IMPORTANT

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Editor: Dr. Antony G. Searle, M.R.C. Radiobiology Unit, Harwell, Oxon. OX11 ORD, England. Next deadline: December 15th

Editor of Mouse Membrane Alloantigen News:

Dr. Peter Démant, The Netherlands Cancer Institute, Sarphatistraat 108, AMSTERDAM-C, The Netherlands. Next deadline: December 1st

Business Manager:

Dr. Michael Festing, M.R.C. Laboratory Animals Centre, Woodmansterne Road, Carshalton, Surrey, SM5 4EF, England.

Distributed by:


N.B. The supplement to Mouse News Letter (Subject-Strain Bibliography Listing) is compiled by Joan Staats and distributed by the Jackson Laboratory. "Inbred Strains of Mice" (companion issue to every fourth Mouse News Letter) is edited by Joan Staats and also distributed by the Jackson Laboratory.

INFORMATION ON HOW TO OBTAIN MOUSE NEWS LETTER, WITH SUBSCRIPTION FORM, IS ON THE LAST PAGE.

MOUSE NEWS LETTER is sponsored by the International Committee for Standardized Genetic Nomenclature for Mice, which is affiliated with the International Committee on Laboratory Animals.
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Subscription increase

Inflation has caught up with us at last and we are compelled to increase our subscription rates for next year to avoid running down steeply into the red. The new rates will be £5 or $12 per annum. Higher subscriptions, more subscribers... what about more contributors of news?

MAMMALIAN GENETICS GROUP

The next meeting of the Mammalian Genetics Group will be held at the Royal Free Hospital Medical School, 8 Hunter Street, London W.C.1 on 26th and 27th November, 1980 (just before the Biochemical Genetics meeting). Will all those who would like to present a paper or just attend please get in touch with Dr. M.F. Lyon, MRC Radiobiology Unit, Harwell, Didcot, Oxon., or Dr. M. Festing, MRC Laboratory Animals Centre, Carshalton.

SEVENTH MAMMALIAN BIOCHEMICAL WORKSHOP

This will be held at the Royal Free Medical School, 8 Hunter Street, London on the 27th and 28th November (just after the Mammalian Genetics Group meeting). The workshop is an informal meeting of geneticists, biochemists, cell biologists and physiologists to consider problems of mutual interest; includes laboratory animals, tissue culture and man. Programme organiser and all enquiries to: Dr. G. Bulfield, Department of Genetics, The University, Leicester, LE1 7RH, U.K.

UNESCO-ICLAS WORKSHOP ON EMBRYO STORAGE AND BANKING IN LABORATORY ANIMALS

With financial assistance from UNESCO, ICLAS and other organisations a workshop to discuss embryo freezing and storage was held at MRC Radiobiology Unit, Harwell on 6-9 May 1980. The full proceedings will be published by Gustav Fischer, Stuttgart, editor G. Zeilmaker. The following is a brief summary of the discussions.

Various methods of freezing embryos are now available. Each method has its own advantages and limitations, and works well in at least some laboratories. Different thawing techniques are appropriate to embryos frozen by the various methods. In some cases success is limited by the difficulties of collecting sufficient embryos (due to poor response to superovulation), or of embryo transfer. If all laboratories achieved results equal to the best obtained, then 60-70% of embryos frozen could be recovered as live young at birth. In practice most laboratories achieve low success rates. No deterioration in viability with duration of storage has yet been detected, over a period of 5 years storage.

Frozen storage is valuable in several ways.

1. As a safety device when stocks are also being maintained conventionally, a bank of frozen embryos protects against loss of the stock by disease or accident.
2. Again when stocks are also being conventionally maintained, a frozen embryo bank protects against genetic change in the stock due either to contamination or to mutation.
3. Rarely used stocks, which might otherwise be discarded, may be preserved.
4. If stocks are transferred to other laboratories as frozen embryos, there is less risk of introducing disease.
5. Depending upon the quarantine requirements, it may be more economic to transfer stocks as frozen embryos than conventionally.

The members of the workshop agreed on the following recommendations.

1. No one specific method of freezing and thawing could be recommended at present.
Rather workers should use whichever method works best in their own laboratory.

(2) When embryos are stored detailed information should be recorded (a) on the method of freezing, and the most appropriate method of thawing, including the culture media to be used, and (b) on the genetics of the stocks and appropriate methods of conventional breeding.

This information is essential, first, for the successful transfer of stored embryos to other laboratories, and second for prolonged storage (many years) in the same laboratory, as the original investigator may be no longer present.

(3) Information on stocks available in the frozen state should be disseminated as widely as possible, in sources such as *Mouse News Letter* and Inbred Strains of Mice which also record conventional stocks, so that maximum value may be obtained.

Contributors to MNL and ISM are urged to record their stored stocks.

M. F. Lyon

**STOCK EXCHANGE**

OXFORD PATHOLOGY would like to find homes for two inbred stocks carrying the Velvet coat mutation, C57BL/6/By-Ve and 129/J-Ve Kl. These were imported from France in 1976 and are the only representatives of this mutant in Britain, as far as we know.

V. Papaioannou

**NOTES FOR THE GUIDANCE OF CONTRIBUTORS**

1. Please give your information in the standard format as far as possible, preferably with use of the following headings:-

   Change of address, Personnel news, Stock exchange (stocks offered or wanted), Inbred strains (only additions, subtractions, new strain or substrain symbols), Mutants added, Mutants discarded, New mutants, Linkage data, Research news, Work in progress, Corrections. Lists of inbred strains should not be given, as they are catered for by "Inbred Strains of Mice" produced every two years by Joan Staats of the Jackson Laboratory. However, new contributors can give concise lists of inbred strains in their first contribution.

2. In the staff list, please underline the name of whoever sent in the contribution.

3. Lists of mutants and mutant stocks should normally only be given in a "Consolidated Issue" which appears every 3 years or so and incorporates this information as well as research news etc. The following extra headings are suggested (after inbred strains):

   Mutant strains and stocks, Mutants, Chromosome anomalies.

4. Please don't underline gene symbols. Genotypes can be given as the bb +c etc., or d se/d se if genes are linked.

5. Check any inbred strain, substrain and subline symbols carefully to see that you have used the standard designation (see Cancer Res., 36: 4333 and the rules in MNL 61).

6. For reproduction without transcription, please use an electric typewriter, single spacing and about 7½ in. (19 cm.) width of type.

7. The following abbreviations are recommended:

   BC, backcross; IC, intercross; Cen, centromere; Chr, chromosome; IP, intraperitoneal; LG, linkage group; MI, metaphase I of meiosis; MII, metaphase II; RF, recombination frequency. For chromosome anomaly symbols, see rules in MNL 61.

8. Research news in MNL are now quotable, like items in a published paper, unless the author indicates otherwise, e.g. by inserting "(non-quotable without permission)." References for quotes should follow those for published items, except that "Private communication" should be used instead of the paper's title.
### NEW GENE SYMBOLS

The following new gene symbols are proposed in MNL 63 or in recent publications:

<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Gene Symbol</th>
<th>Gene Name</th>
<th>Contributor or Reference</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Epa</td>
<td>Epidermal antigen</td>
<td>Mayo Clinic</td>
</tr>
<tr>
<td>8</td>
<td>Es-16</td>
<td>Esterase-16</td>
<td>Universität Freiburg</td>
</tr>
<tr>
<td>17</td>
<td>het</td>
<td>Head-tilt</td>
<td>Jackson</td>
</tr>
<tr>
<td>16</td>
<td>Ifrc</td>
<td>Interferon receptor*</td>
<td>Harwell</td>
</tr>
<tr>
<td></td>
<td>stu</td>
<td>Stumbler</td>
<td>Harwell and Womack (pers. comm.)</td>
</tr>
</tbody>
</table>

* Non-standard symbol IfRec used in publication; present symbol suggested instead (Ed.)

### CHANGED GENE SYMBOLS

<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Gene Symbol</th>
<th>Symbol Changed To</th>
<th>Contributor or Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>hbs</td>
<td>un\textsuperscript{ex}(undulated-extensive)</td>
<td>Cambridge</td>
</tr>
<tr>
<td>2</td>
<td>wt</td>
<td>un\textsuperscript{m}(undulated-minimal)</td>
<td>Cambridge</td>
</tr>
<tr>
<td></td>
<td>pet</td>
<td>hyt(hypothyroid)</td>
<td>Jackson Laboratory</td>
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</table>

### NEW LINKAGE, SYNTENY AND ALLELISM DATA

<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Loci</th>
<th>Contributor</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Sdh-1...pa...a...bp</td>
<td>Bruns et al. (Harwell)</td>
</tr>
<tr>
<td>6</td>
<td>(Gapd)</td>
<td>Otto (Univ. Freiburg)</td>
</tr>
<tr>
<td>7</td>
<td>Tam-1...Prt-4......Prt-5</td>
<td>Peters et al. (Harwell)</td>
</tr>
<tr>
<td>10</td>
<td>v.....dl....Hk-1.....gr.....S1\textsuperscript{con}</td>
<td>Crocker and Cattanach (1979)</td>
</tr>
<tr>
<td>10</td>
<td>S1k allelic with dl = Dl\textsuperscript{a}S1k</td>
<td>Genet. Res. 34: 231</td>
</tr>
<tr>
<td>12</td>
<td>sm</td>
<td>Hawes et al. (Jackson Laboratory)</td>
</tr>
<tr>
<td>15</td>
<td>van</td>
<td>Wallace et al. (Cambridge)</td>
</tr>
<tr>
<td>17</td>
<td>T</td>
<td>Sweet (Jackson Laboratory)</td>
</tr>
<tr>
<td></td>
<td>mld allele with shi = shi\textsuperscript{mld}</td>
<td>Cowen (Harvard Medical School)</td>
</tr>
</tbody>
</table>

### NEW SUBSTRAIN SYMBOL

Tu

Max-Planck-Institut für Biologie
Abt. Immungenetik
7400 Tübingen 1, W. Germany
(Dr. Jan Klein)
Solid vertical bars depict the relative cytological lengths of the chromosome based on an estimated total haploid length of 1600cM including the X chromosome. When observed genetic lengths exceed expected lengths the chromosomal bars are extended by hatched lines. Centromeres are represented by knobs. Nucleolus organizers are symbolized by NO. The numbers to the left of the chromosomes are recombination percentages. Distances between centromeres and proximal markers determined by using Robertsonian chromosomes are enclosed in parentheses because these distances may be underestimated. Genes proved to be syntenic by parapsychic methods are listed at the bottom of their respective chromosomes.

Some conventions have been used to indicate the relative certainty of the position of a locus. The best known are depicted by lines extending to the left of the chromosome beyond others. The positions of most loci are shown by a simple symmetrical line across the chromosome. When the position of a locus is known only with respect to an adjacent locus the line is drawn to but not through the chromosome. When a locus is known to be close to another but no recombination value has been given, it is placed next to the linked locus but no line is drawn to the chromosome. Loci whose position is uncertain because they have been tested with only one other locus, and may show appreciable recombination with it, are not italicized and the locus with which they have been tested is in parenthesis. Brackets enclose loci, some of which have been tested together, for which the order is not known.
This is a revision of that given in MNL 61: 20. It shows approximate positions of breakpoints in inversions (In) and translocations (T), obtained from the data given in Mouse News Letter or published elsewhere. The symbols for these are on the right of the chromosome concerned, while symbols for some key marker loci (especially end markers) are given on the left. Positions given are often a compromise between those obtained by genetic and by cytological methods. Loci etc. at the distal end are not always the correct map distance from their neighbours because of scaling problems, shown by a distal break in the chromosome line. A proximal break indicates that the distance from centromere to nearest locus is not known yet. Robertsonian translocations include all those available as separate stocks.
1. New recombinant haplotypes

<table>
<thead>
<tr>
<th>Haplotype</th>
<th>Origin</th>
<th>Composition</th>
<th>Strain</th>
<th>Reported by</th>
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<tr>
<td>h10</td>
<td>k/b</td>
<td>KAESD</td>
<td>B10.RKB</td>
<td>C. S. David</td>
</tr>
<tr>
<td>at9</td>
<td>h4/t4</td>
<td>KAESD</td>
<td>B10.RSB-1</td>
<td>C. S. David</td>
</tr>
<tr>
<td>a4</td>
<td>h2/h3</td>
<td>KAESD</td>
<td>B10.RKD-1</td>
<td>C. S. David</td>
</tr>
<tr>
<td>12</td>
<td>t6/b</td>
<td>KAESD</td>
<td>B10.RBD</td>
<td>C. S. David</td>
</tr>
<tr>
<td>at10</td>
<td>t4/t4</td>
<td>KAESD</td>
<td>B10.RSB-2</td>
<td>C. S. David</td>
</tr>
<tr>
<td>at11</td>
<td>t3/h2</td>
<td>KAESD</td>
<td>B10.RSB-3</td>
<td>C. S. David</td>
</tr>
<tr>
<td>a5</td>
<td>a1/19</td>
<td>KAESD</td>
<td>AIR1</td>
<td>H. C. Passmore</td>
</tr>
<tr>
<td>g7</td>
<td></td>
<td>KD</td>
<td>BDR7</td>
<td>J. Klein, F. Figueroa</td>
</tr>
</tbody>
</table>

2. New haplotypes

H-2 The STU mouse inbred line obtained from Prof. Dr. W. Schafer, Max Planck Institut f. Virusforschung, Tubingen, carries an H-2 haplotype, H-2, which is a natural recombinant carrying the K region (it is H-2.31 positive) and expressing the private I-A specificity Ia.2 together with Ia.15, 19 and the I-E region specificities Ia.7,32. It is Ss high and expresses the private specificity H-2.118 of the D region of B10.SA48.

(D. Gotze, E. K. Wakeland, J. Klein)

H-2 The T02 mice obtained from Dr. Leslie Brent, St. Mary's Hospital Medical School, London, carry a new H-2 haplotype which was given designation H-2. The T01 mice carry a recombinant haplotype with an unknown K region allele and D.

H-2 is the haplotype of the WLA76 strain developed by Dr. J. L. Guenet, Institut Pasteur, Paris.

H-2 is the haplotype of the strain WPA 76 derived by Dr. J. L. Guenet, Institut Pasteur, Paris.

H-2 (see above) was found segregating in the THF strain (thin fur). It is maintained in homozygous state in a line designated BDR7. The line is positive for Ia antigens m3, m4, m7 and m11 (nomenclature of Klein and Hammerling, Immunogenetics 1979).

(H.-J. S. Huang, F. Figueroa, J. Klein)

H-2 and H-2 are symbols assigned for strains B10.F(13R) (K D) and B10.F(14R) (K D), respectively (C. S. David).
3. **H-2 specificities**

Private class I antigens of NZW. We transferred the **H-2** haplotype of NZW on the NZW on the C57BL/10Sn background and so produced a B10.NZW congenic line. The private K-region antigen of **H-2** is **H-2.20** (shared with **H-2** of B10.PL); the private D-region antigen is **H-2.1.14** (shared with **H-2** of B10.STC77). Thus, the **H-2** is a natural recombinant related to the **H-2** and **H-2** haplotypes. The B10.NZW line carries Ia antigens 7, 15, and 32, and types as SshSlp. (H.-J. S. Huang, F. Figueroa, J. Klein).

4. **I region**

The suggestion of an **I-H** locus (and subregion) mapping between **I-B** and **I-C** in the **H-2** complex (NL 54:27, 1976) was based upon a secondary **k** proliferative response of human lymphocytes primed with **H-2 k** and re-stimulated with B10.A(3R) or B10.A(5R) lymphocytes (Fol. Biol. (Praga) 22: 376, 1976). Recently, a cytotoxic response of the same specificity was obtained with A.TH anti-A.TL or B10.A(2R) anti-B10.A(4R) effector **kk** cells, and it was shown to be due to a crossreaction of **kE F** with **kE F**. Thus, there is no need to postulate a separate locus **I-H** to account for the restimulation of the human lymphocytes.

(K. F. Lindahl)

5. **Ia specificities**

**Ia(m)46**. This specificity is present on T lymphocytes and is defined using either (A.TH x B10.Htt)F anti-A.TL serum or (B10 x B10.D2)F anti-B10.A(5R) serum on B10.A(3R) lymphocytes. It is different from Iat.7(I-J), Ia.7(I-E) and other known specificities mapping to the right of I-J.

(C. E. Hayes)

**Ia(m)47**. It is a T lymphocyte marker mapping between **H-2K** and **I-B** defined by (A x B10.A)F anti-B10.A(5R) serum and present on B10.D2 but not B10.A(2R), B10.A(4R) and LO/Ckc (H-2K) T lymphocytes.

(J. M. D. Plate)
Mta, a new target antigen for unrestricted T cell killing, has been
defined by NZB T lymphocytes, immunized and restimulated in culture with
BALB/c spleen cells. These effector cells kill target cells of nearly
all mouse strains tested (except most NZB substrains), irrespective of
their H-2 or Qed-1 type. Mta is present on normal and mitogen-stimu-
lated T and B lymphocytes and on several tumor lines, e.g. P815, EL4 and
YAC-1. Typing of the NX8 recombinant inbred lines (derived from Mta−
NZB/Icr and Mta+ C58/J parents) suggested that Mta is maternally trans-
mitted. This was confirmed by typing reciprocal F1 hybrids and back-
crosses between positive and negative strains: Mta+ females bear Mta+
offspring and Mta− females Mta− offspring, irrespective of the genotype
of the males. Either all mice have the genetic information to produce
Mta, and there is an epigenetic transmission from negative mothers of a
principle preventing expression of Mta for the lifetime of their offspring,
or Mta is encoded by a maternally transmitted genetic element, likely to
be a virus. Foster nursing experiments suggest that Mta is not milk-
transmitted.

The following strains have been typed:

Mta− - NMRI/Bom, NZB/B1NJ, NZB/CrBom, NZB/Hs, NZB/Icr.
B10.M, B10.Y, CBA/J, C3H/HeJ, C3H/Tif, C3H.SW, C57BL/6, C57BL/10ScCr,
C57L/J, C58/J, DBA/2, LP/J, NZB/B1Pt (and its derivative, NZB/Full,
bred in Füllinsdorf), PL/J, RI11S/J, SJL, SWR.

(K. F. Lindahl, M. Boechieri, R. Riblet)
BRIGHT WOODS BREEDERS

Bright Woods, Route 45
Cornwall Bridge, CT  06745
U.S.A.  

Received:  May 22nd 1980

Lisa Denise Wojan
Phyllis H. Wojan

An introduction, since this is our first submission to Mouse News

Bright Woods Breeders is a small mouse colony maintained since
1967 as an avocation for observation of mouse behavior and experimenta-
tion in nutrition, husbandry, environment, and color genetics.

Started with 3 Jackson mice (a/a and a/a b/b), the colony has
had several additions, all of whose breeding origins were unknown:
2 albinos (c/c with, possibly, p/p or ru/ru) in 1968, 2 wild-type
agoutis (A/A with, possibly, p/p or ru/ru) in 1969, 1 black with
pale belly (a/x/a?) in 1971, 3 Himalayans (c*h/c*h), and 6 piebalds
(s/s? a/a and s/s? A/A) in 1972. By 1973, the colony included 5
phenotypes with black eyes: black, brown, wild type agouti, dark
yellow agouti ("cinnamon" in the mouse fancy), and piebald mice in
the four preceding colors with white. We also had 4 phenotypes with
very dark red eyes: white, "dove" grey (a/a p/p?), "warm, pale"
beige (a/a b/b p/p?), and yellow agouti with both "deep orange" and
"pale golden" coats (A*vy/A*vy p/p?). Piebald phenotypes with dilute
pigmentation did not survive beyond 12 weeks.

In 1974, 18 Jackson mice (6 BALB/cJ, 6 C57BL/6J, 6 03H/HeJ)
were added and interbred with colony mice. Because of the colony's
small size, all mice were out-crossed as much as possible from 1967
to 1978. By 1978, all s?, c*h, and p? phenotypes had been lost in
the colony. In mid-1978, Priscilla W. Lane of Jackson Laboratory
was kind enough to encourage us to go back into our breeding records
to try to retrieve the recessive genes and to start to inbreed by
colour. We were successful in re-establishing the three dilute
pigmentation coat colors and approximately 45% of the colony is now
dilute pigmentation phenotypes. The c*h and s? phenotypes were not
recovered.

Stocks held

We have been inbreeding, sibling to sibling, by coat color since
July 1978 and have reached the following generations in establishing
inbred strains:

Wild type agouti (A/A)  P10
Dark yellow agouti (A*vy/A*vy)   P10
Yellow agouti, dilute (A*vy/A*vy p/p?)  P6
Black (a/a)       P8
"Dove" grey (a/a p/p?)       P6
Brown (a/a b/b)    P10
"Warm, pale" beige (a/a b/b p/p?) P5

We also keep a small stock of random-bred c/c.

Records kept for each mouse include birth date, parents, color,
cause, if known, and date of death; for litters we record number of
live pups, color phenotypes, number dying before weaning. Any unusual
behavioral or physical characteristics also are noted.
Past research (unpublished)

Fructose as a means of modifying duration and severity of epileptic seizures in an a/a b/b male mouse. 1968.

Mouse preferences among wild Northeastern grasses for use as food and/or nest material. 1969.

Leaves, blossoms, and fruits of wild Northeastern plants accepted as food by Mus musculus domesticus. 1970.

Predicting the weather: 3 studies correlating mouse behavior patterns and weather. 1971 and 1972.


Learning patterns in maze running: a comparison of 7 Mus musculus domesticus with 7 Rattus rattus raised in similar environments. 1976 and 1977

Current research interests, 1978-1980

We are interested in the effects of diet on litter size, reproductive span, longevity, and obesity. At present, on our own experimental diet, mean litter size is 11.7 live young; does produce large healthy litters up to the age of 22 months, and less than 2% of their litters are lost before weaning at four weeks. Obesity runs about 15% in our A*vy and A*vy p? mice, and less than 1% in all other coat colors.

Ongoing interests in the observation of social behavior include parental behavior, learning patterns of young mice, the functions of exercise/play devices, and dominance patterns.

And a request

Since we are mouse-keeping amateurs in the true sense of the word, we would be grateful for any corrections or suggestions relating to our use of terminology and gene symbols, and to our reporting procedure. We especially hope that someone will be kind enough to correct, confirm, or comment upon the appropriateness of our gene symbols for color, and give us the names of the dilute pigmentation coat colors. We welcome any correspondence and suggestions.

***************

-7-
BRISTOL UNIVERSITY

Department of Physiology,
University of Bristol Medical School,
University Walk,
BRISTOL BS8 1TD,
England.

Received: June 13th 1980

Research News:

A genetic study has been carried out on a C57BL colony manifesting the dt<sup>J</sup> dystonic mutant gene (autosomal recessive). The colony (C57BL/6J-dt<sup>J</sup>) was maintained in the Department of Physiology, Bristol University, and was bred from three pairs derived from a mutation arising at the Jackson Laboratories, Bar Harbor. The results were compared with those obtained by Duchen, L.W., Strich, S.J. and Falconer, D.S. (Brain 87: 367-378, 1964), for an allelic dystonic mutation which initially arose at the Institute of Animal Genetics, Edinburgh (dt). Unlike the colony in that study (C57BL/Fa-dt), the Bristol C57BL/6J-dt<sup>J</sup> colony showed an incidence of affected mice that was markedly lower than the expected value of 25% for an autosomal recessive condition. Moreover, the pre-weaning mortality was considerably higher in the offspring of the proven pairs than in those of the unproven pairs (see table).

Genetics of the Edinburgh and Bar Harbor derived dystonic mutants

<table>
<thead>
<tr>
<th>Proven Pairs</th>
<th>Surviving Normal Offspring</th>
<th>Mutants No.</th>
<th>Pre-weaning Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C57BL/Fa-dt</td>
<td>20</td>
<td>545</td>
<td>170</td>
</tr>
<tr>
<td>(Duchen et al., 1964)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C57BL/6J-dt&lt;sup&gt;J&lt;/sup&gt;</td>
<td>24</td>
<td>364</td>
<td>73</td>
</tr>
<tr>
<td>(Present study)</td>
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</table>

The generally high values obtained for pre-weaning mortality, relative to the C57BL/Fa-dt colony values, were probably caused by a large number of runted animals a problem arising from the maintenance of a large colony originating from very few breeding pairs. The present observations indicate that in contrast to Edinburgh derived dystonic mutants (dt/dt), an appreciable proportion of Bar Harbor derived dystonic mutants (dt<sup>-</sup>/dt<sup>+</sup>) die before their diagnosis age (10-14 days). Thus although the dt and dt<sup>-</sup> genes are known to be allelic (Duchen et al., 1964), they may not be identical, with the dt<sup>J</sup> gene manifesting as a more severe form of the disease. This suggestion is also consistent with the relatively short survival time of dt<sup>-</sup>/dt<sup>+</sup> mutants; no affected animals lived longer than twenty-one days in the Bristol C57BL/6J-dt<sup>J</sup> colony, whereas dt/dt mutants have survived for several months (Duchen et al., 1964).

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THE UNIVERSITY OF BRITISH COLUMBIA

Department of Medical Genetics,
The University of British Columbia,
Vancouver, B.C. V6T 1W5.
Canada.

Received: May 29th 1980

Research News:

far: The first paper on the first arch malformation, giving evidence for its inheritance as a single autosomal recessive gene (far) and describing its skeletal development from day 15 of gestation to birth (using alcian blue and alizarin red S staining of whole specimens), has been accepted by the J. of Heredity and is scheduled to appear in the fall of 1980. A histological study of the craniofacial development of this mutant is now underway. (Jean McLeod).
Four alleles at the \textit{lid} \textit{gap} \textit{(lg)} locus have been referred to in the literature:

\textit{lg} \textit{(lid gap)} \quad \text{(Ricardo and Miller, Can. J. Genet. Cytol. 9:596, 1967)}
\textit{lg}^{M1} \text{(lid gap-Miller)} \quad \text{"} \quad \text{"} \quad \text{"} \quad \text{"} \quad \text{"} \quad \text{"} \quad \text{"}
\textit{lg}^{St} \text{(lid gap-Strong)} \quad \text{"} \quad \text{"} \quad \text{"} \quad \text{"} \quad \text{"} \quad \text{"} \quad \text{"}
\textit{lg}^{Stn} \text{(lid gap-Stein)} \text{(Stein and Kettyle, Teratology 8:51, 1973)}

However, only \textit{lg} has been listed in the MNL. At present, \textit{lg}, \textit{lg}^{M1}, and \textit{lg}^{Stn} are maintained in this laboratory. Some years ago, \textit{lg}^{St} stock died out and this allele is presumed extinct. It is suggested that \textit{lg}^{M1} and \textit{lg}^{Stn} be added to the gene listing.

Each of the \textit{lg} alleles has arisen in a different strain. In order to standardize genetic background for developmental studies, attempts have been made during the last 15 years to transfer all the \textit{lg} alleles to a common, strong, inbred background strain (SWV, CBA and ICR strains have been used at various times), using a backcross-intercross mating system. Because most of these attempts produced such a low frequency of affected animals from intercross matings that the mutant could not be recovered to continue into the subsequent backcross, the attempts were abandoned after several generations of struggle. However, when affected mice did appear and survived to breed with other affected mice, they produced close to 100\% affected offspring, i.e. the trait could be fixed rapidly, suggesting a relatively simple genetic mechanism. Each outcross (of a given allele to a given strain) was reasonably consistent within itself, e.g. proportions in successive intercrosses were similar. The responses differed in outcrosses of the same allele to different strains, suggesting strain differences in modifier or suppressor genes.

At present, we are testing the hypothesis, derived from these data, that a single autosomal recessive morphological gene causes \textit{lid} \textit{gap}, but that the mutant genotype is not expressed when one or more suppressor genes are present, the particular mechanism depending on both the allele and the strain involved. As the number of proposed suppressor loci increases the test becomes rapidly complex. The first hypothesis being tested, in the cross between \textit{lg} and the ICR strain, involves a single autosomal recessive suppressor. \text{(Muriel Harris).}

\begin{center}
\textbf{CAMBRIDGE}
\end{center}

Department of Genetics
University of Cambridge
Downing Site
Cambridge CB2 3EH
England

Susanna R. Earnshaw
M.J. Evans
Janet M. Ferguson
Margaret E. Wallace

Received: June 9th 1980

\underline{Viable anaemia: is \textit{van} = \textit{mk}?}

Viable anaemia, \textit{van} (MNL 60, 40), is linked with caracul, Ca: 1/24 recombinants. Microcytic anaemia, \textit{mk}, is also close to Ca. A sample of 5 \textit{vanvan} mice (39, 2\textsuperscript{o}) has shown \textit{vanvan} also to be microcytic, and the cell volume pattern is similar to \textit{mk} (Russell, 1979, Adv. in Genet. 20, 357-459). However, \textit{mk} invariably has a high red cell count, which occurred in only 2 of our \textit{van}: and we have not noticed the skin lesions of \textit{mk}. Possibly these two features are a background effect. \text{(Wallace, Ferguson; S Handa and G. Bulfield, Leicester).}
X/Y non-association followed by non-disjunction

During our experiments in which untreated Peru-Coppock mice are hemicastrated to look for chromosome abnormalities at diakinesis/metaphase I, the right testis of a 6th generation mouse showed non-association of X and Y in 23 out of 40 spermatocytes, which is a significantly higher frequency than that found in the rest of the population (6.2%). When mated to several Ta+, his remaining left testis gave 3 phenotypically TaO out of 44 tabby daughters, the three occurring in one litter. Seven sons from this testis showed a normal frequency of non-association. After killing, a sample of 400 spermatocytes from this left testis showed a normal rate of XY non-association (6%), and in G-banded mitotic cells from the bone marrow both X and Y appeared to be normal. The simplest explanation seems to be that an early mitotic mutational event gave rise to clones of spermatocytes giving a high frequency of non-association in both testes; and that the left testis was later repopulated by normal cells. Sons and daughters have been mated, complementarily for Ta, to test whether the tendency to non-association etc. is indeed inherited. Two of the TaO ♀♀ are now breeding, by normal ♂♂, as a test of genotype, and blood will be taken for mitotic karyotypes. (Wallace and Earnshaw; and E.P. Evans, Oxford).

Undulated alleles

Permanent symbols unex and unm, for undulated-extensive and undulated-minimal, have been given for the mutants hunchback syndrome, hbs, and wavy-tail, wt, listed among those arising in the Peru-Coppock mice (NRL 51: 29). (Wallace).

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CINCINNATI

Children's Hospital Research Foundation
Cincinnati, Ohio 45229
U.S.A.

Received: April 28th 1980

Mutant stocks and strains

<table>
<thead>
<tr>
<th>symbol</th>
<th>description</th>
</tr>
</thead>
<tbody>
<tr>
<td>crn, dr, Ds, hpy, oe, Spd, tk, xn (xn received from M.E. Wallace via R.L. Sidman)</td>
<td></td>
</tr>
</tbody>
</table>

Research news

crn and xn produce grossly similar exencephaly. The 25 or so most recent matings between identified heterozygotes of each gave 22.5 and 16.5% affected offspring, respectively (or, assuming mutilated and unascorable ones were abnormal, 25.9 and 18.7%, respectively). Mean litter size was 8.8 and 9.3, respectively. Thus xn is apparently incompletely penetrant, at least in the stock now containing it, as reported by Wallace et al. (1978).

The genes are also different in another respect: >90% of xn/xn's had open eyelid (mostly bilateral); whereas >80% of crn/crn's had closed eyes and in the remainder the defect was usually unilateral. Wallace et al. (1978) show a photograph of a newborn xn/xn with open eyelid, but failed to mention the condition in their article.

Up to this writing 12 +/crn x +/xn matings were made and the pregnant females killed before term. Of 132 implantations, 14 (10.6%) were resorbed, and all were moles or early postimplantation deaths; giving a mean survival rate of 9.8. Of the 118 survivors, 2 (1.7%) had exencephaly, 1 with closed eyes, the other too young to diagnose. The question of allelism is thus still to be decided.

**************

- 10 -
Since my pessimistic report in the last Mouse News Letter of our very low yield of successful recoveries of chimeric mice after injection of aggregated embryos into surrogate pseudopregnant female mice, we have experienced a dramatic turnaround. What look like a minor change in injection technique seems to be responsible for a great increase in successful recoveries (now up to 50 percent!)

The most interesting chimera recovered so far is what is clearly the result of aggregating an embryo homozygous for the l**t** gene with a normal embryo. This mouse shows the severe tibial hemimelia typical of l**t** homozygotes along with some radial hemimelia plus polydactyly on all four limbs. However the radial hemimelia is much less pronounced than in typical l**t** homozygotes. It also appears to be much more vigorous than usual l**t** homozygotes, possible typical of all our chimeras. (Forsthoefel)

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ESSEX

Department of Biology
University of Essex
COLCHESTER CO4 3SQ
England

Received: June 4th 1980

Stocks: We have the C57BL/10ScSn, B10.BR, BALB/cBy and C57BL/6By strains and the CXB recombinant inbred lines together with stocks carrying reduced pigmentation, rp, and satin, sa, mutations.

Research News:

Our interests are in the analysis of quantitative physiological and developmental characters, with particular reference to the endocrine and immune systems and to events in the early embryo.

************

UNIVERSITAT FREIBURG

Abteilung für Chemische Pathologie
Albertstraße 19
D-7800 Freiburg i.Br.
Bundesrepublik Deutschland

Received: June 4th 1980

MUTANT STOCKS: esr, nv, Es-\(\gamma^a\), Es-\(\gamma^d\)
1. Esterase-16, a presumably new locus.

Genetic polymorphism was demonstrated in Peru/Coppock for esterase-16 (Es-16), a high molecular weight carboxylesterase (EC 3.1.1.1) occurring in various tissues (liver, kidney, lung, testis, tongue, not in erythrocytes and in plasma). Esterase-16 is closely linked to Es-6 and Es-9. In the kidney of females, ES-16 is considerably stronger than in males (E. Eisenhardt).

2. Typing of Esterase-9 in different laboratory strains and wild stocks.

Es-9 can be easily typed in kidney, liver, lung, and skeletal muscle using polyacrylamide gel electrophoresis at pH 7.4. Four phenotypes have been distinguished: ES-9A (C57Bl/10Sn, DBA/2J, C3H, A/J); ES-9B (Sk/Cam, M.m. castaneus); ES-9C (M.m.molossinus); ES-9D (Peru/Coppock). The gene order proposed is Es-1 - (Es-9, Es-6) - Es-2 (Th. Wienker).

3. Purification of esterase isozymes.

The following carboxyl esterases (EC 3.1.1.1) have been isolated and highly purified in our laboratory:
1.) Esterase-9A (C57Bl/10Sn);
2.) Esterase-2B (C57Bl/10Sn);
3.) Esterase-1D (M.m.molossinus)

Antisera against these mouse esterases have been raised in rabbits (A. Ronai).

4. Submandibular gland esterproteases.

The recently reported loci, Prt-4 and Prt-5 (MNL 62, page 47) are closely linked to Tam-1 (O/83 recombinations). Using isoelectrofocusing, PRT-4 and PRT-5 can clearly be distinguished from TAM-1 by different isoelectric points, different staining properties and by the different allele distribution on mouse strains (J. Otto).

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Neurological mutant stocks:

bg, cod, cri, dtJ, db, El, gt, jp, jpm,sd, Lc, mea, mh, mh2J, Mblo, Mbr, Mpow, nr, pcd, rd, rl, shi, shimd, sg, shm, spa, stu, syf, tfm, tg, tga.

Tr, TrJ, twi, vb, wv.

We also hold the following markers:

c2J, e2, Ca, d, Esq, gr, Hm, m, Miwh, Os, p, pe, Re, se, Sl, vt, Wv

Research News:

1. Stumbler

A new autosomal recessive mutation, for which we suggest the name stumbler (gene symbol = stu), arose at the Jackson Laboratory in the C3H/HeJ strain, and was provided to us by Eicher. Affected animals are recognized from postnatal day 9 (P9) onward by delay in righting time, and from about P10 by awkward, stumbling gait. On the coisogenic background they die by about P21, but may live somewhat longer in F2 crosses with C57BL/6J. The cerebellum is small but has a normal folial pattern. Total number (but not concentration) of Purkinje and granule cell neurons is reduced by P10. Histological abnormalities in the cerebellar Purkinje neurons include reduced total volume, irregularly increased caliber, and abnormal branching patterns of dendrites, high concentration of normal-looking mitochondria in soma and dendrites, persistent somatic spines and filopodia at least to P21, and focal swellings on axons. These features distinguish stumbler from all other mutants. (Caddy, Sidman)

2. Cerebellar outflow degeneration

The phenotype in cerebellar outflow degeneration, cod (MNL 36:33) has stabilized after the mutation was crossed to the BALB/cSr strain. Affected animals are recognized at P12 by loss of balance when the animal is pushed about and is obvious during unprompted activity by P15. By P45 lethargy, ataxia, nodding of head and trunk, malpositioning of hindlegs, and poor swimming are the outstanding features, and these remain essentially unchanged for the remainder of the normal lifespan. Neither sex is fertile. The earliest recognized pathological change, beginning at about P11, is chromatolysis in vestibular and red nuclei accompanied by degenerative changes in the intermediate and distal portions of their axons, and followed closely by neuron degeneration in deep cerebellar nuclei and brainstem reticular formation. Many additional nuclear groups and axonal systems come to be involved between P25 and P60. Both the distribution and the detailed cytopathological features distinguish this mutant from any previously described. (Sidman)
3. Vibrator, vb, homozygotes show progressive degeneration in selected neurons of spinal cord, beginning before P16, and of brainstem and cerebellum beginning a few days later. The main cytological features are dilated cisternae of cytoplasmic and axoplasmic endoplasmic reticulum with eventual severe intracellular vacuolation and some cell death. Animals in the inbred line die before P30, but outcrossed animals live six months or more and come to show severe cortical atrophy in posterior vermis, and hemispheres of cerebellum, and extensive neuron atrophy and loss in cerebellar nuclei and in intermediate zones of spinal cord gray matter. Intracellular vacuolation of the vibrator type is seen in no other mutant except wobbler, wr, but there the regional distribution of affected neurons is very different. (Weimar, Sidman)

4. New pathological expression has been found in the brain of Purkinje cell degeneration, pcd, homozygotes. In addition to the earlier-described acute loss of cerebellar Purkinje neurons, and slowly progressive loss of olfactory bulb mitral neurons and retinal photoreceptor cells, there is a relatively late but sharply focused loss of several groups of thalamic neurons between P50 and P60. Loss is prominent in mediodorsal, submedial, ventrolateral, reticular, and posterior thalamic nuclei and is virtually complete in the ventral medial geniculate (VMG) nucleus, the main auditory relay station of the thalamus. These are apparently not transneuronal degenerative phenomena, as inferred from Fink-Heimer silver and electron microscopic preparations, but it is not clear what the pcd locus sees in common among the apparently disparate affected populations of neurons. (O'Gorman)

5. Shiverer, shi, and myelin deficient, mld, (kindly supplied by Doolittle) are allelic, the double heterozygote showing an intermediate phenotype compared to the homozygotes. We suggest that mld be designated shimld. (Cowan)

6. We had reported that tottering, tg, homozygotes have epileptic seizures resembling the common human childhood "absence" attacks, and showed autoradiographically altered 14C-labeled 2-deoxyglucose uptake in the brainstem during a seizure attack, compared to the result in control mice (Science 1979 204:1334-1336). We now add that the autoradiographic picture in tg/tg between seizures is indistinguishable from normal. (Noebels, Sidman)

7. We are continuing the electrocorticographic studies of the seizure disorder in tottering and have begun comparable studies in Epilepsy (El), mocha (mh) and other mutants, with special reference to pharmacological parameters. (Heller)

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HARWELL

MRC Radiobiology Unit,
Harwell,
Didcot,
Oxon,
OX11 ORD,
U.K.

Received: June 24th 1980

Research news

1. Fertility of reciprocal and Robertsonian translocations sharing a common arm.

Further to information given in MNL 57: 18, the following male double heterozygotes were fertile in the trans position: T(2;4)13H/Rb(2.18)6Rma; T(2;8)26H/Rb(2.18)6Rma; T(7;15)9H/Rb(4.15)4Rma. (Beechey)

2. A new Robertsonian Translocation

A new Robertsonian translocation has been found in two sibs which were F1 hybrids of the C3H/HeH - 101/H stock. The chromosomes involved were found by G-banding to be 2 and 17 and this has been verified by genetic tests. The Robertsonian has therefore been designated Rb(2.17)4H. (Crocker)
3. Sxr; X-Y separation and spermatogenic impairment

XY, Sxr male mice may show high frequencies of diakinesis cells in which the X and Y chromosomes lie apart (Winsor et al., Cytogenet. Cell Genet. 21: 11-18, 1978). These mice also show some degree of spermatogenic impairment as indicated by reduced testis size. (Cattanach, in The Early Development of Mammals, ed. Balls and Wild. Cambridge Univ. Press, pp. 305-317, 1975). Two series of investigations now indicate that spermatogenic impairment (testis weight) shows a negative correlation with frequency of X-Y separation (r = −0.66 and 0.78). It is not clear how these two parameters are related but reduced testis weight cannot simply be attributed to death of cells which show a lack of X-Y association. Thus, the frequency of the latter event may be as high as 80-90% (mean 75.1 ± 2.7%, cf. 9.1 ± 1.6% for the non-Sxr XY sibs) while testis weight (at 12 weeks of age) is generally reduced by less than one-half (mean = 76.0 ± 4.4mg, cf. 134.8 ± 5.3mg for the non-Sxr sibs) and sterility in these mice is a rare phenomenon. A high frequency of X-Y separation appears to be a more reliable indicator of the XY, Sxr/+ genotype than testis weight reduction. (Cattanach, Crocker and Jones)

4. Expression of dominant genes in trisomic mice

a) Extra toes (Xt) on Chromosome 13.

Female mice heterozygous for both Rb(11.13)4Bnr and Rb(6.13)1H were mated to males heterozygous for Xt. This mating system was designed to produce animals trisomic for chromosome 13 and carrying a single dose of Xt. The trisomic embryos were found to die shortly before birth and therefore to investigate living embryos the females were opened at a range of times from 14 to 18 days post coitum. Developmentally retarded embryos were examined cytologically and approximately 80% of these were found to be trisomic. These trisomic mice, together with normal controls, matched in developmental age were classified for Xt. 15 of the controls proved to be Xt and 18 were +. This close agreement with a 50:50 expectation suggests that Xt is visible at this stage of development. None of the 27 trisomic embryos were classified unequivocally as Xt. However two animals had abnormalities of the hind limbs: one had three fused toes on one hind foot and the other had an extra digit on the outer side of one hind foot which is not typical of Xt. These defects may have been a consequence of the trisomy although an Xt effect cannot be ruled out. It may be concluded that Xt has no effect in the presence of two wild type alleles.

b) Sleek (Dls1k) on Chromosome 10.

Dls1k/Dls1k mice of both sexes were mated to viable aneuploids produced from T199H which are trisomic for the proximal part of chromosome 10 including the dl locus. The aneuploid offspring were recognised by characteristic retardation of development and blunt head shape. These mice should all be of the genotype Dls1k+/+. Although only five such offspring have been obtained because of their low viability and the low fertility of the aneuploid parents, they all showed a similar phenotype which macroscopically resembled Dls1k/+, but with thicker coat and atypically hairy tails. The secondary vibrissa counts were less reduced than Dls1k/+ and under the microscope the body hair resembled that of Dls1k/+ animals with Dls1k still exerting a strong effect in the presence of two + alleles. This phenotypic effect is compatible with a multimeric mode of gene action (Crocker and Cattanach, 1979) but not with simple action of dose nor competitive inhibition by a regulator molecule. (Crocker)

5. Biochemical variants

a) Hexokinase

A genetically determined electrophoretic variant of hexokinase-1 has been found in some mice of the Peru/Coppock stock. This variant has less anodal electrophoretic mobility than hexokinase-1 from all other inbred strains and stocks tested. Genetic analyses indicate that the variant is determined by an autosomal codominant gene. We propose that the common allele is designated Hk-1 and the variant allele found in Peru/Coppock mice Hk-1'.
The electrophoretic mobility of hexokinase-1 appears to be modified in testis where it has a faster anodal mobility than in kidney extracts. In addition a sperm specific hexokinase is also found. These effects are only found in testes where spermatogenesis is unimpaired since in males heterozygous for T(1;7)40H and T(11;19)42H which do not form spermatozoa (Searle, A.G. et al., 1978. Ann. Biol. Anim. Bioch. Biophys. 18: 391) and in males under six weeks of age the electrophoretic mobility of hexokinase-1 is unmodified in testis and sperm specific hexokinase is not found. In heterozygotes for T(5;12)31H where reduced numbers of spermatozoa are found there is reduced activity of sperm specific hexokinase and both the modified and unmodified forms of hexokinase-1 occur in testis. (Peters and Andrews)

b) Sialidase (Neuraminidase)

We have previously shown (MNL 62:52) that Aglp, α-glucosidase processing, is on chromosome 17, closely linked to, and possibly identical with, Apl. The variant allele Aglp<sup>+</sup> is only found in SM/J. In addition, low levels of sialidase (N-acetyl neuraminic acid hydrolase EC 3.2.1.18), often called neuraminidase, are found in SM/J. From quantitative studies of neuraminidase levels in (C3H/HeH x SM/J)F<sub>1</sub> and in mice resulting from the backcross (C3H/HeH x SM/J) it appears that the low levels of this enzyme found in SM/J are determined by an autosomal codominant gene. The symbol Neu-1 has been proposed (Womack, per. comm). Furthermore, our data, though limited, from classifying 16 mice from this backcross, suggests that Neu-1 is on chromosome 17 since no recombinants have been found between Neu-1, Apl and Aglp. These results agree with those of Womack and Potier (MNL 61: 64). It seems probable that the several processing genes Aglp, Apl and Map-2 which are all closely linked on chromosome 17 are one and the same gene, Neu-1. (Peters and D.M. Swallow (MRC Human Biochemical Genetics Unit)).

c) Acid phosphatase

Electrophoretic studies of acid phosphatase at different stages of pre- and post-natal development have revealed the presence of a new isozyme in liver extracts. This new isozyme is detectable in seventeen day old embryos, rises to a peak of activity at around birth and disappears by fourteen days after birth. It has been found in the two strains tested so far, SM/J and C3H/HeH. Work is continuing on this new acid phosphatase. (D.M. Swallow (MRC Human Biochemical Genetics Unit) and Peters).

6. Resorption of bone

Microphthalmic (mini) mice are osteopetrotic with defective osteoclasts. Using stock derived (1955) from Grüneberg and now inbred, we have shown by T<sub>6</sub> marker that syngeneic or H-2 compatible grafts of bone marrow correct the deficiency (Nisbet, Menage and Loutit, Transplantation, 28: 285. 1979).

By means of H-2 compatible grafts of bgbg marrow it was shown in radiation chimaeras that the marker of bgbg, giant lysosomes, appear in osteoclasts of cured mini (Ash, Loutit and Townsend, Nature 283: 669. 1980). In reverse chimaeras (mini spleen given to X-irradiated bgbg) the marker ultimately disappears (Ash et al.. in press). Additional such data should give an estimate of the lifespan of osteoclasts.

From morphometry of bones of reverse syngeneic chimaeras (mini spleen to lethally irradiated normal mice) it appears from preliminary studies that resorption of mature cortical bone as well as primitive trabecular bone is affected by the osteoclastic defect. (Green Howells and Loutit)

7. Haematopoietic stem cells

It is argued that the derivation of osteoclasts is from macrophages derived through monocytes from haematopoietic stem cells (Loutit and Nisbet, Lancet, ii, 26. 1979).

We have confirmed the observation of Harrison that bone marrow from mice doubly mutant at the W locus is capable of curing the radiation syndrome though deficient in CFUs. Marrow from Harwell W<sup>W</sup> mice also cures the osteopetrosis of lethally irradiated mimi mice. In such mice the marrow itself is chimaerical, spared haematopoietic stem cells of the host usually produce the erythrocytes, those of the donor the osteoclastic cells. It is argued that CFUs are but a subset of haematopoietic stem cells controlled by the W locus. (Loutit, Peters and Marshall)
8. Tumour Surveillance

Beige mice are reported as deficient in natural killer (NK) cells. Bbgb mice inbred from a Harwell translocation stock based on (C3H x 101)F1, have a substantial incidence of lymphosarcoma, though the parental stocks and the F1 hybrid are not susceptible (Loutit, Nature Correspondence 285: 661. 1980). (Loutit)

Linkage tests

1. Chr 2: distance of Sd from centromere

Rb(2.18)6Rma has been used to estimate this distance. Outcrosses of Rb6Rma +/− Sd mice gave 32 Rb6Rma, 19 Sd and 1 + offspring, total 52. The RF is 1.9 ± 1.9%. This suggests that the Sd locus is about 2 units from the centromere, although the possibility that heterozygosity for this wild-derived Robertsonian translocation leads to some crossover suppression in this region cannot be ruled out entirely. (Beechey and Searle)

2. Chr 16: distances of T43H and T17H from mdnc

A linkage testcross of T(16.17)43H with mdnc gave the following progeny: 21 T43H, 7 mdnc, 2 +; total 30. Thus the RF is 6.7 ± 4.6%, but with a significant deficiency of non-agouti curly progeny. Since the Chr 16 breakpoint of T43H is actually within the centromeric heterochromatin (MNL 53: 30), this result confirms the closeness of the md locus to the centromere found previously by Roderick et al. (1976, MNL 53: 30) with Rb(16.17)7Bnr.

A linkage testcross of T(8;16)17H with mdnc gave 18 T17H, 6 mdnc, 6 T17H mdnc, 22 +; total 52. The RF is 53.8 ± 6.9%, again with a marked shortage of non-agouti curly progeny. This result is in line with expectation in view of the distal position of T17H on Chr 16. (Beechey and Searle)

3. Chr 18: distance of Tw from centromere

The centromeric marker Dp(18Hc) described by Evans et al. (MNL 61: 56 and 62: 70) has been used to gain further evidence on this. Dp(18Hc) +/− Tw outcrosses gave 33 Dp, 26 Tw, 1 Dp Tw and 10 + progeny, total 70. The RF is 15.7 ± 4.3%, somewhat higher than that obtained by use of Rb(2.18)6Rma (MNL 61: 40) but not significantly so. (Beechey, Burtenshaw, Brown, Evans and Searle)

4. Sxr; position in the linkage map unknown

A further attempt to determine the location of Sxr has proved unsuccessful. The distal region of chromosome 10 has now been investigated using Lop and no evidence of linkage was found. Sxr has been tested with Robertsonian translocations marking all centromeres except chromosome 7, which has been checked by Eicher (personal communication) with Ldr-1, and it is therefore unlikely that Sxr lies proximally in any chromosome. Moreover, the central regions of all chromosomes except chromosome 12 have also been thoroughly screened (the studies of Eicher included) and it would therefore seem that unless Sxr is not fixed in position it can only lie distally in some as yet inadequately marked region of chromosome. (Cattanach)

5. Position of Hk-1 on chromosome 10

Hk-1 has been assigned to chromosome 10 using the technique of somatic cell hybridization. (Lalley et al. 1978 Nature 274: 160). The discovery of a genetically determined electrophoretic variant of hexokinase-1 has enabled us to determine the position of the gene. 96 mice have been classified for d1, Hk-1 and S1con from the mating d1 Hk-1A S1Con/+ Hk-1B + x d1 Hk-1A S1Con/d1 Hk-1A S1Con and the results are as follows: 32 d1 HK-1A S1Con; 45 + HK-1AB +; 1 d1 HK-1B +; 9 d1 HK-1A +; 9 + HK-1B S1Con.

The gene order, with RF's is d1−1.0 ± 1.0-Hk-1−18.8 ± 4.0-S1Con.

66 mice have been typed for v, HK-1 and gr from the cross v Hk-1A gr/+ Hk-1B + x v Hk-1A gr/v Hk-1A gr giving the following results: 17 v HK-1A gr; 38 + HK-1AB +; 1 v HK-1AB gr; 1 + HK-1A gr; 3 v HK-1A +; 6 + HK-1AB gr.

The gene order, with RF's is v−3.0 ± 2.0-Hk-1−13.6 ± 4.2-gr.

Thus Hk-1 is closely linked to d1 and the probable gene order is v−d1−Hk-1−gr−S1Con. (Peters, Andrews and Beechey)
6. Linkage of Sdh-1

Sdh-1 has been located on chromosome 2. 46 mice were classified for SDH-1 and a in the cross Sdh-1\textsuperscript{a}/Sdh-1\textsuperscript{b} × Sdh-1\textsuperscript{a}/Sdh-1\textsuperscript{a} giving the following results:

25 Sdh-1\textsuperscript{A} a; 8 Sdh-1\textsuperscript{AB} a +; 7 Sdh-1\textsuperscript{A} +; 4 Sdh-1\textsuperscript{AB} a. The RF for these two loci is 25.0 ± 6.3. No linkage was found with \( c^\text{ch} \), \( d \), \( s \), \( e \), and \( w-a-1 \). Further testing in which 104 mice were typed for SDH-1, pa, a and bp in the cross Sdh-1\textsuperscript{a} pa a bp/Sdh-1\textsuperscript{b} +/+ × Sdh-1\textsuperscript{a} pa a bp/Sdh-1\textsuperscript{a} pa a bp gave results as follows: 40 Sdh-1\textsuperscript{A} pa a bp; 42 SDH-1\textsuperscript{AB} +/+; 2 SDH-1\textsuperscript{A} +/+; 4 SDH-1\textsuperscript{A} pa +/+; 12 SDH-1\textsuperscript{AB} + a bp; 2 SDH-1\textsuperscript{A} pa a +; 1 SDH-1\textsuperscript{AB} +/a bp; 1 SDH-1\textsuperscript{AB} + a +.

The gene order, with RF's, is Sdh-1-1.9 ± 1.4-pa-15.4 ± 3.5-a-2.9 ± 1.6-bp.

(Peters and Andrews)

*************

HOUSTON

Medical Genetics Center
Univ. of Texas Health Science Center
P.O. Box 20334, Astrodome Station
Houston, Texas 77025

Received: June 4th 1980

Research News:

AKR Substrains

AKR/J and AKR/Cum are Mod-2\textsuperscript{b}, unlike AKR/FuA which has previously been reported to be Mod-2\textsuperscript{a}. This result parallels other cases in which AKR/FuA differs from both AKR/J and AKR/Cum (Acton et al. Nature New Biol 24:48, 1973).

Donor's Sex Can Influence Thy-1 Immunogenicity

The anti-Thy-1.1 antibody responses of C57BL/6J females immunized with B6-Thy-1\textsuperscript{a} male thymocytes (4 x 10\textsuperscript{7} cells injected 6 days before plaque assay) exceeded those of similar recipients immunized with B6-Thy-1\textsuperscript{a} female thymocytes by more than 60-fold in the first experiment and more than 5-fold in the second. In contrast, AKR/Cum females produced weak anti Thy-1.1 plaque-forming cell (PFC) responses when immunized with either AKR/J male or female thymocytes.

<table>
<thead>
<tr>
<th>PFC Response Tested</th>
<th>Expt. 1</th>
<th>Expt. 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>anti B6-Thy-1\textsuperscript{a} female</td>
<td>3.0 ± 0.8</td>
<td>18.8 ± 3.8</td>
</tr>
<tr>
<td>&quot; male</td>
<td>19.4 ± 55.2</td>
<td>95.5 ± 20.0</td>
</tr>
<tr>
<td>anti AKR/J female</td>
<td>8.7 ± 5.3</td>
<td>1.0 ± 0.3</td>
</tr>
<tr>
<td>&quot; male</td>
<td>5.0 ± 1.8</td>
<td>15.5 ± 6.6</td>
</tr>
</tbody>
</table>

*anti Thy-1.1 PFC per 6 x 10\textsuperscript{6} immune splenocytes

These results are particularly interesting because: 1) non-Thy-1 alloantigens can modulate the immunogenicity of Thy-1 determinants expressed on the same cells, 2) male thymocytes express H-Y antigen but female thymocytes lack it, and 3) H-2\textsuperscript{b} strains are high responders to H-Y, but H-2\textsuperscript{K} strains are low responders.

Similar experiments with anti Thy-1.2 PFC responses gave less clear-cut results due to the high control responses of B6-Thy-1\textsuperscript{a} females to C57BL/6J female thymocytes (260.0 ± 56.7 and 65.7 ± 25.3 as compared with anti-male responses of 261.2 ± 67.3 and 163.3 ± 18.9 in two experiments). AKR/J or AKR/Abom females responded poorly when immunized with either AKR/Cum male or female thymocytes.

Work in Progress:

We have begun transferring the Thy-1\textsuperscript{b} allele of AKR/Cum to AKR/J. This strain is currently at N4.

*************

- 18 -
Frozen Embryo Repository.

The following stocks are now preserved as frozen embryos:

<table>
<thead>
<tr>
<th>Stock</th>
<th>Modifier</th>
<th>Parental Strain</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABP/Le</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a/a b+/+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B6C3-a/a-we un a</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B6C3-a/a-opt*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B6C3-a/a-sy*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B6C3-a/a-Wt-33J</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C57BL/6J-a</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C57BL/6J-bJfJ</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C57BL/6J-Ra</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C57BL/6J-1m*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C57BL/6J-1u*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C57BL/6J-1b*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C57BL/6J-ma*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C57BL/6J-md*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C57BL/6J-p*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C3HeB/FeJ-Avy*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBA/2J-d/d</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBA/2J-d/d</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c/c Rk/1+*</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Those stocks marked with an asterisk will be removed from the "breathing state" and available only as frozen embryos by December 1, 1980. The following three stocks are already only in the frozen state, C57BL/6J-p, -Ra, and DBA/2J-d/d.*

The segment of Chr 5 containing rd and le from strain C3H crossed into C57BL/6J has lost the glucuronidase allele from C3H originally contained in this stock. (Mobraaten)

New mutants.

Head tilt (het). An autosomal, recessive, circling mutant characterized by a tilted head, circling behavior, inability to swim and hyperactivity arose in strain GL/Le in 1976. Homozygotes are not deaf. Tests for allele with av, fi, je, qk, sh-1, sh-2, sr, sv, v and wi were all negative. Linkage tests show het linked to T on Chr 17. Backcross data (2/113) give a recombination of 1.77 ± 0.12%. (Sweet)

Linkage data.

1. Pre-1 and Chr 12. In MNL 62:56 we reported 38% recombination between the centromere of Chr 12, marked by Rb(8.12)5Bnr, and prealbumin-1 (Pre-1). We have now collected more mice in this cross. There have been 13 recombinants in a total of 24 backcross offspring which gives a recombination frequency greater than 50%. Since Pre-1 has been assigned to Chr 12 by Eicher and Taylor (MNL 61:42) and Andrews et al. (MNL 61:40), our data support the suggestion by Eicher and Taylor that Pre-1 is on the distal end of the chromosome. (Davisson and S. Langley)

2. Linkage with T(3;4)5Rk. In MNL 57:20 we reported a new translocation, T(3;4)5Rk, found among the progeny of an inversion heterozygote. Linkage studies are now in progress to determine the genetic positions of the breakpoints. The breakpoint on Chr 4 has not recombined with b in a total of 167 offspring from two separate linkage tests. Data from male and female F1's is combined because they do not differ. The Chr 4 breakpoint is in cytological band 4C2 suggesting the b locus is in or very near this chromosomal band.
The Chr 3 breakpoint probably is between my and ma. Two crosses are in progress: a two-point cross with my and a four-point cross with Car-2, ma and Va'. The data to date are as follows:

<table>
<thead>
<tr>
<th>Interval</th>
<th>Recomb./Total</th>
<th>Per cent Recomb.</th>
</tr>
</thead>
<tbody>
<tr>
<td>T3RK - Car-2</td>
<td>13/36</td>
<td>36.1 ± 8.0</td>
</tr>
<tr>
<td>T3RK - my</td>
<td>5/26</td>
<td>5.3 ± 2.3</td>
</tr>
<tr>
<td>T3RK - ma</td>
<td>7/66</td>
<td>10.6 ± 3.8</td>
</tr>
<tr>
<td>T3RK - Va</td>
<td>22/66</td>
<td>33.3 ± 5.8</td>
</tr>
<tr>
<td>Car-2 - ma</td>
<td>15/36</td>
<td>41.7 ± 8.2</td>
</tr>
<tr>
<td>ma - Va</td>
<td>23/66</td>
<td>34.8 ± 5.9</td>
</tr>
</tbody>
</table>

The data from the four-point cross so far suggest that the breakpoint is probably on the centromere side of ma. The translocation breakpoint does not appear to reduce recombination since the distance between ma and Va is comparable to this distance on the linkage map.

The cytological breakpoint on Chr 3 is in band 3D indicating that my and ma are in the vicinity of this cytological band. (Davisson)

3. Location of syndactyli sm (sm) on Chr 12. The search for this linkage has been a long one. Extensive published and unpublished data of several investigators confined further searches to a relatively few regions. In searching for possible markers for an inversion on Chr 12, we found linkage between sm and Pre-1. Data on homozygous sm males from an intercross revealed three recombinants in 15 animals. A backcross to a male homozygous for sm yielded five offspring to date all of which are parental. The five consisted of two sm/sm and three sm/+ males. The distance between sm and Pre-1 based on their preliminary data is approximately 9cM. (N. L. Hawes, S. H. Langley, Roderick)

Strain Characterization.

As opportunity permits, we are continuing to characterize inbred strains for polymorphic loci. The following are new data:

<table>
<thead>
<tr>
<th>Strain</th>
<th>Gene</th>
<th>Allele</th>
</tr>
</thead>
<tbody>
<tr>
<td>A/WySn</td>
<td>Np-1</td>
<td>a</td>
</tr>
<tr>
<td>RF/J</td>
<td></td>
<td>a</td>
</tr>
<tr>
<td>LP/J</td>
<td></td>
<td>a</td>
</tr>
<tr>
<td>C57BL/cdJ</td>
<td></td>
<td>a</td>
</tr>
<tr>
<td>C57L/J</td>
<td></td>
<td>a</td>
</tr>
<tr>
<td>BALB/cJ</td>
<td>Amy-1</td>
<td>a</td>
</tr>
<tr>
<td>ST/bJ</td>
<td></td>
<td>a</td>
</tr>
<tr>
<td>A/WySn</td>
<td></td>
<td>a</td>
</tr>
<tr>
<td>C57BL/10J</td>
<td></td>
<td>a</td>
</tr>
<tr>
<td>C57BL/KsJ</td>
<td></td>
<td>a</td>
</tr>
</tbody>
</table>

The following are confirming data:

<table>
<thead>
<tr>
<th>RIIS/J</th>
<th>Apk</th>
<th>a</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sdh-1</td>
<td>a</td>
</tr>
<tr>
<td></td>
<td>Got-1</td>
<td>a</td>
</tr>
<tr>
<td></td>
<td>Got-2</td>
<td>b</td>
</tr>
<tr>
<td></td>
<td>Pre-1</td>
<td>a</td>
</tr>
<tr>
<td></td>
<td>Mor-1</td>
<td>a</td>
</tr>
<tr>
<td></td>
<td>Car-2</td>
<td>b</td>
</tr>
<tr>
<td></td>
<td>Es-1</td>
<td>b</td>
</tr>
</tbody>
</table>

(S. H. Langley, E. G. Holt, Roderick)

Change of name and gene symbol.

Petite (pet) to hypothyroid (hty). In MNL 56:41, a report was made of a recessive mutation in RF/J strain mice that caused substantial dwarfism in the homozygous condition. Allelism tests with Snell's dwarf (dw) were negative. The mutants were reported to be highly responsive in terms of body growth and reproductive activity when desiccated thyroid was administered via the diet. Subsequent studies have shown that homozygous mutants are hypothyroid by several criteria because of the failure of the thyroid to respond to pituitary thyroid stimulating hormone. The mutant gene is therefore renamed hypothyroid (hty); its assignment to the genetic linkage map is yet to be made. (W. Beamer and Eicher)
Stocks to be discontinued.

1. The strain, PH/Re, maintained in Dr. E. S. Russell's colony, will be discontinued December 1, 1980. This stock will not be preserved in the frozen embryo repository since the Patch gene has been put on a C57BL/6J background and that line is being frozen down. (Mobraaten)

2. We now maintain in the Mouse Mutant Stocks Center two alleles of motor end plate disease, med^10, and jolting, med^16. We plan to discontinue the B6C3-a/A-med^16 by September 1980 and will supply breeders to anyone willing to maintain it. We will continue to maintain B6C3-a/a-Ca med^10/++. (Sweet and Lane)

**************

J. H. Moutschen
M. Moutschen-Dahmen
J. Rigot-Monfils
F. Gillet

LIEGE

Université de Liège,
Laboratoire de Génétique,
15, rue Forgeur,
B-4000 Liège,
Belgium

Received: June 2nd 1980

Strains:

C57BL
C57BR
Q from Edinburgh and X0 and X Ta 0 9 in this Q strain
Tailless T tf/t^6+
Tailless T tf/t^12+

Research news:

1. A t^v haplotype derived from T tf/t^6+ has been isolated.
   Male fertility was reduced in matings with normal-tailed females.
   The t^v allele showed a high transmission ratio. In combination with
   the t^6 alleles, it does not show further decrease of male fertility.
   New matings are realised to test this allele.

2. In the progeny of tailless T/t^6 x T/t^12, a short-tail male was
   recovered and was found to be partly fertile. Short-tail mice
   appeared in the offspring. This system is under investigation.

**************
Research News

Since our first report of the new neurological mutant twitcher (twi) (MNL 61, 47) extensive light and electron microscopic studies of central and peripheral nervous system abnormalities have been done. The myelin sheaths of CNS and peripheral nerves are formed normally until the 15th day of life when they begin to break down. Macrophages accumulate in the areas showing demyelination and contain polyhedral or tubular inclusions which are similar to those seen in human and canine globoid cell leucodystrophy (Krabbe's disease). Collaborative studies with Suzuki and Kobayashi (Albert Einstein College of Medicine, New York) have shown a deficiency of the enzyme galactosylsphingomyeline in the nervous system and other organs of twitcher. This enzyme is known to be deficient in Krabbe's disease and it seems therefore that in the twitcher mouse we have an exact counterpart of the human disease.

The development of the demyelination is under investigation quantitatively and experimentally by the use of nerve grafts. When twitcher sciatic nerves are grafted into the nerves of unaffected littermates the morphological features of the demyelinating disease are reproduced. This finding indicates that twi Schwann cells rather than axons are involved in the pathogenesis of this leucodystrophy. Further studies are in progress.

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LÜBECK
Medizinische Hochschule Lübeck
Institut für Pathologie,
Ratzeburger Allee 160
2400 Lübeck 1
Fed. Rep. Germany

Received: June 9th 1980

Research News

Postnatal Survival of Mouse Trisomy 19

Late fetal progeny and newborn litters with Ts 19 individuals were obtained from matings of Rh(9.19)163H/Rh(8.19)1Ct males with NMRI females. The frequency of Ts 19 among the balanced in utero-mates was about 28%, 24%, and 19% on days 17, 18 and 19 respectively. 22 females were allowed to bring forth young, and the litters were carefully observed from birth on. Several litters were transferred to a foster mother. In some litters, suspected Ts 19 newborns were karyotyped only when death appeared to be impending, although few newborns escaped the karyological control, because they died unexpectedly. The longest span of survival noted was until day 13. In some other litters, suspected Ts 19 and control mates were sacrificed and karyotyped on days 2, 4, 6 and 8. Despite the different modes of ascertainment, an approximate mean total frequency of 5% Ts 19 can be assumed for days 1 to 12 after birth. The present observations supplement those of B. White et al. (Cytogenet. Cell Genet. 11: 363, 1972 and 13: 217 and 332, 1974), showing the selective elimination of Ts 19 is effective only around or at birth, and in the following first days of life. At least, many Ts 19 anlagen are born, and may even survive at least one week up to day 13. Their phenotype is characterized by marked hypoplasia. No gross malformation is observed. Death seems to be caused primarily by respiratory failure. (G. Grohé and A. Gropp)
Trisomy 7 of the Mouse Embryo

Ts 7 has been induced by Rb(6.7)l3Rma/Rb(7.8)28Lub x NMRI matings. Doubly heterozygous males of this type may be sterile (5/15) or subfertile (4/15), but some fertile males (6/15) can be observed. Fertility is not obviously impaired in female double heterozygotes (10/10).

Using matings with male Rb heterozygotes, 5 pregnant NMRI females were dissected on each of days 9, 10, 11, 12, 13, 14 and 16 (vaginal plug = day 1). The frequency of Ts 7 among live fetal progeny was estimated 30% on day 11, and 5% on day 13. No trisomics were found after day 13. These data show that lethality of Ts 7 is relatively early. The affected embryos are morphologically retarded corresponding to developmental stages of day 9-10.

In order to examine possible variations of phenotypic expression of Ts 7 depending on different genetic background, C57BL/6J and BALB/c females were mated with Rb doubly heterozygous males and dissected on days 11, 12, 13 and 14. On day 11, the frequency of trisomics was 31% in C57BL and 13% in BALB/c. However, in both cases, no trisomic embryos were found after day 12.

The preimplantation loss is remarkably high in BALB/c mating (43% of oocytes) compared to 27% in NMRI and 26% in C57BL, while the mean total postimplantation loss, evaluated on days 11 to 14, is highest in C57BL/6J (48% of implants) compared with 38% in NMRI and 37% in BALB/c. (U. Jüdes, H. Winking and A. Gropp)

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MANCHESTER

Paterson Laboratories
Christie Hospital
Manchester M20 9BX
England

Received: June 4th 1980

The following inbred strains are maintained

DBA/2J obtained from Jackson Laboratories in 1966
C57BL/6J obtained from Jackson Laboratories in 1966
BALB/c obtained from LAC 1979

The main production mouse is B6D2Fl

Occasionally DBA/2J and C57BL/6J and B6D2Fl mice are produced in excess of our requirements.

Mutants held: bg, hr, S1, S1\(^d\), W, W\(^v\)

***************
New mutant

Epidermal antigen, Epa. An apparent loss mutation in C3H strain mice has revealed a locus that determines alloantigens on mouse epidermal cells that are weakly expressed on mouse lymphocytes. The antigen, Epa.1 is present on epidermal cells of strains AKR, B10.RR, B10.K, CBA and RF but absent from C3H epidermal cells. Both C3H/He and C3H/AnfCum were tested; hence the mutational event must have occurred before Heston derived his C3H substrain from Andervont. There is a 1:1 distribution of Epa.1+ and Epa.1- offspring in the (C3H x CBA)F1 x C3H backcross population. Anti-Epa.1 cytotoxic T lymphocytes are evoked by priming and boosting C3H spleen cells with epidermal cells of any of the Epa.1 positive strains. Specificity of the anti-epidermal cell cytotoxicity was established by cold-target inhibition and suicide techniques. (Steimuller, Tyler and David)

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MRC MAMMALIAN DEVELOPMENT UNIT

Wolfson House, (University College London)
4 Stephenson Way,
London NW1 2HE

Received: June 6th 1980

Research news

GPI in the mouse testis

Cellulose acetate ("Cellogel") electrophoresis has revealed that glucose phosphate isomerase (GPI-1) from mouse testis and sperm shows a small subsidiary band running slower than the main band. The distance from main to subsidiary bands is constant in both GPI-1A and GPI-1B forms, and heterozygotes (with three main bands) show three sub-bands as well. Sub-bands of this type were not present in any somatic tissue examined, nor in the testis of new-born males. In a study correlating the presence or absence of GPI sub-bands with the first appearance of sperm in the testes of maturing males, no sub-bands were seen until 35 to 40 days of age, when over half the seminiferous tubules contained maturing spermatzoa. Testes of X0 Sx+/ males (in which few to no normal spermatzoa were seen) showed weak or no sub-band activity. (M. Buehr and A. McLaren)

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MONTPELLIER

Laboratoire d'Evolution des Vertébrés
Université de Montpellier II
Pl. E. Bataillon
34060 - MONTPELLIER Cedex
FRANCE

Received: June 10th 1980

RESEARCH NEWS

1/ - Robertsonian populations of mice from Northern Italy, Alpi Orobie (22 chr) and Milano II (24 chr), were screened for genetic variation at 30 enzymatic loci. These populations clearly belong to Biochemical Group n° I and show no significant genetical differences between different Robertsonian populations or with surrounding 40 chr. populations (ref. J.B-D et coll. C.R.Acad.Sc. Paris, t. 290,D, 195-198, 1980).

*************
Personnel News

Dr. I.M. Roupova from the Institute of Roentgenology and Radiobiology, Sofia, Bulgaria, will spend one year as an IAEA fellow in our laboratory. Also, Dr. P.S. Chauhan from the Bhabha Atomic Research Centre, Bombay, India, will be in our laboratory as a guest scientist for the next year.

Research News

The LDH mutant described in the last issue of MNL (62 (1980), 66-67) has been further characterized. This mutant, detected by polyacrylamide gel isoelectric focusing, exhibited an altered LDH pattern. The mutation affected the locus coding for LDH of the muscle type and Ldh-1N was suggested as the provisional allele symbol. LDH specific activity was measured in homozygous wildtypes (Ldh-1+/Ldh-1+), in heterozygous mutant (Ldh-1+/Ldh-1N) and in homozygous mutant (Ldh-1N/Ldh-1N) genotypes. In the liver of (Ldh-1+/Ldh-1N) and (Ldh-1N/Ldh-1N), LDH activity decreased respectively to about 50% and 9% of the (Ldh-1+/Ldh-1+) value. LDH activity in (Ldh-1+/Ldh-1N) and (Ldh-1N/Ldh-1N) was more reduced in the tissues where LDH-A predominates (liver, muscle, spleen) than in those where LDH-B predominates (heart, kidney, brain, lung, blood). Thermal stability experiments, performed with liver, muscle and heart extracts, showed that (1) LDH from the heart was more sensitive than that of muscle or liver, (2) in liver and muscle (where LDH-A predominates) a more pronounced heat inactivation of LDH activity in (Ldh-1+/Ldh-1N) and much more in (Ldh-1N/Ldh-1N), whereas in heart (where LDH-B predominates) LDH heat inactivation was almost the same in (Ldh-1+/Ldh-1N), (Ldh-1+/Ldh-1N) and (Ldh-1N/Ldh-1N). Homozygous mutants, which are fully viable and fertile, exhibited a special character: the spleen is about ten times heavier than those of (Ldh-1+/Ldh-1N) and (Ldh-1+/Ldh-1+N). The number of lymphocytes in (Ldh-1N+/Ldh-1N) is half of that in the (Ldh-1+/Ldh-1N), but the number of granulocytes in (Ldh-1N+/Ldh-1N) is two times that in (Ldh-1+/Ldh-1N).

(Daniel J. Charles and Walter Pretsch)

*Non-standard; see rule 4c of MNL 61: 5 (Ed. note)
C57BL/6 (B6) mice and most of their F1 hybrids produce low anti-Thy-1 responses when immunized with B6.PL(74NS) thymocytes. It was suggested that this is due to the lack of carrier effect of non-H-2 molecules when Thy-1 congenic strains were used as the donor and responder. However, F1 hybrids of B6 mice that are b/d heterozygous at the centromeric portion of the H-2 complex produced a good response upon immunization with thymocytes of the 74NS strain. It has been postulated that, in this case, an H-2 controlled moiety expressed by b/b homozygous donors but absent in b/d heterozygous responders might be providing the carrier effect required for a good anti-Thy-1 response. Results shown in the table below suggest that, if this concept is correct, such a moiety would be determined by genes at the IA and/or IB subregions.

Studies to exclude a possible complementation between H-2d-linked and B6-associated Ir genes are presently in progress (Zaleski, Gorzynski).

<table>
<thead>
<tr>
<th>Responder</th>
<th>b/d heterozygosity</th>
<th>PFC/spleen</th>
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<tr>
<td></td>
<td>K</td>
<td>IA</td>
</tr>
<tr>
<td>B6D2F1</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>B6D2GOF1</td>
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<td>+</td>
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<tr>
<td>B6LG/JF1</td>
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<td>+</td>
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<tr>
<td>B6LG/CkF1</td>
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<td>-</td>
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Induction of a CML response to the Thy-1 antigens has been attempted. The table below shows the experimental design and the results obtained.

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<tr>
<th>Responder (spleen cells)</th>
<th>Thy-1 phenotype type</th>
<th>In vivo priming</th>
<th>Thy-1 phenotype type</th>
<th>In vitro boosting</th>
<th>Thy-1 phenotype type</th>
<th>Percent specific lysis of target thymocytes from</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>AKR.B6</td>
</tr>
<tr>
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<td>-</td>
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<td>-1.3(3)</td>
<td>-2.1(3)</td>
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<tr>
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<td>-</td>
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<td>1.1(4)</td>
<td>1.4(4)</td>
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<td>-1.7(1)</td>
</tr>
<tr>
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<tr>
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<td>AKR.B6</td>
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<td>74NS</td>
<td>1.1</td>
<td>2.0(2)</td>
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<tr>
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<td>AKR.B6</td>
<td>1.1</td>
<td>AKR.B6</td>
<td>1.1</td>
<td>3.0(4)</td>
</tr>
<tr>
<td>C3H.B10</td>
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<td>74NS</td>
<td>1.1</td>
<td>74NS</td>
<td>1.1</td>
<td>63.6(2)</td>
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<tr>
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<td>1.1</td>
<td>C3H.B10</td>
<td>1.2</td>
<td>C3H.B10</td>
<td>1.2</td>
<td>-</td>
</tr>
</tbody>
</table>

The number of experiments is indicated in parentheses. The E/T ratio employed was 40:1 though in some instances a higher ratio was tested also with essentially the same results.

As can be seen, a positive CML reaction could be demonstrated only when the responder was both primed in vivo and boosted in vitro with thymocytes from donors with multiple minor histocompatibility differences. On the other hand, no response could be detected when the donor of priming or boosting cells shared entire genotype except Thy-1 alleles with the responder. Similar results were obtained in some other strain combinations. Thus, the response appears to be directed to minor alloantigens but not to the Thy-1 antigens. The experiments employing competitive inhibition demonstrated no or negligible inhibition by Thy-1 congenic cells used in an I/T ratio of 40–80:1, supporting the conclusion that Thy-1 disparity does not seem to elicit a CML reaction (Freimuth, Zaleski).
Research News:

1. The mouse colony containing our collection of histocompatibility gene mutations has been successfully transferred from Dr. Henry Kohn's laboratory in Boston and currently includes 25 H-2 mutations (21 recovered from this colony) and 23 non-H-2 mutations (all recovered in this colony). We are adding the C3H/HeJ strain to the BALB/cKh and C57BL/6Kh strains already being monitored for such mutations. (Melvold)

2. A spontaneous mutation was found which has simultaneous effects on pigmentation and ocular development. The mutation (currently designated KH-179) is semidominant and is very reminiscent of M1Wh--the heterozygote is "leaden" in color (on a non-agouti C57BL/6 background) and the eyes are reduced in size, frequently never opening. The homozygous mutants are entirely white and the eyes are essentially absent. However, genetic complementation tests with M1Wh indicate that a separate locus is involved. Furthermore, the spotting which is characteristic of M1Wh is not seen in KH-179/+ animals. Interestingly, the effect on pigmentation involves only eumelanin--the production of phaeomelanin is unaffected and the activity of the agouti (A) gene is observable. KH-179 homozygotes which are AA or Aa have white hairs with a subterminal band of phaeomelanin and are "buffy" in appearance. Comparable bands of phaeomelanin are seen in the KH-179 heterozygote. Collaborative studies with Dr. Jan Leestma are in progress to investigate the deficiencies in the development of the eye. (Melvold)

3. Studies of neural circuitry in normal and neurological mutants are being done with enzyme and isotope tracer techniques. In particular, cortical circuitry and pathways related to cerebellar pathways (deep nuclei and red nucleus) are being examined to determine the extent to which the brains of neurological mutants are "rewired". (Shipley)

4. We are investigating the short and long term effects of in vitro exposure to carcinogens on preimplantation development in BALB/c mice. We have found small but statistically significant decreases in the incorporation of thymidine and leucine into the blastocyst following exposure to methylnitrosourea at concentrations that do not affect embryo viability. Uridine incorporation is decreased only after 18 hours of culture following exposure. (Iannaccone)

We are also trying to identify shifts in the latent period of chemically induced tumors in chimeric mice. (Iannaccone)

5. The susceptibility of various mouse strains to antibody against the major idioype (Tl5id) of BALB/c anti-phosphorylcholine antibody has been compared. After treatment of neonates with anti-idioype antibody within two days of age, these mice become tolerant to the antigen with some degree of difference in the levels of tolerance depending on the strains. In contrast to continuous sensitivity of BALB/c mice, the resistance to the antibody treatment in C57BL/6, (BALB/c x C57BL/6)F1 and CB-20 mice was much more apparent when anti-idioype antibody was injected after 10-15 days of age. Idiotypes and suppressor cells are further analyzed to examine the possibility that the idioype of the "primordial clones" specific for phosphorylcholine may be cross-reactive with Tl5id and the differentiation of B cell repertoire may occur during early life under a genetic influence of the IgCh genes. (Kim)
6. SJL/J (H-2^5) mice, a strain that has a high incidence of spontaneous reticulum-cell carcinomas (RCS), exhibit several interesting characteristics regarding immune recognition of and response to syngeneic, transplantable RCS lines. These transplantable RCS cells are Thy-1.2^e and sig^f, but do express the private I-A specificity (Ia.4) of the s haplotype (J. Exp. Med. 146:132, 1977). Irradiated RCS cells stimulate marked proliferation of Ly-1^e,23^SJL/J T cells (Int. J. Cancer 14:808, 1974; Cell. Immunol. 41:157, 1978; Cell. Immunol. 43:209, 1979), a characteristic that appears to be mediated by the RCS-associated Ia determinants. Despite this T cell proliferative response, there is no development of RCS-specific cytotoxic effector cells in either primary in vitro mixed lymphocyte-tumor cell cultures or after repeated immunization of SJL mice with irradiated RCS cells (Cell. Immunol. 32:10, 1977). We recently observed that cells taken from RCS-enlarged lymphoid tissues can act as efficient cytotoxic effectors against the natural killer (NK) cell-susceptible targets, YAC-1, RL0 1 and RBL-5 (Cell. Immunol. 43:185, 1979). It appears that the overall NK activity detected in RCS-bearing SJL/J mice involves two components: RCS-induced, host mediated NK activity and a contribution by the RCS tumor cells themselves. This is not particularly intriguing, since SJL/J mice are considered a genetically low (if not defective) NK strain that cannot be boosted with other known inducers of NK cells (e.g., Poly I:C, BCG or C. parvum). We are currently investigating the RCS-associated NK activity in an attempt to identify the nature of the NK effector population(s) present, and to what degree interferon (a potent inducer of NK cells) might influence the development of NK cytotoxicity in SJL/J mice. (Ponzo)

7. Current work in our laboratory involves the T cell regulation of the immune response to the synthetic linear copolymers of L-glutamic acid^90-L-alanine^30-L-tyrosine^10 (GAT) and L-glutamic acid^50-L-tyrosine^50 (GT). Genetic control of specific antibody responses to these thymus-dependent antigens are controlled by I region genes in the H-2 complex. Mice bearing the appropriate immunotype (Ir) gene are able to make antibody responses to these synthetic antigens. GT is not immunogenic in any of more than 20 inbred mouse strains so far tested, although all strains make anti-GT antibody against GT when it is coupled to the immunogenic carrier protein, methylated bovine serum albumin (GT-MBSA). GT preimmunization has a suppressive effect on GT-MBSA responses in strains bearing the H-2^d,f,k^s haplotypes, but not in those with H-2^a,b,q haplotypes. This suppression is mediated by antigen-specific suppressor T lymphocytes. Antigen-induced suppressor T cell activity can be abolished by pretreatment of mice with cyclophosphamide. The development of GT-specific suppressor T cells is controlled by cis or trans complementing immune suppressor (Is) genes, tentatively mapped into the I-A/I-J-B and I-C/S subregions of the H-2 complex. GT-induced suppressor T cells produce a soluble suppressor factor (TsF) which has an antigen-binding site, bears I-J subregion-encoded antigenic determinants, has a molecular weight of approximately 50,000 daltons, and functions in large measure by inducing, together with antigen, a second set of suppressor T cells in unprimed syngeneic or allogeneic T cell populations. Current work in the laboratory centers around the characterization of an analogous helper factor for GT, a factor which has the ability to allow an immune response in normally unresponsive strains of mice. (Waltenbaugh)


Sir William Dunn School of Pathology, University of Oxford, South Parks Road, OXFORD. OX1 3RE England

Received: June 12th 1980

Personnel News:

Dr. Andrew Copp has moved to the Pediatric Research Unit, Guy's Hospital Medical School, London. SE1 9RT.

Research News:

\(X^{mO}\) Mice

Pgk-\(1^b, \text{In}(X)1H/Pgk-1^b\), + females were crossed with Pgk-\(1^a, +/Y\) males to produce \(X^{mO}\) mice. 112 adult female progeny were typed for Pgk-1 (using peripheral blood) and resulted in 86 Pgk-1AB(XX), 24 Pgk-1A(XPO) and 2 anomalous Pgk-1B females. Breeding experiments suggested that both of these Pgk-1B females were \(X^{mO}\), and further experiments are planned to determine whether the Pgk-1A, +/Y males produce significantly more than the expected 0.1 - 1.1% spontaneous \(X^{mO}\) females reported by Russell (1968) in "Effects of Radiation on Meiotic Systems", pp. 27 - 41.

A sample of 100 mitotic cells from the bone marrow of one of the PGK-1B females was analysed, 84 contained 39 chromosomes and 16 contained 39 chromosomes with an additional centric fragment about 2/3 the size of a normal Y chromosome. C-banding revealed the presence of an inverted X in all the cells, thus providing unequivocal proof of the mouse's \(X^{mO}\) constitution. The fragment, although morphologically similar to the Y chromosome in being uniformly stained, possessed a C-band, which must rule out the possibility of it being part of this chromosome. Since the female came from an \(\text{In}(X)1H/+\) mother, in which a dicentric and acentric chromosome could be formed at meiosis, it is tempting to speculate that the fragment resulted from the breakage and reunion of part of the dicentric either at first anaphase or at some subsequent division.

(West, Brown, Burtenshaw and Evans)

Pachytene observations in inversion heterozygotes

Through the use of conventional air-dried preparations and C-banding, our previous meiotic studies of male mice heterozygous for paracentric inversions have enabled us to identify characteristic inversion bivalents at diakinesis/metaphase I. Their appearance could be related to both numbers and position of chiasmata formed in the proximal, inverted and distal segments. Since the air drying technique does not readily lend itself to the study of meiotic prophase, we have used our sedimentation/silver staining technique to study the pairing of the lateral axes under the light microscope. Unfortunately, such preparations from mice heterozygous for \(\text{In}(2)5RK\), \(\text{In}(2)2H\) and \(\text{In}(3)9RK\) yield only a low observed frequency (< 10%) of the expected classical inversion loop in pachytene spermatocytes. Moses (in Chromosomes Today, Vol. 6, pp. 71 - 82, 1978) has suggested that this results from the elimination of loops relatively early in pachytene, by a process of synaptic adjustment, so that 20 normal looking bivalents are presented. At the synaptonemal complex level in the mouse it is not possible to identify any particular autosomal bivalent, so it is difficult to follow the pairing sequence and synaptic adjustment with confidence.
To overcome this problem we combined Rb(2.18)6Rma with the homozygous lethal In(2)5 RK, which enables ch 2 to be recognised with certainty. Some of the males produced were killed for pachytene studies whilst sexually immature, a few were briefly fertility tested before sacrifice. The following conclusions could be made:

1) The presence of the metacentric, enabled the segmental relationship of the inverted and non inverted ch 2 to be clearly followed, both during loop formation and its unravelling.

2) The observed loop frequency was still low (8 - 13%) which suggests that the stage of pachytene during which it forms is short, and synaptic adjustment is a rapid process.

3) Male mice doubly heterozygous for Rb(2.18)6Rma/In(2)5RK are fertile, but preliminary results show a possible marked reduction in fertility as compared to that of single heterozygotes. This observation needs further testing.

(Burtenshaw, Brown and Evans)

Ts 18 studies

Rb(4.18)3H/Rb(2.18)6 Rma males are fertile, in contrast to some other metacentric combinations with common arms (monobrachial homology), and produce high frequencies of specifically unbalanced gametes by non-disjunction at anaphase I.

Matings between 3H1 females and Rb3H/Rb6 Rma males resulted in 49.1% (85/174) post implantation loss (uncorrected data). Cytogenetic analysis of implantations at 9 - 12 days post coitum (Plug = day 0) indicates 21.5% (31/144) Ts 18 embryos. Two males were subsequently killed for metaphase II analysis, giving 27% (54/200) nullosomic counts and 24.5% (49/200) disomic counts, the latter being in good agreement with the trisomic frequency.

On a gross morphological basis the survival of Ts 18 embryos after day 9 appears variable, with most trisomics recorded as retarded and/or dead on days 10 - 12. The degree of retardation ranges from '1/4 size - dead' at 11 days, to living 12 day embryos which, when fixed, have a body mass 64 ± 8.0% of balanced controls.

(Evans, Brown and Burtenshaw)
RESEARCH TRIANGLE PARK

NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH SCIENCES
P. O. Box 12233
Research Triangle Park, North Carolina 27709
USA

Laboratory of Animal Genetics

Laboratory of Biochemical Genetics

C.-Y. Lee

H. Burgess
F. M. Johnson
H. V. Halling
E. Porter

RESEARCH TRIANGLE INSTITUTE
Laboratory for Genetics
Chemistry and Life Sciences Group
P. O. Box 12194
Research Triangle Park, North Carolina 27709
USA

Received: June 9th 1980

L. Barnett
R. Batten
S. Lewis
P. MacDougal
R. Thomas

Mutant Stocks: Mod-1\textsuperscript{B}, Pgm-1\textsuperscript{n}, Idh-1\textsuperscript{v}, Pk-3\textsuperscript{r},

A\textsuperscript{v}, Bld, c\textsuperscript{3H}, c\textsuperscript{6H}, c\textsuperscript{25H}, c\textsuperscript{14COS}, c\textsuperscript{112K},
t\textsuperscript{w5}, Ts

Research News:

We have tested the effects of chronic doses of procarbazine on viability, fertility, and induction of mutations. Male mice from the DBA/2J strain were injected with procarbazine at a dose of 200 mg/kg body weight twice weekly until an accumulated dose of 2400 mg/kg was reached. Most of the treated animals died as a result of exposure and all survivors became temporarily sterile. After regaining fertility the few survivors were repeatedly mated with C57BL/6J females over several weeks time to generate a population of F\textsubscript{1} animals. The parental animals and the F\textsubscript{1} were subsequently analyzed by electrophoresis for the occurrence of newly arisen mutations of spermatogonial origin. A mutation at the Pep-3 locus was found. (F. M. Johnson, P. MacDougal, S. E. Lewis).

During the biochemical screening involved in a mutagenesis program, a new electrophoretically expressed Idh-1 allele was discovered. The altered phenotype was first recognized as an absent IDH-A homodimer in F\textsubscript{1} (DBA/2J x C57BL/6J) mice. The heterodimer however, was present and normal in mobility. In homozygous mutant animals, the IDH-A band is normal in position but streaks away from the origin. This mutation arose spontaneously in the C57BL/6 strain. (S. Lewis, L. Barnett, F. M. Johnson).

In a preliminary experiment, using Ethynitrosourea as the mutagen, 3 newly arisen electrophoretically expressed mutations were found among the 263 progeny of treated C57BL/6J males; while none were found in the 265 progeny of treated DBA/2J males. (F. M. Johnson, P. MacDougal, R. Batten, L. Barnett, S. Lewis).

***************

NO CHANGE

The following laboratories report no change since their last contribution:

UNIVERSITY OF LONDON (University College) .................. G.M. Truslove
NLMEGEN ............................................................. J.H.F. van Abeelen
MOUSE NEWS LETTER ACCOUNTS

Carshalton

Period: 1st October, 1978 - 30th September, 1979

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The Jackson Laboratory

Period: 1st November, 1978 to 31st October, 1979

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Audited by:

Rafael A. Hernandez
Accountant
28 February 1980
IMPORTANT

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************

Scientific contributions to the News Letter should be addressed to:

Dr. A.C. Searle,
MRC Radiobiology Unit,
Harwell,
Oxon. OX11 ORD,
England.

The next issue, Mouse News Letter No. 65 will be dated February 1981. Items for inclusion should reach Harwell promptly by December 15th 1980.

************

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