Ohno and Ta/Y males from J. Tfm is carried in balance with Ta to facilitate the identification of female carriers from males which are phenotypically females. There may be some residual heterozygosity in the region of the X chromosome that lies between these two loci. Ren-2n, TfmH , Thy-1b, Ly-2b, B2mb, I-Ad. Maintained by Wl.

TH

TKDU
Inbr (Dn) 69. Grey: a,d. Also carries du (duky) and tk (tail kinks). Origin: du from C.Keeler to G.D.Snell 1948, to M.C.Green 1956, to Eicher 1972, to Davisson 1980. Cross to DBA then bxs to F10, crossed to BALB/c then bxs to F4. Crossed to DBA/1 then bxs to F2. Crossed to C57BL/10 then bxs to F2. Crossed to BALB/c-tk (from Gruneberg 1961) and maintained as balanced stock. tk arose in BALB/c (probably An substrain) in 1953 at the Chester Beatty R.Inst. Maint. by Dn.

TL

TM

Charac: Lung tumours 48%, fore-stomach papillomas 27%, pyloric tumours 13%, brain nerve cell tumours 11%. Low mammary cancer. A low incidence of heart tumours has also been observed (Szepsenwol and Boschetti, 1975).

TP
Inbr (RI) 68+. Black or slate-grey. a,tp/+ . Origin: Jackson Laboratory. Taupe mutation in C57BL/10J in 1948. Six generations of backcross-intercross to C57BL/10, then b x s. Homozygous tp mice viable, but females have difficulties during gestation and can not rear young due to abnormal nipples. Maint. by RI.

TPS

TR
Inbr (Dn) 81. Irregular patches of full-coloured and very lightly coloured fur: Mo^o. Origin: Dickie 1952 from tortoise (Mo^o) mutation in non-inbred obese stock. Maintained by matings of Mo^o females with normal males. Line has been maintained by trio matings for 21 years as offspring do not survive as well from pair matings. Mottled females with much white in coat do not usually mature. Darker ones survive well. Maint. by Dn.

TRE/Ko
Inbr: 64+. Origin: Kobozieff 1937, from waltzing mice of unknown origin. Charac: Malformation of one or both ears in low incidence.

TS/Wf

TSI

TSJ
Inbr (Le) 64. Cinnamon. b. Origin: mutation to Ts (tail-short) arose at NCI in 1946. From Morgan to G.D.Snell 1950. Crossed to C57BL/6, C57BR/cd and BALB/cSn then b x s. Homozygous lethal before 6 days of gestation. Heterozygotes have short kinked tails with numerous other skeletal abnormalities, and a transient anaemia. Maint. by Le.

TT6
Inbr (Le) 54. Black: a,s,ln,fz/ln,fz,v/+ . Origin: 'TR' stock from M.F.Lyon to M.C.Green in 1961, to Le 1970. One cross to jittery-grizzled stock, b x s to F7, one cross to B6CBAF1-A+1/A in 1972, then b x s. Carries T (brachyury), t6, and tf (tufted). Useful for study of t-alleles. Maint. by Le.

V
Inbr (Le) 49. Grey, and grey-piebald (?): a,ln,s. Origin: G.D.Snell from a stock carrying v (waltzer), s (piebald) and ln (leaden) from Ludwin in 1947. Crossed to C57BL/10, then to fz (fuzzy) stock in 1960 and non-sib mated. To Lane 1969, then b x s. Homozygous waltzer waltzer females are poor mothers. Maint. by Le.

VC

VL

VM
Inbr. 81 (Dk). Albino. Genet. $a, b, c, d, H^{-2}b$, $Mls^a$. Origin: Inbred as '5M' from Moredun Inst. stock (Dickinson and Mackay, 1964), and name later changed to conform with nomenclature rules (Dickinson et al., 1968).

Charac: Long incubation of ME 7 scrapie agent and short incubation of 22A compared with most other strains (Dickinson and Meikle, 1971). Spontaneous astrocytomas occur at 1.5% incidence. Tumours are largely confined to white matter areas, and have not been seen in sixteen other strains. Also has high incidence (1/6) of developmental defects, including cleft palate, subcutaneous blebs, facial and tail defects and cranial meningocoele (Fraser, 1971), including spina bifida (Dickinson, 1977).

VP/WF
Inbr: 30. Genet: $A^{vy/a}, A^v/b, P/p, DSe/dse$. Origin: Female $A^{vy/a} x$ male $A^v/b$. Sib-mating with forced heterozygosis at five loci.

VY
Inbr (Nctr) 74. Variable yellow to agouti and black: $A^{vy/a}$, $a/a$. Origin: The $A^{vy}$ mutation occurred in C3H/HeJ in 1960. Crossed with a C57BL/6J male, then backcrossed to C57BL/6J. One N3 male and two N1 females from M.M.Dickie, Jackson Lab. to Inst. Cancer Research in 1962. These were mated and the offspring sib mated. Maintained by $A^{vy/a}$, $a/a$ matings since then. To the Nctr in 1972 at F35. Caesarian derived and fostered on C3Hf in 1977 at F47. Pseudoagouti phenotype about 10% of $A^{vy/a}$ mice. Spontaneous hepatocellular tumours in 24% of male yellow mice but only 13% of black mice. Phenotypically yellow mice differ from agouti ones in a range of biological properties (Wolff et al. 1990). Maint. by Nctr.

WB
Charac: $WW$ homozygotes are anaemic, sterile, lack pigmentation in coat; heterozygotes have normal blood picture and fertility, but white ventral spotting; anaemics die at approximately 11 days. Many other anaemia-producing mutant alleles are maintained congenic with this strain. Resistant to lethal effects of ozone (19/22) (Goldstein et al., 1973). High lymphocyte phytohaemagglutinin response (4/43) (Heiniger et al., 1975).

WC
Inbr (J) N58F37. Colour grey, white or black depending on genotype: $a, S_{1}$. Origin: $S_{1}$ (steel) mutation backcrossed to strain WB-+/+. Phenotype of $S_{1}S_{1}$ homozygotes rather similar to $WW$ homozygotes, but defect is due to abnormal environment for stem-cell development. Maint. by J.

WH
Inbr: 90. Genet and Origin: As WB. Charac: As WB, except that anaemics die at approximately 5 days.

WHT
WK
Inbr: 74. Genet and Origin: As WB. Charac: As WB, except that anaemics die at approximately 10 days. The mutant $d_{y^{2j}}$ arose in this strain.

WLHR/Le
Inbr: F92 (Le). chocolate. Genenet: $a$, $b$, $w_{l}^+/+$. $hr$. Origin: Mutation to $w_{l}$ (wabbler-lethal) occurred in pirouette stock in 1948. Crossed to $hr$ from Hummel in 1947, Dickie to Lane 1958 and inbred as balanced stock. Charac: Homozygous $w_{l}/w_{l}$ die before weaning.

WLL

WN
Inbr: F60 (Nga). Colour depends on genotype. Genet: $a$, $B$, $C$, $D$, $S$, $w_{n}$. Origin: New mutant at $W$ locus. $w_{n}/w_{n}$ die at 16-18 days gestation with anaemic syndrome, seldom survive birth, but die in a few days. Heterozygote has normal blood, viable and fertile. Amount of white spotting is greater than in $W_{v}/+$. 

WR
Inbr (Y) 36. Black spotted: $a_{w_{v}}/+.$. Origin: Developed by selection for dilution of pigmentation of $a/a$, $W_{v}+/+$ mice and a high incidence of somatic reversion of $W_{v}$ to + in a cross of 129 and 129xC57BL/6-$W_{v}/+.$. Inbred since 1975. Anaemic $W_{v}/W_{v}$ die at birth. $W_{v}/+$ mice have light coat colour, 25% with black spots. Maint. by Y.

X

Characteristics
No spontaneous tumours of any kind. They are resistant to the induction of tumours by urethane, producing only 3% tumours, at dose levels that would induce 80-90% in other strains. Also resistant to tumour induction by X-rays. Combined X-ray and urethane produces only 6% tumours. The mice do not produce murine leukaemia virus antigens in their milk, and are resistant to polyoma virus, Friend leukaemia virus and FBJ osteosarcoma virus. They also have high immune response against sheep erythrocytes, pronounced splenic phagocytic activity, high levels of antibody to mammary tumour virus and a tendency to spontaneous amyloidosis. The low tumour incidence is attributed to high immune competence and absence of an apparent oncogenic virus as revealed by electron microscopy (Goldfeder et al., 1966; Goldfeder, 1972). Resistance to carcinogens does not appear to be due to differences in biochemical response to such chemicals (Grantham et al., 1976).

XLII

XVII

Charac:
No mammary tumours or leukaemia; pulmonary adenomas 19.5% after 13 months; mammary tumours 1.3%, leukaemia females 0.4%, males 1.6%; lung tumour females 5.1%, males 8.8%; sensitive to Graffi leukaemia agent (Krischke and Graffi, 1962); blood catalase has low specific activity (7/7) (Magdon, 1962); strong reactivity against specific antigens of carcinogen-induced sarcomas (Pasternak, 1963); very susceptible to lung oncogenesis by chemical agents.

**YBR**

**YS**
Inbr (Nctr) 84. Mottled yellow, pseudoagouti, black, piebald, depending on genotype. $A^v/a$, or $a/a, s$. Origin: Chase to Inst. of Cancer Research 1959 at F38-39. In 1962 outcrossed to an N3 male from a (C3H/HeJxC57BL/6)xC57BL/6 cross. $A^v/a$ mice were backcrossed to the YS strain to N35. To Nctr in 1972, then caesarian derived and fostered on C3Hf. Strain has impaired glucose tolerance on which the impairment of glucose tolerance due to obesity of $A^v/a$ mice is superimposed. High level of serum-glutamyltranspeptidase activity. Pseudoagouti phenotype in about 16% of $A^v/a$ mice. Hepatoma incidence in males at 12-16 months: 11% in $A^v/a$, 3% in $a/a$. Large testes weight (1/8) (Shire and Bartke, 1972). Maint. by Nctr.

**YT**
Inbr (Y) 45. Near-white. $a, b, c^h, p, d, ln$. Origin: From crosses involving C57L, DBA/2, C57BL-go, 129/J. Inbred since 1967. Carries multiple recessive genes including go (angora), producing long-hair. Used for mutagenesis studies. Maint. by Y.

**YX**

**101**

**102**

**129**
Inbr and colour depends on substrain (see below). Origin: Dunn 1928 from crosses of coat colour stocks from English fanciers and a chinchilla stock from Castle. This strain has a common origin with strain 101. It is best known for the high incidence of spontaneous testicular teratomas, though the incidence differs between substrains. A number of major substrains, which trace back to the Jackson Laboratory in 1948 are recognised as follows:

129/Re
129/RrJ
Inbr (J) 97. Pale yellow, or albino. A^, c^h (or c), p. Origin: Jackson Laboratory 1948. Maint. by J.

129/Sv-ter/+ Inbr (Sv) N8 F49. Agouti with light belly: A^, c^h, p^+. Also carries a gene ter causing a high incidence of testicular teratomas. Origin: A substrain to determine the effect of the W gene on incidence of testicular teratomas. The W gene was backcrossed repeatedly to 129, and at generation N8 a female produced 38 male offspring of which 8 had testicular teratomas. All subsequent members derived from that mating. The W gene has been eliminated. Incidence of testicular teratomas now 30% (Stevens, 1973). Maint. by J.

Behaviour

Life-span and spontaneous disease
Long life-span in conventional conditions (18/22 = 679 days in males, 15/22 = 648 days in females) (Storer, 1966). Long life-span in SPF fostered conditions (16/17 = 699 days in males, 11/17 = 666 days in females) (Festing and Blackmore, 1971). Low overall tumour incidence (7% in males, 21% in females), including lymphomas 2% in males and 7% in females, soft tissue sarcomas 2% in males and 1% in females and benign tumours 2% in males and 3% in females (Smith et al., 1973). Lung tumours 4-46% (Festing and Blackmore, 1971). Testicular teratomas about 1% in most substrains, but 30% in the terSv substrain (Stevens, 1973). Congenital malformations about 4% in RrSvKt-jt substrain (Kalter, 1968). High incidence of urinary calculi (Russell and Meier, 1966).

Normal physiology and biochemistry

Anatomy
Large brain/body weight ratio (3/20) (Roderick et al., 1973). Small spinal cord (21/25) (Roderick et al., 1973). Small forebrain volume (8/9) and neocortex (8/9) (Wimer et al., 1969). A large proportion of 129/Ola mice have major shunts between the hepatic portal system and the vena cava, allowing the passage of microspheres up to 50µm in diameter. These shunts are associated with resistance to Schistosoma japonicum cercariae (Coulson and Wilson 1989).

Drugs
Sensitive uterine response to oestrogens (1/5) (Chai and Dickie, 1966; Drasher, 1955).

**Immunology**

High lymphocyte phytohaemagglutinin response (12/43) (Heiniger et al., 1975). Responder to synthetic polypeptide (Glu^{37}, Lys^{36}, Ala^{5}) (cf. 3/7) (Pinchuck and Maurer, 1965). Erythrocytes have a high agglutin ability (cf. 14/25) (Rubinstein et al., 1974). High responder to Dextran (cf. 4/10) (Blomberg et al., 1972).

**Infection**

Carries no detectable endogenous ecotropic MuLV DNA sequences (Jenkins et al., 1982).

**Reproduction**

Poor breeding performance (19/22), colony output 0.8 young/female/wk, litter size at weaning 4.5 (19/22) (Festing, 1976a).

**Miscellaneous**

Recommended host for transplantable tumour haemangioendothelioma BW6473 (Kaliss, 1972).

201


615

Inbr 64. Chocolate: a,b. Origin ?: Tianjin, China. Well characterised at polymorphic loci.
REFERENCES


Moloney, J. B.), National Cancer Institute Monograph 22, pp. 619-629


Belyaev, D. K., Gruntenko, E. V. and Videlets, I. YU. (1970). Genetic differentiation of the thymus in mice of different strains with respect to malignant growth communication. II. Differences in the weight of the thymus in various strains of mice. Sov. Genet., 6, 47 (transl. of Genetika)


Birnbaum, A. (1972). The random phenotype concept, with applications. *Genetics*, 72,739


Brooke, M. S. (1965). Natural haemogglutinins in mice: their occurrence and properties. *Immunology*, 8, 375


Committee (The) on Standardised Nomenclature for Inbred Strains of Mice (1952). Standardised nomenclature for inbred strains of mice. Cancer Res., 12, 602

Committee on Care and Use of Spontaneously Hypertensive (SHR) Rats (1976). Spontaneously hypertensive (SHR) rats: guidelines for breeding, care and use. ILAR News, 19, G1

Committee on Standardised Genetic Nomenclature for Mice (1963). A revision of the standardised genetic nomenclature for mice. J. Hered., 54, 159


Committee on Standardised Nomenclature of Mice (1972). Standard karyotype of the mouse Mus musculus. J. Hered., 63,69


Daniel, W. L. (1976). Genetics of murine liver and kidney arylsulfatase B. Genetics, 82, 477


De Maeyer, E., Jullien, P., De Maeyer-Guignard, J. and Demant, P. (1975). Effect of mouse genotype on interferon production. II. Distribution of If-1 alleles among inbred strains and transfer of phenotype by grafting bone marrow cells. *Immunogenetics*, 2, 151


Dickinson, A. G. and Mackay, J. M. K. (1964). Genetical control of
the incubation period in mice of the neurological disease, scrapie. *Heredity*, 19, 279


Eckner, R. J. (1973). Helper-dependent properties of Friend spleen...
focus-forming virus: effect of Fv-1 gene on the late stages of virus synthesis. J. Virol., 12, 523


Flaks, A. (1968). The susceptibility of various strains of neonatal mice to the carcinogenic effects of 9, 10-dimethyl-1,2-benzanthracene. Eur. J. Cancer, 4, 579
Fuller, J. L. and Sjursen, F. H. (1967). Audiogenic seizures in eleven mouse strains. J. Hered., 58, 135
Ghaffar, A. and James, K. (1973). The effect of antilymphocyte antibody on the humoral immune response in different strains of mice. Immunology, 24,455
Goodall, C. M., Christie, G. S. and Hurley, J. V. (1975). Primary epithelial tumour in the right atrium of the heart and inferior vena cava in NZR/Gd inbred rats: pathology of 18 cases. J. Pathol., 116,239
Green, E. L. (1941). Genetic and non genetic factors which influence the type of skeleton in an inbred strain of mice. *Genetics*, 26, 192


Henderson, N. D. (1970). Genetic influences on the behaviour of mice can be obscured by laboratory rearing. J. Comp. Physiol. Psychol., 72, 505

526


527


James, K. and Milne, I. (1972). The effect of anti-lymphocytic antibody on the humoral immune response in different strains of mice. I. The response to bovine serum albumin. Immunology, 23,897


Kano, K. and Mizuma, Y. (1974). Comparison of the total blood volume in four inbred strains of mice with different hemoglobin types. Exp. Animals (Japan), 23, 123


Kishimoto, Y. (1972). Breeding of coisogenic mice for studying natural resistance to infection. Exp. Animals (Japan), 21,47
Klein, J. (1973). List of cogenic lines of mice. I. Lines with
differences at alloantigen loci. *Transplantation*, 15, 137
Springer-Verlag, Berlin
Klein, J., Bach, F. H., Festenstein, H., McDevitt, H. O.,
Genetic nomenclature for the H-2 complex of the mouse.
*Immunogenetics*, 1, 184
Klein, T. W. and DeFries, J. C. (1970). Similar polymorphisms of
enzyme activity of livers of various strains of mice. *Cancer
Res.*, 30, 1846
Relationships between arylhydrocarbon hydroxylase inducibility
and sensitivity to chemically induced subcutaneous sarcomas in
abhängigkeit der Wirkung des virus der myeloischen leukamie der
Maus von versuchiedenen biologischen Bedingangen der infizierten
tiere. *Arch. Geschwulstforsch.*, 20, 22
non-pregnant nulliparous mice: a genetic investigation. *Behav.
Biol.*, 13, 113
musculus*. I. Genetic control of inbred strains of mice using
starch gel-electrophoresis. *Biochem. Genet.*, 14, 319
Kryshkina, V. P. and Malashenko, A. M. (1973). Genetic variation of
non-inbred laboratory mice with the use of analyser strains
carrying specific loci. *Genetika*, 9, 52 (in Russian; English
summary)
C57BL/6 mice. I. Sex differences in survival curves. *J.
Gerontol.*, 30, 157
Kutscher, C. L. and Schmalbach, N. L. (1975). Effects of water
deprivation, NaCl injection, and seven aversive taste stimuli on
drinking in two normal mouse strains and one with diabetes
insipidus. *Physiol. Behav.*, 15, 659
deficiency associated with diabetes insipidus in the SWR/J mouse.*
*Physiol. Behav.*, 14, 815
Genetics of the immune response. I. The immune response to the
J. Immunol.*, 1, 201
Lamoreuux M.L. and Galbraith D.B. (1986). DK/Lm: A strain of
laboratory mouse with an unusual expression of the lethal yellow
Lane, P. W. and Murphy, E. D. (1972). Susceptibility to spontaneous
pneumonitis in an inbred strain of beige and satin mice. *Genetics*, 72, 451
Lane-Petter, W. and Bloom, J. L. (1957). Control of genetics
variation. Laboratory Animals Centre Collected Papers, 6, 51
Law, L. W. (1966a). Studies of thymic function with emphasis on the
role of the thymus in oncogenesis. *Cancer Res.*, 26, 551


Lynch, C. J. The so-called Swiss Mouse. Lab. Animal Care, 19, 214


Nagasawa, H., Yanai, R. and Miyamoto, M. (1972). Differences in mammary development and pituitary and placental mammotropin
levels of two inbred strains of mice (DDD and DSD) established from the common ancestor (dd). *Exp. Animals (Japan)*, 21,205


Naruse I. and Kameyama Y., Developmental abnormalities of the mouse brain in hereditary polydactyly (polydactyly Nagoya, Pdn). *Teratology* 22, 17A.


535


Pelzer, C. F. (1965). Genetic control of erythrocytic esterase forms in Mus musculus. Genetics, 52, 819


Sakellaris, P. C., Peterson, A., Goodwin, A., Winget, C. M. and Vernikos-Danellis, J. (1975). Response of mice to repeated photoperiod shifts:

539


Shepard, C. C. and Habas, J. A. (1967). Relation of infection to tissue temperature in mice infected with *Mycobacterium marinum* and *Mycobacterium leprae*. *J. Bacteriol.*, 93, 790


Snell, G. D. and Bunker, H. P. (1965). Histocompatibility genes of mice. V. Five new histocompatibility loci identified by congenic resistant lines on a C57BL/10 background. Transplantation, 3, 235


Steinberg, A. D., Pincus, T. and Talal, N. (1971). The pathogenesis of autoimmunity in New Zealand mice. III. Factors influencing the formation of anti-nucleic acid antibodies. *Immunology*, 20, 523


Szepsenwol, J. and Boschetti, N. V. (1975). Primary and secondary heart tumors in mice maintained on various diets. *Oncology*, 32, 58


Vesell, E. S. (1968). Factors altering the responsiveness of mice to hexobarbital. *Pharmacology*, 1, 81


Wahlsten, D. (1974). Contribution of the genes albinism (c) and retinal degeneration (rd) to a strain-by-training procedure interaction in avoidance learning. *Behav. Genet.*, 3, 303


Webster, L. T. (1933a). Inheritance and acquired factors in resistance to infection. I. Development of resistant and susceptible lines of mice through selective breeding. J. Exp. Med., 57, 793

Webster, L. T. (1933b). Inheritance and acquired factors in resistance to infection. II. A comparison of mice inherently resistant or susceptible to Bacillus enteritidis infection with respect to fertility, weight and susceptibility to various routes and types of infection. J. Exp. Med., 57, 819


Weibust, R. S. (1973). Inheritance of plasma cholesterol levels in mice. Genetics, 73, 303


Weir, J. A. (1962). Hereditary and environmental influences on the sex ratio of PHH and PHL mice. Genetics, 47, 881


Wolff, G. L. and Pitot, H. C. (1973). Influence of background genome on enzymatic characteristics of yellow (Av<sup>y</sup>/a, Av<sup>+</sup>y/a) mice. Genetics, 73, 109


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Research News

Esterase-29. Following disc electrophoresis of serum, genetic variation in activity of a single-banded carboxylesterase termed esterase-29 (ES-29) was detected. Three phenotypes were distinguished. ES-29A is a null type and was found in SEG/1, a partly inbred (F14) line of M.spretus. ES-29B designates strong expression and is widely distributed among laboratory inbred strains such as BALB/cJ. ES-29C comigrates with ES-29B but is only weakly expressed. It was found in MOLF/Ei and in other derivatives of M.m.molossinus. Es-29 is considered to be a structural locus.

Esterase modifying locus-1 (Mse-1). ES-29 (see above) appeared as a double band in the experimental strain LFA/Fre (this strain was described by Scherer et al., Genomics 5,275, 1989). The more anodal band of this doublett could not be distinguished from the above described ES-29B. A presumably modifying locus termed Mse-1, was found to control presence (Mse-1m) or absence (Mse-1a) of the second band. Backcrosses involving LFA/Fre and BALB/cJ revealed that Mse-1 is very closely linked to Pre-2 on chromosome 12. The allele m originates from wild M.m.molossinus which we obtained from V.Chapman in 1977. The same allele was also observed in MOL3/JA and in Cas-Bgr; the latter strain was kindly supplied by K.Moriwaki.
Research News

1. Detection of large autosomal deletions

Cytological study of dominant mutations deriving from radiation experiments has revealed that a significant proportion are associated with cytologically-visible deletions. Two deletions at the steel (Sl) locus have already been described (Cattanach and Rasberry, MNL 80:156-157; 157-158, 1988; Evans et al, MNL 81:66, 1988; Mouse Genome 86:230, 1990. Two further mutations at the Sl locus deriving from spermatogonial irradiation have since been found to carry deletions of chromosome 10 with up to 10% of the chromosome being missing. A dominant spotting mutation has been found to carry a small deletion in a proximal region of chromosome 9; crosses with animals carrying curly whiskers (cw), short-ear (se) and tail-kink (tk) has shown that cw is expressed when carried in trans to the deletion. A dominant mutation characterised by a large rounded head and wide set eyes carries a large deletion from a central region of chromosome 1 which reduces the length of the chromosome by about 25%. One of two mutations that closely resemble chylous ascites (Chy) on chromosome 11 has a large deletion from the middle of chromosome 8. A recessive spotting mutation (s) has also been found to be associated with a large deletion that has taken out approximately 30% of a distal region of chromosome 14. All these deletions, with the possible exception of the chromosome 8 deletion in males, are fully fertile. They are all characterised by variably severe runting, however. The precise locations of these deletions in the banding map is now being established. The findings clearly establish that large deletions induced in spermatogonial stem cells can be transmitted through meiosis and inherited by the progeny. (Cattanach, Rasberry, Burtenshaw, Evans).

2. Three new translocations

a) T(1;7)49H

This translocation arose in an F1 female carrying a
recessive spotting (s) mutation detected in a combined hydroxyurea-X-ray specific locus mutation experiment (spermatogonial treatment). Reduced litter sizes in the allelism tests suggested the presence of a translocation and this was confirmed in banding studies. However, the s mutation (chromosome 14) and the translocation were proven to be independent events as the breakpoints were found to be located at 1D (or at the C/D junction) and at 7F1 (or the E/F junction).

b) T(6;7)51H

This translocation was detected in a further specific locus mutation experiment in which 101/H males were given 2 doses of 3 Gy X-rays 24h apart. The rearrangement first appeared in an F1 female which was retained for testing because of an abnormal foot and short tail. The physical abnormalities proved not to be inherited on breeding, but reduced litter sizes suggested a translocation was present. Banding studies confirmed the existence of the translocation and located the breakpoints to 6A3 or 6B1, and 7D1 or distal 7C.

c) T(1;12)52H

A similar specific locus mutation test using the 3 + 3 Gy 24h fractionation regime, but applied to C3H/HeH x 101/H F1 hybrid males produced a mutation at the brown (b) locus. A reduced litter was observed in the allelism test suggesting the presence of a translocation. Further breeding tests established that the b mutation and the translocation were independent events. This was further confirmed in banding studies which showed the translocation breakpoints to be located at 1F and 12D3. (Rasberry, Cattanach, Burtenshaw, Evans).

3. T(7;18)50H Refinement of breakpoints

Nesbitt and Beechey (MNL 60, 45, 1979) determined the breakpoints as being within bands 7E2-7F2 and 18B3-18D. These have now been refined to bands 7E2 and 18B2. (Evans).
Waggler (wag).

Waggler (wag) is a recessive neurological mutation that arose in the MRL/MpJ-lpr colony maintained at The Jackson Laboratory by Dr. Charles Sidman in 1988. It has been mapped to Chromosome 15 near Gpt-1 using the MEV/1Ty linkage testing stock (Cross 1) and a Chr 15 marker stock homozygous for uw - Gpt-1b - Gdc-1d (Cross 2). The linkage data are summarized in the tables. We believe the order to be cen - uw - Gpt-1 - wag - Gdc-1 - Ca because in Cross 2 (1) the wag - Gdc-1 distance is less than the Gpt-1 - Gdc-1 distance and (2) Gpt-1 - Gdc-1 recombinational events separated Gptrl and wag from Gdc-1 in 9 chromosomes and wag and Gdc-1 from Gpt-1 in 3 chromosomes. The distances between uw - Gpt-1 - Gdc-1 - Ca are similar to those on the GBASE consensus map and determined by us previously (Davisson et al., Genet. Res. 1990, 56:167).

Mutants are characterized by whole body tremor, instability of gait, and growth retardation; they occasionally develop hydrocephaly. Hearing tests by Dr. Lawrence Erway, University of Cincinnati, have shown the mutant is not deaf. It has been placed on the C57BL/6J inbred background to eliminate the effects of the lpr mutation. Both sexes breed. Pathologic studies have revealed no gross neuroanatomical or histopathologic lesions. The mutant will soon be available only from the Frozen Embryo Repository. (Sweet, Bronson, Cook, Spencer, Davisson).

Cross 1. MEV/1Ty cross, partial backcross, wag +/+ Ca x wag +/- +.

<table>
<thead>
<tr>
<th>Progeny:</th>
<th>+ +</th>
<th>wag</th>
<th>+ Ca</th>
<th>wag Ca</th>
<th>Total</th>
<th>RE±SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>F1 female</td>
<td>38</td>
<td>18</td>
<td>56</td>
<td>4</td>
<td>116</td>
<td>19.12 ± 6.72</td>
</tr>
<tr>
<td>F1 male</td>
<td>33</td>
<td>23</td>
<td>54</td>
<td>6</td>
<td>116</td>
<td>19.43 ± 6.72</td>
</tr>
</tbody>
</table>

Cross 2. Chr 15 marker stock cross, intercross<sup>a</sup>, F1 = uw Gpt-1b + Gdc-1d/+ Gpt-1a wag Gdc-1b

<table>
<thead>
<tr>
<th>Progeny Phenotype:</th>
<th>uw</th>
<th>Gpt-1</th>
<th>wag</th>
<th>Gdc-1</th>
<th>Number</th>
<th>RE±SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>uw a</td>
<td></td>
<td></td>
<td>wag</td>
<td>b</td>
<td>4</td>
<td>uw - wag = 32.07 ± 5.33</td>
</tr>
<tr>
<td>uw a</td>
<td></td>
<td></td>
<td>wag</td>
<td>b</td>
<td>1</td>
<td>uw - Gpt-1 = 32.14 ± 4.41</td>
</tr>
<tr>
<td>a/b x wag x b/d</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
<td>uw - Gdc-1 = 43.75 ± 4.69</td>
</tr>
<tr>
<td>a wag</td>
<td></td>
<td></td>
<td>wag</td>
<td>b</td>
<td>24</td>
<td>Gpt-1 - wag = 5.41 ± 2.63</td>
</tr>
<tr>
<td>ab x wag</td>
<td></td>
<td></td>
<td>wag</td>
<td>b</td>
<td>3</td>
<td>Gpt-1 - Gdc-1 = 17.1 ± 2.5</td>
</tr>
<tr>
<td>a wag</td>
<td></td>
<td></td>
<td>wag</td>
<td>bd</td>
<td>9</td>
<td>wag - Gdc-1 = 13.51 ± 3.97</td>
</tr>
</tbody>
</table>

<sup>a</sup> All mice were scored for uw and wag. Total numbers in phenotypic classes for uw and wag were 98 + +, 55 uw +, 40 + wag, 5 uw wag = 198.
2. Mapping of Iapp and Kap genes to Chr 6.

During our ongoing studies of the predisposition to spontaneous insulin-dependent diabetes in the nonobese diabetic (NOD/Lt) mouse strain, we established a linkage of the islet amyloid polypeptide gene (Iapp) and the kidney androgen-regulated protein gene (Kap) with Ly-49 on the distal part of Chr 6. Using cDNA probes for Kap (kindly provided by Dr. G. Watson, The Jackson Laboratory), and Iapp (kindly provided by Dr. G. Bell, University of Chicago, Chicago) for southern blot analysis of DNA from NOD/Lt and nonobese normal (NON/Lt) mice, we identified allelic variants between both strains (Iapp/PstI fragment: NOD-20 kb, NON-7.5 kb; Kap/DraI fragment: NOD-8.3 kb, NON-7 kb). Segregation analysis of 22 [(NOD x NON)F1 x NOD] BC1 offspring revealed no recombination of Iapp and Kap with Ly-49, indicating a close linkage of these loci. Ly-49 maps to a region of Chr 6 marked by Prp, Tpi-1, and Gapd, that is homologous with human 12p13. Human IAPP has been also positioned on 12p, and our Iapp data expand the homologous region in the mouse (Prochazka and Leiter).

<table>
<thead>
<tr>
<th>Results:</th>
<th>Kap</th>
<th>Iapp</th>
<th>Ly-49</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>NOD</td>
<td>NOD</td>
<td>NOD</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>F1</td>
<td>F1</td>
<td>F1</td>
<td>12</td>
<td>12</td>
</tr>
</tbody>
</table>
Spontaneous transformation in cell lines derived from A\textsuperscript{VY}/a mice

The viable yellow, A\textsuperscript{VY}, mutation (agouti region, chromosome 2) enhances hyperplastic and neoplastic responses to toxic stimuli. Fibroblastoid clonal cell lines have been derived from 2.5 day-old A\textsuperscript{VY}/a and a/a mice from the YS/WffC3Hf/Nctr-A\textsuperscript{VY} and VY/WffC3Hf/Nctr-A\textsuperscript{VY} strains. A high, although variable, frequency of spontaneous transformation has been observed in these cell lines, whereas no transformation has been observed in cell lines derived from a/a mice from the same strains. We have also recently established cell lines and clones from 7 day-old mottled yellow A\textsuperscript{VY}/a and pseudoagouti A\textsuperscript{VY}/a mice from both inbred strains. Work is underway to define growth characteristics of these cell lines and to determine the relation of the high frequency of spontaneous transformation to the in vivo effects of the gene. (Wolff, Lyn-Cook, North) (W. L. Hsiao, University of California-Irvine) (G. S. Barsh, Stanford University)

Recent publications:


Possible prevention of neural tube defects (NTDs) in splotch-delayed (Sp<sup>d</sup>/Sp<sup>d</sup>) by food deprivation.

Copp et al. (Development, 104:297, 1988) reduced the incidence of spinal NTDs in curly tail mice by food deprivation of pregnant females for 48 hours prior to posterior neuropore closure. The splotch-delayed NTD model (Moase and Trasler, Teratol., 36: 335, 1987) was tried with the same regime. Pregnant females were taken off food for 24 hours or 48 hours starting gestation day 8. On day 16 they were sacrificed and the embryos genotyped by their Idh-1 isotype. In the 48 hour treatment 3/6 litters survived and had 54% resorptions (r) and 2/11 Sp<sup>d</sup>/Sp<sup>d</sup> embryos one of which had no NTD. For the 24 hour treatment 10/16 litters survived and had 22% r. and 14/48 Sp<sup>d</sup>/Sp<sup>d</sup> embryos 4 (22%) of which had no NTDs. The controls (not deprived of food) were 13/13 litters which had 13% r. and 16/66 Sp<sup>d</sup>/Sp<sup>d</sup> embryos all of which had NTDs. Thus the 24 hour food deprivation increased mortality of whole litters and also significantly prevented NTDs in Sp<sup>d</sup>/Sp<sup>d</sup> (4/14 without NTD versus control 0/16, p = 0.0365). The NTDs were either exencephaly ± spina bifida, spina bifida or curly tail and their overall frequency was not changed by food deprivation (22% vs. 24%). Previously (Teratol., 40: 67, 1989) it was found that 3/27 (11%) Sp<sup>d</sup>/Sp<sup>d</sup> embryos had no NTDs which does not differ significantly from the present 4/14 (29%) found after 24 hour food deprivation. Thus food deprivation for only 24 hours induced 38% loss of litters, did not change NTD type or frequency, but apparently exerted a similar effect to that found by Copp et al. Namely some Sp<sup>d</sup>/Sp<sup>d</sup> mutant embryos appeared outwardly normal. As shown by Copp et al., for curly tail, a possible growth imbalance between the neural tube and surrounding tissues in Sp<sup>d</sup>/Sp<sup>d</sup> neurulation may in some cases have been corrected by the food deprivation. (Mehin and Trasler).
Two new mouse strains with homozygous Rb(8.17)1lem Robertsonian translocation are proposed as the models of the spontaneous mammary tumorigenesis and the experimental infections (Pseudomonas mallei, P. pseudomallei and Mycobacterium tuberculosis H37Rv).

1. BLRB-Rb(8.17)1lem (F25). Characteristics:
- about 70% of virgins have mammary tumors at age 14.1 months, tumor-female survival is 15.9 months;
- 95% of mammary cancer in breeding females, mammary tumors are developed at the age 12.3 months after female short breeding life and at the age 8.4 months after their long breeding period (5 months and more);
- reduced size of the 1st litter up to 3.6 sucklings, then about 5.5 ones until the 6th litter, suckling coefficient is about 60%;
- the most susceptibility to P. pseudomallei and mallei from 7 tested strains;
- very resistant to experimental tuberculosis;
- genetics: a, H-2b, Mup-1k, Hbb3, Trf1, Pre-10, Pre-2a.

CBRB-Rb(8.17)1lem (F29). Characteristics:
- about 50% of mammary cancer in virgins at the age 17.5 months, tumor female survival is 18.4 months;
- about 50% of mammary tumors in breeding females at the age 15.2 months after short breeding life, about 90% of mammary tumors at the age 10.6 months after long breeding period;
- the susceptibility to P. pseudomallei, P. mallei, M. tuberculosis is decreased in comparison with the CBA/CaLacSto;
- genetics: +, H-2k, Mup-1a, Hbb2, Trf2, Pre-10, Pre-2b.
Mouse line subcode symbol - The Institute of Laboratory Animal Resources, National Research Council, Washington, D. C. assigned a "subcode symbol" to be used as part of the description for all transgenic mice that have been or will be produced in our laboratory. It has been registered with the International Committee on Standardized Nomenclature for Mice. The symbol that has been assigned to our laboratory is "Bri."

Transgenic nomenclature - Lists of published transgenic mouse lines generated in this laboratory that are currently being maintained here or in other laboratories appeared in Mouse News Letter No. 84:112-116, 1989 and Mouse Genome No. 87:96-98, 1990. We plan to publish yearly updated lists of mouse lines that have appeared in print since the previous list. The list below includes those lines published during the last year with a reference cited for each line. The designation for each line was determined after conversations with members of the International Committee on Standardized Nomenclature for Mice. Tg = transgenic mouse line. (xxxxxx) = chromosomal location if known and details of the inserted gene. Chromosomal location and gene details are separated by a (;). Gene symbols are those standardized for each species. Gene constructs involving fusions between different genes employ (,). Coinjection of separate gene constructs is indicated by (+). Bri is the laboratory where the mice were generated. The final # indicates the transgenic mouse line assigned a standardized name.

For example: Tg(6;Mt-1,TK)Bril

This is the first transgenic mouse line generated in the Brinster laboratory given a standardized name. It carries a fusion gene construct that has integrated on chromosome 6 and is composed of the mouse metallothionein-1 regulatory region fused to the Herpes Simplex Virus thymidine kinase structural sequences.
Personnel News:

Dr. Pradip K. Ghosh is a postdoctoral Fellow in the Department of Physiology. Drs. Marcelo Cecim, D.V.M. and Kechun Tang, M.D. are Ph.D. students in the Department of Physiology.

Research News:

1- Transgenic mice:

   a- Corticosterone levels in mice expressing human or bovine growth hormone transgenes:

   Ectopic expression of human or bovine growth hormone genes in six lines of transgenic mice (mMT/hGH, mMT/hGH-B, mMT/bGH, PEPCK/bGH1, PEPCK/bGH5 and PEPCK/bGH25) was associated with a significant increase in the circulating levels of corticosterone. All the lines with the metallothionein promoter have one inserted gene copy, whereas the lines with the PEPCK promoter have either 1, 5 or 25 inserted copies of the gene. The elevation in corticosterone was detected in both sexes under basal conditions and after stress. The adrenal activity of 3-beta-hydroxysteroid dehydrogenase was measured in two of these lines, and was found to be significantly increased in transgenic mice. The circulating corticosterone levels did not correlate with known differences between the various lines in male and female fertility or in the life span. (Cecim, Ghosh & Bartke, SIU) (Esquifino, Universidad Complutense de Madrid) (Wagner & Yun, Ohio University).
b- Pituitary and hypothalamic function in mice expressing human or bovine growth hormone transgenes:

The expression of the mouse metallothionein-I (mMT) promoter/human growth hormone (hGH) fusion gene in transgenic mice leads to female sterility and major alterations in the function of the hypothalamic–adenohypophyseal system. These alterations include increases in median-eminence norepinephrine turnover and circulating LH levels, and a decrease in circulating prolactin (PRL) levels in intact males, and an increase in median-eminence dopamine turnover combined with the suppression of plasma PRL levels in ovariectomized (OVX) females. To further characterize these changes and to determine whether they are due to the lactogenic or somatotropic activity of hGH, we have studied hypothalamic and pituitary function in transgenic mice expressing mMT/hGH, mMT/hGH-B, or mMT/bGH fusion genes. In males, the expression of the hGH-B transgene was associated with a reduction in pituitary PRL release in vitro and an increase in LH response to GnRH stimulation, while the bGH transgene did not affect any of the examined parameters of LH and PRL release. Median-eminence norepinephrine turnover was increased in each of the three types of transgenic male mice, while median-eminence dopamine turnover was reduced only in mice expressing the hGH-B transgene. In OVX females, plasma LH was suppressed by hGH-B expression, while median-eminence norepinephrine turnover was suppressed in both hGH-B and bGH mice. The turnover of dopamine was increased in the median eminence of females expressing either of the human genes and reduced in the median eminence of OVX bGH females. We conclude that the hGH-B transgene is weakly lactogenic in mice, and that the chronic stimulation of either GH receptors by bGH, or both GH and PRL receptors by hGH or hGH-B, can lead to profound alterations in the metabolism of hypothalamic neurotransmitters and pituitary hormone release. (Steger, Bartke & Tang, SIU) (Parkening & Collins, U. Texas Medical Branch) (Buonomo, Monsanto Co.) (Wagner & Yun, Ohio University).

2- Andrology:

a- Testicular function in old OF/B mice:

Old (21-22 month) mice were compared to young adult (4-5 month) mice from the same breeding colony. Body and seminal vesicle, but not testicular, weights were higher in old versus young mice. Basal testicular testosterone levels were lower in old than young mice. No differences in basal testicular progesterone and 17-hydroxyprogesterone levels were detected. Injection of hCG elevated testicular 17-hydroxyprogesterone to higher levels in young than in old mice. Testicular progesterone and testosterone rose to
similar levels in response to hCG injection, in old and young mice. These changes appear to be related to a decrease in the efficiency of 17-hydroxylase. When testes from these animals were incubated with or without hCG, no differences in basal media steroid concentrations were detected between old and young mice. Although hCG stimulated the in vitro production of steroids in old and young mice untreated in vivo, the hCG stimulation of 17-hydroxyprogesterone and testosterone production was smaller in old than young mice. Injection of hCG was less effective in increasing basal media progesterone, 17-hydroxyprogesterone and estradiol levels in old mice compared to young ones. This effect on media testosterone seemed to be similar in both types of mice. Injection with hCG reduced the ability of media hCG to elevate progesterone and estradiol levels in old, but not in young, mice. It also reduced the ability of hCG to elevate 17-hydroxyprogesterone and testosterone levels in both types of mice. Basal in vitro aromatase efficiency was lower in old than in young mice. Incubation with hCG caused this parameter to become similar in both old and young mice. In contrast, injection with hCG increased the efficiency of aromatase in young, but not in old mice. Combination of both treatments, cancelled the effects of each other in the two types of mice. (Amador & Bartke).

b- Further studies on the testicular function of NOD mice

When young adult (13-15 week old) male NOD mice were compared with ICR mice of a similar age no significant differences in the testicular concentration of either progesterone, 17-hydroxyprogesterone or testosterone could be detected between the two types of mice. However, plasma levels of testosterone were statistically much lower in NOD than in ICR mice. As a consequence of this, the efficiency of the secretion of testosterone into the circulation was also statistically lower in NOD versus ICR mice. Also, although the testicular steroid levels were not different, when the efficiency of the conversion of testicular progesterone to testosterone was analyzed, it was found to be much less in NOD than in ICR mice. This was found to be the result of a decrease in the efficiency of 17-hydroxysteroid dehydrogenase. When the in vitro studies on these animals were expanded from those previously reported (Mouse Genome 89:265, 1991), it was observed that basal testes incubation media levels of progesterone, 17-hydroxyprogesterone and estradiol were significantly lower in NOD than in ICR mice. Administration of hCG in vitro caused a significantly smaller increase in the media levels of progesterone, 17-hydroxyprogesterone and estradiol in NOD when compared to ICR mice. These findings parallel those observed for media testosterone levels. (Amador & Bartke, SIU) (Mayerhofer, Universitaet Ulm).
Lined \((Li)\), is a semidominant X-linked gene located in the distal portion of the X near Hyp. Although \(Li/+\) females are fully viable, hemizygous males are not found at birth.\(^1\) In order to determine the fate of \(Li/Y\) males, \(Li/+\) females were mated to normal (C3H/HeH \(\times\) 101/H \(F_1\) hybrid) males and their uterine contents examined at 11.5 - 15.5 days gestation. As the \(Li\) stock is maintained on a C3H/HeH \(\times\) 101/H hybrid background, hybrid females were used as controls.

Table 1. Corpora lutea counts and uterine contents from outcrosses of \(Li/+\) females and ++ controls. Mean values per female with S.E are given in parenthesis.

<table>
<thead>
<tr>
<th>female genotype</th>
<th>n</th>
<th>corpora lutea</th>
<th>total implants</th>
<th>normal embryos</th>
<th>dead/retarded embryos</th>
<th>deciduomata</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Li/+)</td>
<td>10</td>
<td>95</td>
<td>93</td>
<td>61</td>
<td>29</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(9.5±0.5)</td>
<td>(9.3±0.5)</td>
<td>(6.1±0.6)</td>
<td>(2.9±0.5)</td>
<td>(0.3±0.1)</td>
</tr>
<tr>
<td>++</td>
<td>7</td>
<td>76</td>
<td>73</td>
<td>70</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(10.8±0.7)</td>
<td>(10.4±0.6)</td>
<td>(10.0±0.5)</td>
<td>(0.4±0.2)</td>
<td></td>
</tr>
</tbody>
</table>

The results (Table 1) do not show any difference between \(Li/+\) and ++ females in the numbers of ova shed or implantation sites. A significant reduction \((p=0.0002)\) in the mean number of live normal embryos from \(Li/+\) females was however indicated, the loss being represented by dead or retarded embryos. Three of these embryos was exencephalic and two had herniation of the abdominal contents, but the remainder had no obvious abnormalities although the degree of retardation was variable. Dead embryos were not seen at the earliest age studied, although retardation of some embryos was evident. The first deaths were noted at 12.5 days. To determine if these retarded and dead embryos represented the \(Li/Y\) class missing at birth chromosome preparations were made from the embryonic membranes of all embryos from 3 \(Li/+\) females at 11.5, 12.5 and 14.5 days gestation. Embryonic sex was determined by examination of C-banded karyotypes to distinguish the Y chromosome.

Table 2. Karyotypes of embryos from outcrosses of \(Li/+\) females.

<table>
<thead>
<tr>
<th>Live normal embryos</th>
<th>Dead/retarded embryos</th>
</tr>
</thead>
<tbody>
<tr>
<td>XX</td>
<td>XY</td>
</tr>
<tr>
<td>12</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>1</td>
</tr>
</tbody>
</table>

In all, 26 embryos were successfully karyotyped (Table 2) of which equal numbers were male or female. Of the 17 live
normal embryos karyotyped 12 proved to be XX and 5 XY. However, all but one of the 9 dead or retarded embryos were XY. It is therefore likely that these represented the Li/Y class. It may be concluded that development of Li/Y males slows at about 11 days gestation with death occurring soon afterwards.

The relatively long pre-natal survival of Li/Y males is surprising in view of the extreme non-random X chromosome expression observed in Li/+ females! Were this due to cell selection, a very early embryonic loss of Li/Y would have been expected. The alternative explanation, namely, that Li influences primary X chromosome inactivation would seem unlikely in view of the gene's distal position on the chromosome 1, far removed from the centrally-located X-inactivation centre, putatively Xce. However, further investigation of this possibility is warranted.

REFERENCES

PRODUCTION OF HIGH AND LOW BODY WEIGHT \textit{lit/lit} DWARVES


Lines for high and low body weight were established to investigate the consequences of selection on fat free body mass, (P-Lines) \cite{1}. After 20 generations the initial replicates were crossed and selection changed to 10 week weight in both sexes \cite{2}. At the start of this experiment 34 generations of selection had been carried out in total and adult body weight in the high line was about twice that in the low line.

As part of an investigation into the involvement of growth hormone (GH) in the differences between the P-Lines \textit{lit} dwarfism was backcrossed into them. \textit{lit/lit} dwarves on a C57BL/J background were mated to unselected mice from the lines. Test matings were carried out in each generation to ensure that the \textit{lit} gene was being propagated. Three generations of backcrossing have been carried out giving a background which is 93\% P-Line on average. The backcrossing program is continuing. \textit{lit/lit} dwarves which differ in body size might be useful for studies into growth.

Effects on body weights of males from birth to 6 weeks of age can be seen below. Clearly GH is not the sole factor in the difference between the high and low P-Lines. This accords with the results of backcrossing \textit{dw} dwarfism, which has greater pituitary effects than \textit{lit} into the lines selected to diverge in growth rate \cite{3}.

\begin{figure}[h]
  \centering
  \includegraphics[width=\textwidth]{growth_graph.png}
  \caption{Growth of male offspring of \textit{lit/+} matings from birth to 42 daye old}
\end{figure}

\begin{enumerate}
  \item I.M. Hastings and W.G. Hill (1989) Animal Production, 48,229-233
  \item H.G. Pidduck and D.S. Falconer (1978) Genetical Research Cambridge, 32, 195-206
\end{enumerate}
IDENTIFICATION OF THE MUS SPRETUS XCE ALLELE

B M Cattanach and C Rasberry
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Three alleles at the putative mouse X-inactivation centre locus, Xce, have been recognised, Xce\(^c\), Xce\(^b\), Xce\(^a\).\(^1\) They were identified by the non-random X-inactivation observed with X-linked genes in heterozygotes; an X carrying Xce\(^c\) is more likely to be the active chromosome in Xce\(^c\)/Xce\(^b\) heterozygotes, and an X carrying Xce\(^b\) is more likely to be the active chromosome in Xce\(^b\)/Xce\(^a\) heterozygotes. More extreme non-random X-inactivation is found in Xce\(^c\)/Xce\(^a\) animals. The relative "strengths" of the three alleles are therefore Xce\(^c\)> Xce\(^b\)> Xce\(^a\). The data supporting this conclusion have been based partly on subjective scoring of the levels of expression of X-linked marker genes acting upon coat colour,\(^2\) partly by the more objective method of scoring the reduction in vibrissa number in tabby (Ta) heterozygotes,\(^3\) and partly by more quantitative enzyme assays available with phosphoglycerate kinase (Pgk-1)\(^1\) and glucose 6 phosphate dehydrogenase (G6pd) enzyme variants.\(^4\)

Ashworth et al (1991)\(^5\) have recently suggested that the Mus spretus X chromosome carries a "strong" Xce allele, this conclusion being based on the low levels of expression of Ta in the coat and ornithine transcarbamylase (Otc) in the liver of heterozygotes carrying a spretus X chromosome. In order to investigate this possibility more objectively the strength of the spretus Xce allele was assessed using the Ta vibrissa test. This provides a measure of the influence of the Xce allele carried on the non-Ta X upon the expression of Ta in Ta/+ females. The influence is mediated through modification of the random X-inactivation process. The vibrissa number in normal mice is 19 but this is reduced in Ta animals. A reduction to 12-14 in Ta heterozygotes (large effect of Ta) indicates the presence of a "weak" Xce\(^a\) allele on the non-Ta X; a near-normal score of 17-18 (small Ta effect) has indicated the strongest known allele, Xce\(^c\). A more marked reduction is found in the Ta/Y males. Scores are based on the mean vibrissa number of 15 or more females.

Mus spretus males were therefore crossed with Ta/Ta females and the vibrissa numbers both of their Ta/+ daughters and Ta/Y sons were scored. The latter provided a control for any non-X chromosomal influence upon the expression of the Ta gene. The mean vibrissa score of the Ta/Y sons was found to be 6.86±0.21 which is consistent with the range of Ta/Y scores obtained on a variety of different genetic backgrounds (6.96-7.64).\(^3\) A non-X chromosomal effect upon Ta expression is not therefore evident. However, the scores of the Ta/+ daughters (18.77±0.078; n=44) were well outside the range typically observed in Ta heterozygotes other than those carrying the "strong" Xce\(^c\) allele on their non-Ta X. Although the score was not significantly different from Xce\(^c\) scores (eg 18.35±0.21; t=0.78), a stronger allele than Xce\(^c\) might not easily be distinguished with this test system. Comparison of the spretus allele with Xce\(^c\) could best be made by
quantitative analysis of PGK-1 isoenzyme activity in \(Pgk-1^a\) \(Xce^c/Pgk-1^b\) \(Xce^{spretus}\) females. An experiment of this kind has been initiated.

REFERENCES

The Ta<sup>25H</sup> mutation represents a small deletion in the X chromosome spanning the tabby (Ta) and testicular feminisation (Tfm) loci. Because Ta<sup>25H</sup> heterozygotes show a characteristic X-inactivation mosaic phenotype it has been concluded that the X-inactivation centre (putatively Xce), although closely-linked to Ta, is not included within the deletion. The Xce allele present on the Ta<sup>25H</sup> X may be assumed to be Xce<sup>a</sup> on the basis that the deletion was induced in an animal carrying a C3H/HeH (Xce<sup>a</sup>) X chromosome, and this provides a means of determining whether the randomness of X chromosome expression in Ta<sup>25H</sup> heterozygotes is modified.

To test this possibility the level of reduction in vibrissa number due to Ta in Ta<sup>25H</sup>/+ females was compared with that in heterozygotes for a presumed point mutation at the Ta locus (Ta<sup>23H</sup>), which having been induced in a C3H/HeH X chromosome, is also carried in coupling with the Xce<sup>a</sup> allele. Both the Ta<sup>25H</sup> deletion and the Ta<sup>23H</sup> mutation are maintained by crossing the heterozygous females with C3H/HeH x 101/H F<sub>1</sub> hybrid males. Genetic background influences were therefore not expected. The mean vibrissa score of a sample of 20 Ta<sup>25H</sup>/+ females was found to be 17.65±0.43 (indicating little effect due to Ta) which is significantly greater (t=4.80; P=0.3x10<sup>-6</sup>) than that (15.21±0.25) of a larger sample of Ta<sup>23H</sup>/+ females obtained earlier. An exceptional feature was the high proportion of females (40%) with a normal vibrissa count. These observations suggest that the deleted X chromosome of Ta<sup>25H</sup>/+ females is less likely to be expressed than the normal one. In view of the deleterious effects of the deletion upon both Ta<sup>25H</sup>/+ and Ta<sup>25H</sup>/Y animals, the non-random X expression might be considered a consequence of cell selection operating against cells carrying the deleted X in the active condition. However, non-randomness is not evident in heterozygotes for a number of mutant genes eg mottled alleles, which cause more severe post-natal or even pre-natal lethalities. The possibility therefore remains that the deletion may have modified the Xce locus with the consequence that the ability of the Ta<sup>25H</sup> X to become the active chromosome is impaired.

REFERENCES

TIBIALESS (Tba): A NEW SEMI-DOMINANT MUTANT IN THE MOUSE

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Mutations causing mirror image polydactyly, ectrodactyly and hemimelia in the mouse are by no means uncommon (1): as a group they are of interest because the combination of these effects seen in a particular genotype and the fact that they are often differently expressed in fore or hind limbs suggests that different mutants may represent the subtly different mechanisms of embryonic development which underlie different adult phenotypes. Tibialess warrants description as an original combination of phenotypic entities.

A single male with a deformed haunch appeared in a line being selected for an increased rate of chromosome damage. This was derived from an unselected inbred line descended from mice trapped at site 5, part of the pure wild Peru—Coppock. This stock is itself remarkable for its high rate of chromosome damage (2). The abnormal male and his descendants were crossed within the Peru stock and outcrossed to two other laboratory stocks, AG/Cam (an inbred line) and line 4 (a closed colony). Segregation ratios were recorded from affected x normal, reciprocal, and abnormal x abnormal crosses. Mice were classified at the age of 0–8 days.

Fifty adult Tibialess mice and fifty normal littermates were examined under the dissecting microscope after being cleared and stained with alizarin red S (JLC,DRJ).

| Table 1. Segregation of Tibialess and normal mice. |
|---------------------------------|------|------|------|---------------|------|--------|-------|
| Affected Normal Total No of |   |   |        | No              |       |       |       |
| litters 0–8d tested            |   |   |        | dead tested      |       |       |       |
|--------------------------------|---|---|------|---------------|------|-------|
| affected m x normal f          | 148| 242| 390  | 72             | 8    |        |       |
| affected f x normal m          | 14 | 24 | 38   | 11             | 2    | 1:1    | 25.27 | <0.001 |
| affected f x affected m        | 102| 82 | 184  | 43             | 4    | 2:1    | 10.45 | <0.01  |
|                                |    |   |      |                |      | 3:1    | 37.56 | <0.001 |

litters and so were used sparingly. In matings between affected and non—affected mice the ratio of abnormal:normal (presumed heterozygote:normal) differs significantly from the expected 1:1 with a deficiency of abnormals. In abnormal x abnormal matings there is a very poor fit to the expected 3:1 ratio, but a less poor fit to a 2:1 ratio which assumes that the homozygous abnormal is a prenatal lethal. Thus there is a severe deficiency of abnormals from all types of mating. Litter size was small (affected x normal 5.2; affected x affected 4.3).

Linkage was tested with hammer—toe and grey—coat (Hm,gc chromosome 5) and
extreme non-agouti (a⁵, chromosome 2). A preliminary report has been given elsewhere (3). The results, presented in Table 2, give no indication of linkage.

Table 2. Linkage test crosses.

<table>
<thead>
<tr>
<th>Mutants* tested</th>
<th>Total</th>
<th>Ratio</th>
<th>X² test!</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>M</td>
<td>m</td>
<td>m</td>
</tr>
<tr>
<td>M</td>
<td>16</td>
<td>8</td>
<td>15</td>
<td>13</td>
</tr>
<tr>
<td>Tba +/+/ + Tba x + + /+ +</td>
<td>15</td>
<td>5</td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>Tba +/+ gc x + gc/ + gc</td>
<td>45</td>
<td>14</td>
<td>42</td>
<td>19</td>
</tr>
</tbody>
</table>

Hm = hammer toe, gc = greycoat, Tba = tibialess. Mutants: 1st row: M = Hm, m = normal toes 2nd row: M = normal coat, m = gc; 3rd row: M = A, m = a (non-agouti) or a⁵ (extreme non-agouti). The X² tests the equality of N (non-recombinants) with R (recombinants) in the previous column.

The insignificant deficit of recombinants in the segregation with hammer-toe is more likely to be due to synergism between these skeletal mutants than to loose linkage.

In 50 tibialess mice and 50 normal controls examined as alizarin clearance preparations no abnormality was seen in the forelimbs. In the hind limbs a series of abnormalities of varying severity was present. In some cases digit I was replaced by a digit with 3 phalanges (26 limbs: 15R;11L) or missing (38:17R;21L). In eight limbs digit I was represented by a proximal metatarsal splint (3R;5L). In a further 6 limbs digits I and II were affected (3R;3L), digit I being absent and digit II represented by a metatarsal splint, a claw plus the distal end of a phalanx or absent. Frank polydactyly was seen as a prehallux of claw plus distal part of phalanx in 3 limbs (all R). Soft tissue syndactyly (10:4R;6L) and bony syndactyly (3:0R;3L) were seen occasionally.

In addition the tibia was often absent (36:21R;15L), represented only by its proximal end (30:17R;13L) or thinned (2R). The fibula was almost universally bowed and thickened, often with evidence of healed fractures. The femur was involved three times, bilaterally in one individual and on the right side in the other. In the bilaterally affected individual the femora were represented by bony knobs: one on the left and two (femoral head + remainder?) on the right. The sciatic foramen was incomplete in all three cases.

The segregation data presented above indicate that Tibialess behaves as a semi-dominant condition. In matings of normal x Tibialess individuals there was a significant deficit of Tibialess mice not accounted for by deaths between birth and classification by 8 days. We conclude that a proportion of Tba/+ Tibialess mice die between conception and birth. At and after birth there are further sporadic losses of affected individuals. Penetrance of the condition is complete, since there was never any difficulty in classifying individuals as normal or grossly abnormal, i.e. there were no intermediates.

Assuming a perfect zygotic ratio of 1:1 the number of deaths in our affected x normal matings (Table 1) is 104 or 0.39 of the expected zygotic number of heterozygotes. Similarly from the affected x affected matings a perfect zygotic ratio of 2:1 for
heterozygotes to normal (assuming no surviving homozygotes) assumes 62 deaths or 0.38 of the expected zygotic number. A perfect zygotic ratio of 3:1 (assuming survival of homozygotes) would indicate 144 deaths or 0.58 of the affected ones. The close agreement in death rates where 1:1 and 2:1 are expected proves the lethality of the homozygotes.

The age of death of the heterozygotes is unknown, but must be mainly prenatal since classification, at birth only, produced few dead affected neonates (data not shown). The average litter sizes (5.2 for affected x normal, 4.3 for affected x affected) are slightly closer than expected with the 0.38 antenatal death rate. This would be expected if the homozygotes die early since this would reduce intrauterine competition and allow more heterozygotes to survive from the latter than from the former.

The lack of linkage with markers on chromosomes 2 and 5 suggests that Tba is unlikely to be allelic with Strong's luxoid (1st, 4) or luxate (lx,5). Neither of these mutants is regularly lethal in homozygous form.

A number of other conditions in the mouse produce syndromes comprising polydactyly, ectrodactyly, and hemimelia, often in different combinations or affecting different limbs (1). Tibialless resembles various members of this group in some aspects of the syndrome but not in others. For instance, like dominant hemimelia (Dh,6), it is dominant with a lethal homozygous form (though Dh/Dh sometimes survive to breed). Extra — toes (Xt,7) is also dominant with a prenatally lethal homozygote but expresses polydactyly rather than hemimelia in the heterozygote. Green's luxoid (lu,8) is recessive but also affects the forelimbs.

Tibialess produces a seemingly unique combination of dominant inheritance, a prenatal lethal homozygote and full penetrance. The usual minimal expression of triphalangy of the hallux or an extra preaxial digit without tibial involvement are absent in our sample. It seems likely that the stock of mutations affecting the limbs of mouse and chick reflect deviations in normal embryology. In the past (see 1 for summary) studies of mutants have complemented experimental embryology, and comparisons between mutant and normal limbs have produced much valuable data on normal development. Recent studies with homeobox genes have extended the potential greatly: we may well soon be able to spotlight the exact areas where a gene product has to be abnormally expressed in order to promote polydactyly or ectrodactyly. With this increase in resolution subtly different mutations may well enjoy increased utility and popularity.

AN ENU INDUCED MUTATION IN THE β-GLOBIN GENE, Hbb-b1, RESULTS IN THE LOSS OF AN Rsa I SITE

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INTRODUCTION

A mutation in one of the genes encoding adult β-globin (Hbb-b1) has been recovered following treatment of spermatogonial stem cells from 101/H mice with the mutagen N-ethyl-N-nitrosourea (ENU) (1). The mutant allele, Hbb-b1d-ml, is characterised by substitution of the β145 amino acid, tyrosine, with cysteine (1) and is likely to be the result of a point mutation. The DNA sequence at β145 tyrosine is known to be TAC in the BALB/c inbred strain (2) and is contained within the recognition sequence, GTAC, for the restriction enzyme Rsa I. If β145 tyrosine in the 101/H strain also has the codon TAC, the mutation should be detected as an Rsa I restriction fragment length variation between the Hbb-b1 alleles of wild type and mutant (Hbb-b1d and Hbb-b1d-ml, hereafter referred to as [d] and [d-ml]).

METHODS

Genomic DNA was obtained by standard proteinase digestion and phenol extraction procedures. PCR was performed on 5µg genomic DNA in a final volume of 100µl containing 40pmol of each Hbb-b1 specific primer, 100µM of each dNTP, 0.1% (w/v) Nonidet P50, 10mM Tris-HCl pH 8.3, 40mM KCl, 1.5mM MgCl2 and one unit of Taq DNA polymerase. Thirty amplification cycles (1 min at 94°C, 1 min at 55°C and 2 min at 72°C) were performed and the aqueous phase was recovered following successive chloroform, phenol/chloroform and chloroform extractions. Following ethanol precipitation and resuspension in 50µl water the amplified sample was digested at 37°C for 16 hours with 10 units of Rsa I, extracted with phenol/chloroform, precipitated in 2 volumes ethanol and resuspended in 20µl TE and 2µl loading buffer. The digested (+) and undigested (-) samples were subjected to electrophoresis in a 2% agarose gel in TPE buffer containing 0.5µg/ml ethidium bromide.

RESULTS & DISCUSSION

The Hbb-b1 specific primers from the region that flank the β145 codon (Fig 1), were used to generate a 250 bp fragment containing Exon III of the Hbb-b1 gene from the wild type and mutant alleles. On Rsa I digestion of wild type amplification product the 250 bp fragment was replaced with a new band of approximately 200 bp (Fig 1). However, the amplification product from the mutant allele [d-ml] was not digested on treatment with Rsa I and in heterozygotes ([d]/[d-ml]), both bands were seen. These findings show that the codon for β145 tyrosine in the Hbb-b1 gene of 101/H is TAC and that this sequence is not present in the mutant, confirming the results of the amino acid analysis.

REFERENCES

HEREDITARY HYPERTYROSINEMIA MOUSE

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INTRODUCTION

Hypertyrosinemia in humans, a congenital amino acid metabolic disorder, is currently known to be of three types, each of which is caused by an abnormality in a different metabolic enzyme.

We have been screening the blood levels of amino acids in 24 strains of mice including 19 inbred strains newly obtained from the ICR outbred strain. As a result, one strain with hypertyrosinemia was found. In this study, we performed enzymological and genetic studies on this disorder.

MATERIALS and METHODS

The blood levels of amino acids were measured using whole blood or plasma. Tyrosine metabolic enzymes were measured using liver as the materials.

RESULTS and DISCUSSION

Amino acid screening showed that the III strain is hypertyrosinemic (about 20 mg/dl at 1mo of age) and the others are low (<3 mg/dl). This strain does not show any clinical symptoms by appearance, and reproductive activity and life span of the strain are normal.

Three tyrosine metabolic enzymes were measured in the III strain and the IST strain as control. Activities of fumarylacetoacetase, cytosolic tyrosine aminotransferase and mitochondrial tyrosine aminotransferase in both strains were normal. Activity of 4-hydroxyphenylpyruvic acid dioxygenase was high in IST, but undetectable in III. Urinary excretion of three kinds of tyrosine metabolites (4-hydroxyphenylpyruvic acid, 4-hydroxyphenyllactic acid and 4-hydroxyphenylacetic acid) in the tyrosinemia mice was markedly increased.

Mating experiments were performed using the III and IST strains. The tyrosine value of F₁ was low (<3 mg/dl). When the tyrosine values were examined in 75 backcross mice (40 females and 35 males) obtained by mating F₁ and III, the ratio of the high (>7 mg/dl) to low (<3 mg/dl) values was 42 : 33. The ratio of females to males showing high values was 23 : 19 and that showing low values was 17 : 16. 10 backcross mice obtained by mating F₁ and IST showed low tyrosine values.

In summary, a mouse strain, III, had hypertyrosinemia which showed an autosomal recessive mode of inheritance. The hypertyrosinemia was due to deficiency of 4-hydroxyphenylpyruvic acid dioxygenase, but the mode of inheritance of the enzyme deficiency has not yet been tested. Thus the locus has been named provisionally Hpd, 4-hydroxyphenyl pyruvic acid dioxygenase, and the low activity allele found in strain III, as Hpd<sup>h</sup> to indicate the hypertyrosinemia. Strain III is a good model for human Type III tyrosinemia.

REFERENCES

HIGH THROUGHPUT MOUSE DNA ISOLATION FOR SOUTHERN BLOT
PROGENY GENOTYPING

John Schimenti
Case Western Reserve University, Dept. of Genetics

INTRODUCTION
RFLPs continue to be the primary tool for mouse genome mapping. They are critical both to specific reverse genetics projects and large-scale genome projects. The spretus/musculus backcross has been utilized for both purposes with good success. These endeavors involve isolating DNA from hundreds of progeny. During the course of an experiment requiring Southern analysis of several thousand progeny, we developed the following procedure to process large numbers of samples easily and rapidly. It was optimized for implementation by unskilled individuals.

METHODS

DNA isolation  Cut off a newborn pup’s head, and squeeze the brain into a 1.5 ml microtube. Store at -20° until the next step. Add 600 µl of NTES-Mod2 (10 mM Tris pH=8.0; 25 mM EDTA; 50 mM NaCl; 0.5% SDS) and 15 µl of 10 mg/ml Proteinase K. Rock overnight at 55°. Add 600 µl of 1:1 phenol : chloroform, and rock vigorously for 30 minutes. Spin 10 min. in microfuge. Using clipped-off 200 µl pipetman tips, take off 100 µl of the top (DNA) phase and transfer to a microtitre dish. Discard the remainder.

Restriction Digest  Each reaction will have: 3µl DNA; 2.8 µl 10X enzyme buffer; 0.2µl of 10 mg/ml RNase; 21 µl H2O; 1 µl enzyme. Do the digests in 96 well plates. Use an 8 channel pipettor to transfer the DNAs from the storage microtitre dish to another for digestion. Make a digest mixture containing everything but the DNA. Add 25 µl of the mix to each well using the the 8-channel pipettor. Incubate the reactions in a CO2 incubator (the moisture prevents evaporation) for 3 hours. Use the 8 channel pipettor to add 3 µl loading dye. Load all on gel.

RESULTS and DISCUSSION
The key features of this procedure are a single organic extraction step, elimination of alcohol precipitation, and adaptation to the microtitre dish format. This was achieved by minimizing the SDS and EDTA concentrations in the lysis solution, and using a clean, abundant source of DNA (brain). Since only a small portion of the phenol:chloroform extracted DNA needs to be recovered, the interface is completely avoided (which makes it simple for novices). If two microfuges are available, 100 samples can be processed in 45 minutes once the first wave of samples start spinning. The DNA is digestible by all the enzymes tested :EcoRI, BamHI, BglII, PstI, PvuII and TaqI. A set of EcoRI digested DNAs are show in the figure.
Juvenile depilation (*jd*): a new hair loss mutation in the mouse.
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The Jackson Laboratory, Bar Harbor, ME 04609 USA

Introduction

We describe a new autosomal recessive mutation that causes dorsal hair loss in weaning age homozygotes and a rough unkempt looking coat in adult homozygotes. The *jd* mutation has been mapped to mouse Chromosome 13 near muted.

Materials and Methods

The *jd* mutation arose in The Jackson Laboratory's Animal Resources C3H/HeJ colony in 1984. Genetic mapping crosses are described in the Results. To define the hair defect samples of hair from 3-4 *jd/jd* and +/+ littermate controls of both sexes at 26-28 days and 6 months of age were collected on sticky tape, placed on glass slides, and examined for hair type. To look for pathologic lesions in the skin hematoxylin and eosin stained paraffin sections of skin from two *jd/jd* and two *jd/+* 21 day old mice and from two *jd/jd* and two *jd/+* 26 day old mice were prepared.

Results

Phenotype: Some homozygotes (*jd/jd*) can be identified at 11 days of age when hair starts to thin around the eyes, top of head, and nape of neck. Vibrissae are normal. Weaning age (21-25 days) *jd/jd* mice are easier to identify. The dorsum changes from skin with sparse hair to skin devoid of hair. A very fine stubble of hair with an occasional longer bristle can be seen with the aid of a dissecting scope. Hair remains on the snout, around the legs and tail and on the belly. Hair on the dorsum begins to regrow at 28 days, concurrent with the second normal hair growth cycle. Thereafter, the dorsum is never devoid of hair during successive hair cycles, although the adult coat appears unkempt and becomes thinner with age. Hair loss is more noticeable among aging *jd/jd* females than males.

Hairy types: Four types of hair are present in the normal mouse coat; guard hairs, awls, auchenes and zigzags. Guard hairs, awls and auchenes comprise the over hairs, zigzag hairs comprise the undercoat. The majority of hair types observed in the coat of both juvenile and adult *jd/jd* homozygotes were awls and auchenes. Guard hairs were thin and few in number. Very few zigzag hairs were found, and those observed had no medulla. Numerous short broken pieces of fine septate hair observed in dorsal hair samples from 26-28 day old homozygous *jd/jd* mice could not be classified. These short segments of hair were not found in dorsal hair samples from adult homozygotes.

Histology: Longitudinal and cross sections of skin from 21 and 26 day old *jd/jd* and +/+ mutant and control mice were similar. At 21 days, the hair follicles were in the atrophic catagenic stage in both. At 26 days of age, both had numerous follicles in the hyperplastic stage of development.

Genetics: Linkage was found between *jd* and pearl (*pe*) on Chr 13. Crosses and results are listed below. The estimates of recombination values were calculated using Finney's tables. The combined RE from crosses 1-4 for *jd* - *pe* is 16.55 ± 1.84.

<table>
<thead>
<tr>
<th>Mating</th>
<th>++</th>
<th>jd+</th>
<th>+pe</th>
<th>jdpe</th>
<th>Total</th>
<th>RE ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. jd +/- pe x jd +/- pe</td>
<td>164</td>
<td>70</td>
<td>60</td>
<td>1</td>
<td>295</td>
<td>16.31 ± 5.54</td>
</tr>
<tr>
<td>2. jd +/- pe x jd pe/jd pe</td>
<td>9</td>
<td>56</td>
<td>33</td>
<td>12</td>
<td>110</td>
<td>19.09 ± 3.75</td>
</tr>
<tr>
<td>3. jd pe/+ x jd pe/+ +</td>
<td>180</td>
<td>12</td>
<td>25</td>
<td>28</td>
<td>245</td>
<td>18.75 ± 2.89</td>
</tr>
<tr>
<td>4. jd pe/+ x jd pe/jd pe</td>
<td>26</td>
<td>4</td>
<td>3</td>
<td>34</td>
<td>67</td>
<td>10.45 ± 3.74</td>
</tr>
</tbody>
</table>
Cross 3 was a three point intercross with muted (mu) in repulsion to jd and pe. No recombinants between mu$^+$:jd were detected in 245 F2 progeny, giving a maximum estimated recombination frequency (95% upper confidence level) between mu and jd of 22% and suggesting jd was close to mu and proximal to pe. (We have omitted mu from the table to simplify presentation of the jd - pe data.) To confirm the position of jd a three point cross using a Robertsonian chromosome Rb(11.13)4Bnr was made: Rb +/+ jd pe x + jd pe/+ jd pe. The data from this cross (shown below) gives the recombination percentages shown and the order cen - jd - pe.

<table>
<thead>
<tr>
<th>Phenotype of Progeny</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rb+ +</td>
<td></td>
</tr>
<tr>
<td>+ jd pe</td>
<td></td>
</tr>
<tr>
<td>Rb jd pe</td>
<td>37</td>
</tr>
<tr>
<td>+ + +</td>
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<tr>
<td>+ + pe</td>
<td>1</td>
</tr>
<tr>
<td>Rb - jd:</td>
<td>31/149=20.81±3.33</td>
</tr>
<tr>
<td>jd - pe:</td>
<td>36/149=24.16±3.51</td>
</tr>
<tr>
<td>Rb - pe:</td>
<td>63/149=42.28±4.05</td>
</tr>
</tbody>
</table>

Because jd maps near the hair loss mutation furless (fs)$^{(2)}$, we tested jd for allelism with fs. Eight normal progeny were obtained from a mating between a homozygous jd/jd female and a homozygous fs/fs male, proving that jd is not a remutation to furless.

Discussion: We describe a new hair loss mutation in the mouse that affects the first hair growth cycle and causes permanent failure of zigzag hair growth. We hypothesize the locus causes a defect in hair follicle development. Guard hairs are produced from follicles initiated in the 14-17 day embryo, awls and auchenes are produced by follicles initiated at 17-19 days, and zigzags are produced from hair follicles initiated after birth$^{(3)}$. Despite the striking loss of hair beginning at 21 days of age in jd/jd mice, there were no differences histologically between mutants and controls at that time or later after hair regrowth had begun. We hypothesize that the mutation affects the intrafollicular environment such that the first growth of hairs of all 4 types is brittle resulting in their loss during the first catagenic stage. Thereafter the mutant hair coat recovers with the exception of the zigzag hairs. Developmental studies to test this hypothesis would require a closely linked marker detectable before birth to differentiate mutants from controls when hair follicles are developing. We plan to store the mutation as frozen embryos only.

References

Acknowledgements
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MEV/2Ty-at Ps Mlw Cja, AN IMPROVED MEV LINKAGE TESTING STOCK

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INTRODUCTION

In 1989 we reported the construction of a new linkage testing stock, MEV/1Ty, bearing 11 ecotropic murine leukemia virus proviruses located on ten different chromosomes (2). The presence or absence of these proviruses can be individually scored in Southern blots of HindIII digested, genomic DNA using the ecotropic MuLV-specific probe pEcB4. In addition to these three dominant visible markers had been introduced: hammer-toe (Hm), steel (Sl), and caracul-J (CaJ). However, because Hm and Sl are located on the same chromosomes as two of the proviral markers (Emv-24 and Emv-25, respectively), this choice of visible markers was sub-optimal. Furthermore, although all eleven proviruses could be discriminated in Southern blots of HindIII DNA, PvuII is the preferred restriction enzyme for analysis of ecotropic proviral analysis because it produces more dispersed 3' junction fragments. However, two of the proviruses (Emv-13 and Emv-25) could not be separated in PvuII digests. Here we describe a new version of the MEV stock that incorporates a new combination of dominant visible markers, and contains ten proviruses separable in either PvuII or HindIII digests.

MATERIALS AND METHODS

The source of the dominant visible markers white (Mlw, Chr 6) and black-and-tan (at, Chr 2) was the MWT/Le strain, while the source of polysyndactyly (Ps, Chr 4) was the C57BL/6J-Ps stock. Methods for DNA isolation, Southern analysis with the ecotropic-specific probe, and computing the distances swept have been described (2). The positions of individual markers and the genetic lengths of individual chromosomes are taken from the most recently published comprehensive map (1).

RESULTS

To be able to use PvuII as the restriction enzyme without sacrificing a marker, we have introduced the visible black-and-tan allele (at) of the agouti locus on Chr 2 and, simultaneously eliminated the closely linked Emv-13 provirus. Black-and-tan is an excellent marker because it is generally easy to classify, dominant to both nonagouti and wild-type, and appears not to affect viability and reproduction, even in homozygotes.

The inclusion of the markers Ps and Mlw provides needed markers for Chr 4 and 6, respectively. Since both of these markers are near the middle of large linkage groups, they sweep maximal regions. Both of these markers are fully penetrant on different backgrounds. The Mlw mutation may interfere with classification of certain coat color mutations. However, Mlw is itself easier to classify than Sl on the MEV (a/a, d/d) background. The new strain breeds as well or better than the original MEV/1Ty-Hm Sl Cja strain.
it is anticipated that the stock is still segregating in some regions of the genome, but we have confirmed that the stock is homozygous for each of the \emv loci present in MEV/1Ty, with the exception of \emv-13, which has been deliberately eliminated. When the new stock is more inbred, we will characterize the stock with respect to common isozyme polymorphisms.

All three of the new markers have been backcrossed to the MEV stock at least 10 generations, and three or more of the last backcrosses were to the inbred MEV/1Ty subline. It is our intent to maintain the new stock segregating for \ps, Miwh, and CaJ, but fixed for at. This stock has proviral or visible markers on 13 of the 19 autosomes and is estimated to sweep 66% of the autosomal genome in a backcross scored for 50 fully informative gametes. This is an improvement over the ~50% coverage provided by the original MEV/1Ty-Hm SI CaJ stock. The new stock is designated MEV/2Ty-at \ps Miwh CaJ. The original stock will continue to be available, at least temporarily.

Mice from the new stock can be obtained from The Jackson Laboratory. We are continuing to map novel proviruses which were discovered in the MEV stock and have been propagated to fixation. We hope to release in the near future a new MEV stock bearing additional proviruses that map to different chromosomes.

Acknowledgments

Mutant mouse stocks were obtained from the Mouse Mutant Resource. This work was supported by NIH grant CA33093.

REFERENCES

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