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A Robertsonian translocation involving the *X*-chromosome of the mouse

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A Robertsonian translocation between the *X* and chromosome 2 was recognized in a son of a female carrying the reciprocal translocation T(5;13). The original Rb(*X*.2)*Y* male was mated to (101/El × C3H/El)F_1 females with a mean litter size of 6·5±2·7 (four litters). The line was made homozygous. Two distinct centromeric heterochromatin blocks are present in C-banded trivalents at meiosis and in the metacentric chromosome in C-banded mitotic cells. The inactivation of the *X*.2 element was tested with *a* (non-agouti) as a marker on chromosome 2 and *Ta* (tabby) as a marker on the *X*-chromosome. The results indicate that there is no influence of the Robertsonian translocation on random *X*-inactivation and that the inactivation does not spread beyond the centromere to *a*. The non-disjunction of sex-chromosomes in Rb(*X*.2)*Y* males studied in spermatocytes at the second meiotic division showed 3% cells with 18,*X*.2,*Y* and 4% cells with 19,0. Tail-tip preparations of newborns performed for matings to obtain a homozygous Rb(*X*.2)-stock revealed 4% double *X*-males but no triple-*X* females among the progeny. The (*X*.2)*XY* and (*X*.2)(*X*.2)*Y* males were sterile, with spermatogenic breakdown prior to pachytene. The synaptonemal complex in Rb(*X*.2)*Y* males viewed in the electron microscope showed complete pairing between *X*.2 and the acrocentric chromosome 2 only in one out of 52 cells. Association between *Y* and the unpaired segment of 2 formed a ring in 11% of the cells, 80% of these were in late pachytene. The Rb(*X*.2) is the first available Robertsonian translocation involving the *X*-chromosome and will serve as a valuable tool to study a number of biological problems.

EUROGEN – European Central Mouse Facility

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To the Commission of the European Communities a proposal has been presented for a concerted action at the European level in Life Science, more particularly in the field of murine genetics of cancer. Such a concerted action programme can include a Central Facility. In this instance a central facility (or facilities) will be directed to mouse genetics related to cancer research. This action will fit in the Second Strategic Framework Programme for the period 1987/91 of the European Community – Biotechnology – Medical and Health Research Programme. The European Commission suggests that new action has to be taken on cancer. This action will be directed to improve services, infrastructures and communication in the Community.

This European Indirect Action Programme has been broadened and will also include the Genetics Division of the Medical Research Council Radiobiology Unit (Dr M. F. Lyon/Harwell) and the Unité de Génétique des Mammifères d'Institut Pasteur (Dr J. L. Guénet/Paris). These three institutes house and use large numbers of genetically defined strains of mice. Amsterdam, Harwell and Paris provide breeding stocks to many other workers in the EC and other countries and collaborate with many other laboratories. The Amsterdam, Harwell and Paris mouse genetics facilities therefore constitute a valuable resource for the EEC. There is clearly a 'supra-national' element in that the collaboration of the three laboratories in holding complementary ranges of stocks provides for the EEC a greater availability of strains than any one country could hold. It is proposed that these three institutes should jointly form an organization known as EUROGEN with the following aims:

(1) Maintenance of a wider range of wild mice stocks, mouse inbred strains, congenic strains, recombinant-inbred strains, mutants, polymorphisms, transgenics and chromosome anomalies. The stocks maintained in the

three laboratories would be complementary and would be made available to other EEC workers.

(2) Dissemination of information concerning mouse genetics and mouse stocks.

(3) Training and collaboration in mouse genetics by means of short-term fellowships to be held in EEC countries, and by facilities for visiting workers.

(4) Research in mouse genetics relevant to the program.

During the Mouse Genetics Group meeting the design of this indirect Action Program will be discussed. Recent developments concerning involvement of Japanese cooperation will be described.

Impaired gap junctional communication in mouse embryos of the semi-lethal cross DDK female × C3H male

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Female mice of the inbred strain DDK are normally fertile when mated to DDK males (mean litter size 7·9) but have much smaller litters when mated to males of other strains (e.g. DDK × C3H/Bi yields a mean litter size of 1·0). When cultured *in vitro*, DDK × C3H embryos undergo compaction, but most decompact and die at the 16-cell stage. A few undergo cavitation, but most of these eventually die and fewer than 10% become expanded blastocysts. We examined intercellular communication via gap junctions in DDK and control embryos by injecting Lucifer Yellow into one blastomere of the 8- or 16-cell stage and noting the time necessary for the dye to transfer to all cells of the embryo. This time was significantly slower in DDK × C3H embryos (median 280·5 s) than in DDK × DDK embryos (median 169·0 s), which were themselves significantly slower to fill than were controls (C3H × C3H: median 110 s). Incubation of embryos in the weak base methylamine (thought to improve gap junction communication by raising intracellular pH) significantly decreased the time necessary for Lucifer Yellow to fill all cells of DDK × C3H embryos to a median of 140 s. Incubation in methylamine also improved the proportion of DDK × C3H embryos developing *in vitro* to the expanded blastocyst stage from less than 10–35%.

Could sex-reversed conditions be genetic artefacts?

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Recent studies upon *XY* sex-reversed conditions in the mouse, in man, and in other mammalian species suggest that autosomal and *X* chromosomal factors are involved in sex determination. On this basis, the genetic control of sex determination is much more complex than the simple, switch mechanism originally postulated. However, in one of the mouse sex reversals chromosome 17 deletions are involved; another can be thought to derive from a sub species cross; ovarian development in *Sxr* mice is normally found only in the presence of a translocation, and then more commonly when one *X* is of feral origin; *XY* female wood lemmings have a deleted *X*; and, in man, several examples of male pseudo-hermaphroditism have been found associated with unbalanced chromosome constitutions. The sex-reversed conditions may therefore represent genetic artifacts that do not specifically involve sex determination. It is proposed that any genetic anomaly that retards early foetal or gonadal growth enhances the probability of a gonad undergoing ovarian differentiation despite the presence of a *Y* chromosome. This hypothesis is similar to that proposed by Mittwoch as the mechanism for sex determination.

An abnormality of the extracellular matrix underlies development of spinal neural tube defects in mutant curly tail mouse embryos

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The mouse mutant *curly tail* produces spinal neural tube defects (NTD), in 50–60% of homozygous embryos, via delayed closure of neural folds at the caudal neuropore (Copp, *J. Embryol. exp. Morph.* **88** (1985), 39–54). Embryos destined to develop spinal NTD can be recognized at the stage of 27–29 somites before the time of neuropore closure in normal embryos, by virtue of their enlarged neuropores. The hypothesis was examined that extracellular matrix glycoconjugates are disturbed during development of NTD in curly tail mice. Embryos were labelled with [^{3}H]glucosamine *in vitro* for 5 h, until embryos reached the 27–29 somite stage, and then were assigned to 'future normal' and 'abnormal' groups according to neuropore size. Protein-free glycoconjugates were extracted from various embryonic regions and analysed by ion-exchange chromatography. Of the five molecular species defined by this technique, only hyaluronate showed a difference between normal and abnormal embryos: accumulation of newly synthesized hyaluronate was reduced by 10–20% in the caudal neuropore region, but not elsewhere, in abnormal embryos. Autoradiography of sections of labelled embryos revealed that hyaluronate accumulation is markedly reduced in abnormal embryos in the vicinity of the basement membranes underlying the neuroepithelium, surrounding the notochord and overlying the dorsal surface of the hindgut. These results suggest a primary defect of hyaluronate synthesis, or turnover, in basement membranes during development of NTD in curly tail embryos.

Detecting genetic contamination with the mouse karyotype

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Centric heterochromatin comparisons of many of the commonly used laboratory inbred strains reveal differences in certain chromosomes. These differences can be used as markers to show genetic contamination of one strain by another. An example involving two substrains of the 101 inbred strain will be discussed.

Xcat: an *X*-linked cataract mutation in the mouse

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In a recent experiment in which DBA/2 males were treated with 3+3 Gy (24 h fractionation interval) and mated to untreated T-stock females, a female F_1 offspring expressing a posterior subcortical opacity was shown to be heterozygous for an *X*-linked cataract mutation. Linkage studies have shown the cataract mutation to be 22 cM from the tabby locus. Together with W. Pretsch it was shown that male hemizygotes and female heterozygotes are normal for glucose-6-phosphate dehydrogenase activity. This negates an initial hypothesis that the recovered mutation represents an *X*-linked G-6-PD deficiency and the mechanism of cataractogenesis was due to a disruption of glucose metabolism. We further have shown the lenses of male hemizygotes and female heterozygotes for a G-6-PD deficiency mutation, recently recovered by W. Pretsch, to be normal. Thus we could employ the G-6-PD mutant as an *X*-linked marker. Preliminary results indicate the cataract mutation to be distal to tabby. The cataract mutation is expressed in hemizygous males as a total lens opacity. In heterozygous females expression may vary from a total opacity to totally clear lenses, including intermediate phenotypes. Intermediate phenotypes may vary from an opaque lens nucleus with a normal subcortical region to a normal lens nucleus with an opaque subcortical region. These results suggest the mode of action of the cataract mutation to be endogenous to the lens cells. The site of lens opacity reflects the time in lens development at which the mutant allele is expressed, central opacities being expressed earlier and peripheral opacities later. It is proposed the mutant be assigned the symbol *Xcat* (*X*-linked cataract). *Xcat* represents an additional identified locus which is screened in the dominant cataract mutation test. It is homologous to an *X*-linked cataract mutation documented in

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humans. *Xcat* should prove valuable as an animal model with which to study human hereditary cataract and represents an interesting genetic system with which to study the process of *X*-chromosome inactivation.

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Genetic variation in the response to xenobiotics *in vitro*

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We have developed a new technique for studying genetic variation in the response of primary cultures of somatic cells to xenobiotics *in vitro*. Resident peritoneal macrophages were cultured with the test compound for up to 6 days. Response was quantified by assaying total protein. Macrophages from 5 strains with the Coh^h genotype were significantly more sensitive to coumarin than those from 13 Coh^l strains. While response to most xenobiotics appeared to be under polygenic control, response to sodium butyrate was consistent with control by a single locus, though this has not yet been confirmed. This is a humane and economical alternative to *in vivo* studies of xenobiotic response.

Gene expression in mouse embryos with blocked morphologic development

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Expression of paternal *Gpi-1* and maternal *Pgk-1* was assayed in preimplantation embryos of the mouse, whose morphologic differentiation was inhibited by continuous cytochalasin treatment from day 2 of development onwards. Activation of paternal *Gpi-1* was observed only rarely in cleavage-blocked two-cell embryos at day 5 of gestation, confirming results published by Petzoldt (*Devl Biol.* **113** (1986), 512–516). In contrast, expression of maternal *Pgk-1* was never observed in such embryos. In another set of experiments the *in vitro* expression of paternal *Pgk-1* was assayed in mouse embryos. Paternal *Pgk-1* activation was observed under conditions where attachment of embryos to petri dishes and subsequent morphologic differentiation were possible. However, when attachment and trophectoderm outgrowth were inhibited by culture in hanging drops or serum free medium, activation of paternal *Pgk-1* was never observed.

Radiation-induced aneuploidy in mouse oocytes

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The question of whether ionizing radiation can increase the incidence of Down's syndrome is an important one. The mouse is a convenient animal model to use to investigate this question. Since the majority of oocytes in the ovary are in the primordial and early maturation stages, the effect of low doses of radiation on aneuploidy induction in these cells has been investigated. Young (4-week-old) female mice of the F_1 hybrid strain C3H/HeH × 101/H were given acute X-rays (0·1, 0·3, 0·5, 10. Gy) at 0·76 Gy/min or chronic gamma radiation (1, 2, 3 Gy) at 0·0001 Gy/min. Four or eight weeks after irradiation oocytes at the metaphase II stage were scored for numerical and structural chromosome anomalies. The frequency of chromosome anomalies increased with dose after both acute and chronic irradiation. After chronic irradiation, however, a humped dose–response was found. The reduction in response at the highest dose given of chronic irradiation may be related to the high sensitivity to cell-killing of primordial oocytes.

Distribution of loci on the mouse *X* chromosome and comparison with the human *X*

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The distribution of known loci on the genetic map of the mouse *X* shows relative crowding of loci in the centre and wider spacing at both proximal and distal ends. *In situ* hybridization has shown that, in the proximal region, the *spf* (*or Oct*) locus is in band A2 and *Hprt* in band A6. Other data from translocated breakpoints and recent *in situ* hybridization of the *jp* (or *Plp*) locus (Dautigny *et al.*, *Nature* **321** (1986), 867) provide information on the position of loci at the distal end of the *X*. Taken together, the results suggest that the apparent wide spacing of loci at the distal end is due to preferential chiasma formation in the F bands, but at the proximal end there is a true shortage of known loci. Comparison of mouse and human *X* chromosomes shows conserved segments. One group of loci from *Tfm* to *Ags* is found on proximal Xq in man and in the centre of the mouse *X*. A segment including *Hprt* and *G6pd* is distal to the first group in man but proximal in mouse, and loci which are on Xp in man are found at both ends of the mouse *X*, with a possible third group in the centre. Such comparisons enable predictions of the positions of so far unmapped loci, and postulations of possible mouse homologues of human genetic diseases.

The *steel* locus in Harwell mice

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The *steel* locus is highly mutable. Recently, to the Harwell alleles Sl^{con}, Sl^{gbH} nine new ones have been added Sl^{9H}–Sl^{17H}. The *steel* locus concerns hair colour, fertility, viability and haematopoiesis. Sl^{con} homozygotes and most compounds are grey. Other homozygotes and compounds are usually black-eyed-whites, but Sl^{11H}/Sl^{17H} are grey and white. Sl^{17H}/Sl^{con} is unique, spotted agouti with pigmented genital papilla. Fertility of the double mutant is usually reduced in males or females or both, but Sl^{17H}/Sl^{con} (again) and Sl^{11H}/Sl^{17H} are notable with fertile males and females. To haematologists Sl/Sl^{d} mice (Jackson Lab.) are well known for macrocytic anaemia and inability to support the growth of colonies of haematopoietic cells in the spleens of X-irradiated mice. Loutit *et al.* 1986 (*Int. J. rad. biol.* **50**, 1103) show that Harwell Sl^{con}/Sl^{con} and Sl^{con}/Sl^{gbH} have anaemia but no defect of colony formation. Tests to date of newer compounds indicate that most are colony formers but Sl^{17H}/Sl^{17H} like Sl/Sl^{d} poorly so. When lethally irradiated and then grafted with normal bone marrow, those not too radiosensitive accept the graft and recover, but the erythrocytes from the graft exhibit macrocytosis. *Sl* alleles thus influence erythropoietic cells through the environment. From work on Sl/Sl^{d} mice others have deduced an environmental defect for earlier pluripotent haematopoietic stem cells. This cannot be shown in radiation chimaeras induced in Harwell Sl^{*}/Sl^{*} mice unless the donor haematopoietic cells are from compound W^{*}/W^{*} mice. Normal performance of stem cells thus usually depends on interaction of wild-type alleles at the two loci producing intrinsic and extrinsic factors respectively. Defect of either system alone is generally insufficient, according to this test, to impair stem cell function.

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The dominant lethal assay using a battery of strains of mice.

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There is increasing awareness of the effects of genetic variability in the materials used in genetic toxicology testing. Various authors, for instance, have pointed to the lack of experiments to determine the relative sensitivities of various strains in dominant lethal studies. The induction of dominant lethality following oral dosing of males with 200 mg/kg of cyclophosphamide was investigated using a factorial experimental design. Males from three genotypes, BALB/c, CBA/Ca and CBA/Ca × C57BL/6JF$_1$ hybrid (CBB6F$_1$), were mated to six females of the same genotype as the males over 3 weeks. Cyclophosphamide reduced the mating frequency of the BALB/c and

CBA/Ca males. The total number of implants/female was reduced in all three genotypes with the greatest effect in the first 2 weeks after the males were treated. The proportion of early deaths/litter was significantly increased in CBA/Ca and CBB6F$_1$ but the increase was smaller and non-significant with BALB/c. There was a high incidence (29·8%) of early deaths in the control BALB/c females. The implications of the use of a factorial design in dominant lethal assays for the detection of strain variation in mutagenic response without an increase in animal usage is discussed.

This research was supported by the Ministry of Agriculture Fisheries and Food.

Y-chromosomal and non-*Y*-chromosomal factors influencing testis size – some possible implications.

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Differences in testis size between two inbred strains of mice could be shown to be affected by the origin of the *Y* chromosome, the *X* chromosome, the autosomes and by maternal factors. The relative contributions of the different components varied with age. The available data on ovary size showed a correlation with testis size, suggesting that non-*Y*-chromosomal factors affecting testis size may influence the growth of the gonad in both sexes. Taking into account the relative lengths of the *Y* and the *X* chromosomes in relation to total chromosome length, it was concluded that the relative effect on testis size by *Y*-chromosomal loci was greater than that of X-chromosomal loci, and that of *X*-chromosomal loci greater than that of autosomal loci. The question arises as to whether these loci might be identical with testis-determining loci. Data on ethnic differences in human testis size and the cytogenetics of hermaphroditism would seem to support such a relationship.

Changes in DNA methylation during mouse embryogenesis

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Stage- and tissue-specific global demethylation and remethylation occur during embryonic development. The egg genome is strikingly undermethylated and the sperm genome relatively methylated; thus methylation could be a molecular mechanism for imprinting. During preimplantation development there is a decrease in methylation and on the fourth day of pregnancy DNA from the whole blastocyst or from isolated inner cell mass (ICM) cells is strikingly undermethylated. Onset of DNA methylation may be detected in isolated ICM DNA around the time of implantation and methylation progressively increases in post-implantation embryonic lineages. Extra-embryonic trophectoderm and primary endoderm are delineated at a time when methylation is very low, and onset of methylation occurs later in extra-embryonic DNA and to a lesser final extent. Independent methylation in different lineages may be correlated with differential programming. Foetal germ-cell DNA is markedly undermethylated, like blastocyst DNA, and *X*-chromosome reactivation in the female germ line is not associated with a specific demethylation event. It is proposed that the germ-cell lineage escapes methylation, being set aside before extensive methylation occurs.

Data suggesting that a gene(s) in the pseudoautosomal region may be involved in *tda-1 XY* sex reversal

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tda-1 XY sex reversal occurs when the *Y* chromosome of at least some populations of the European house mouse, *Mus musculus domesticus*, is placed into the C57BL/6J (B6) genome. All *XY* foetuses develop either ovotestes or ovaries. Eicher and colleagues have proposed that the testis determining gene of the *M. m. domesticus Y* (Y^{dom}) interacts improperly with a putative gene called *testis-determining, autosomal-1* (*tda-1*) resulting in differentiation

of only ovotestes or ovaries and never a normal testis. Although *XY* progeny possess only ovotestes or ovaries as foetuses, growth of these gonads vary considerably such that adults may possess bilateral testicular gonads (male phenotypes), be overt hermaphrodites or possess bilateral ovarian gonads (female phenotypes). Progeny data obtained from a colony of B6 mice possessing the Y^{dom} chromosome (*B6.*Y^{dom}*/Na*) suggest that the percentages of adults in the three phenotypic categories have remained relatively constant from the N4 backcross generation. The steroid sulphatase gene was used as a genetic marker for the pseudoautosomal region of the *X* and *Y* chromosomes. By crossing B6.Y^{dom} male phenotypes to steroid sulphatase-deficient C3H/An females, then backcrossing the F1 male phenotypes to C3H/An or B6 females and testing the foetal progeny for steroid sulphatase activity, data were obtained suggesting that some progeny were being lost from sperm derived from the *X* and *Y* chromosomes that had undergone recombination. One explanation is that a gene(s) in the pseudoautosomal region is lost during crossing-over and recombination resulting in reduced viability. A similar situation may exist in the B6.Y^{dom} strain and may partially explain the constant percentages of adult phenotypes. Work is in progress to gain further insight on the phenomenon by using recombinant DNA probes specific for the murine *Y* chromosome and with probes believed to be specific for the *X*-*Y* pairing/recombination region.

The effect of hybridization on mandible morphology in an island population of the house mouse.

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In April 1982, R. J. Berry and his associates introduced house mice from the island of Eday in the Orkney archipelago on to the Isle of May off the Fife coast of Scotland. This introduction has provided an excellent opportunity to study hybridization between morphologically differentiated populations of the house mouse. Significant changes due to inherited factors were observed to take place over a very short period. This study shows unequivocally that hybridization can be a considerable factor in the evolution of natural populations.

The role of melanocytes in the development of the inner ear, studied using the viable dominant spotting mouse mutant

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The white areas of *viable dominant spotting* (W^V/W^V) mice have no recognizable melanocytes, so this mutant was chosen to study the effect of a lack of melanocytes on the development of the cochlea. In normal animals, melanocytes form the intermediate cell layer of the stria vascularis in the cochlea. During development the simple cuboidal epithelial marginal cells extend many processes and interdigitate extensively with the mesenchymal intermediate cells below, and capillaries are incorporated into the stria. This process of differentiation immediately precedes the first sign of functional activity of the stria: onset of the endocochlear potential, a d.c. resting potential which eventually reaches about 100 mV in the endolymph facing the marginal cells. In most mutants, however, the marginal cells begin to differentiate but fail to interdigitate normally with the cells below, remaining as a distinct cell layer throughout development, and no endocochlear potential can be recorded. Strial capillaries are significantly thinner in the mutants compared with controls. Normal capillary size seems to be associated with the presence of melanocytes, because in a few mutants the basal turn stria is pigmented and has normal blood vessels, while the apical turn stria shows no pigmentation and markedly thin capillaries.

Two separate *Y*-chromosome effects on the expression of male-specific antigens in the mouse

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The expression of male-specific antigens was compared in male mice from various congenic lines carrying *Y* chromosomes derived from two inbred strains, CBA/FaCamSt and C57BL/FaSt. Cytotoxic T-cells from mixed lymphocyte cultures were significantly more effective in killing spleen lymphocyte target cells carrying the CBA-derived *Y*-chromosome. In contrast, a sperm/sheep red-blood cell rosette serological absorption assay using a monoclonal antibody showed significantly lower expression of male-specific antigen on spleen lymphocytes with the CBA-derived *Y* chromosome. These experiments show that there are variant Y-chromosomal alleles at separate loci controlling the expression of H-Y antigen and of male-specific antigen (as defined by the antibody), and that the two techniques recognize immunological determinants which differ in some way. The possible relationship of these loci to previously defined functional effects of the *Y* chromosomes (*Genet. Res.* **47** 1985, 29–34) remains to be established.

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