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Expression of human α_1-antitrypsin in the mammary gland of transgenic mice.*

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Our objective is to entrain the mammary glands of transgenic sheep to produce commercially important proteins. The efficient tissue specific expression of the sheep milk protein gene, β-lactoglobulin, in mouse milk has demonstrated the value of transgenic mice as a model for our sheep work (*Nature* **328**, 530, 1987). Hereditary or smoking-induced emphysema is a consequence of having inadequate levels of α_1-antitrypsin to control the activity of alveolar elastase. Alpha$_1$-antitrypsin (α_1-AT) is a prime candidate for production by biotechnological means as the dosages required for treatment are beyond the supplies currently available by isolation from human plasma. A hybrid gene, in which human α_1-antitrypsin cDNA sequences have been inserted into the 5′-untranslated region of the sheep β-lactoglobulin gene, has been microinjected into the pronuclei of fertilized mouse eggs. Four lines of transgenic mice have been established. Two of the lines arose from the segregation of the two independent integration sites present in one of the founder mice. The presence of human α_1-antitrypsin in transgenic mouse milk has been detected by radioimmunoassay (RIA) in two of the three lines analysed. This result has been confirmed by SDS–PAGE and Western blotting which also shows that the foreign protein is the correct molecular weight for human α_1-AT. RNA from liver and mammary tissue has been examined by Northern blot analysis. Although no abundant human α_1-AT specific transcripts have been seen in either of these tissues, a transcript of $\sim$ 2 kb has been consistently observed in mammary RNA from one line.

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Dendrograms inferring ontogenetic relationships between murine tissues from the variability of phenotypic mosaics of PGK-1B and PGK-1A expressing cells

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Statistical derivations of developmental stages of organogenesis, 'backwards' from measurements of between- and within-individual variation of genetically marked cellular mosaics, require sufficiently large bodies of data. Recently confirmed observations (Bücher, Rabes & Nielsen, *Cancer Res.* **42**, 3220, 1983; Wareham & Williams, *J. Embryol. exp. Morph.* **95**, 239, 1986) of site-specific discordant mosaic compositions in murine liver lobes were extended by increasing the number of individuals tested, 40-90 female mice homozygous at *Xce* but heterozygous at *Pgk-1*, and by testing other organs. Sampled tissues were sorted according to similarity between individuals of measured mosaic compositions (% PGK-1B of total activity) by 'Clustan' computer procedures. Squared euclidian distances, covariances and product-moment correlations served as parameters. Assuming, as a first approximation that, according to multistage models of organogenesis, (1) similarity of mosaic composition reflects sharing of developmental stages but (2) only differentiation events which happen to rather few cells are discriminated, results are presented for discussion in three categories. (1) Similar at lower variance: white and red cells, lymph nodes and spleen; brain sampled symmetrically at specified sites; duodenal segments. (2) Similar

vs. dissimilar segments at higher variance: liver lobes in three groups; pancreas lienal tail vs. mesenterial tail vs. head at bile duct vs. head at lower duodenal site; lung lobes right vs. left lobe. (3) Dissimilar: m. masseter right vs. m. masseter left; m. pectoralis right vs. m. pectoralis left.

Genetic control of male-specific membrane proteins of the mouse

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There has been much interest in male-specific (H-Y) antigens and their possible roles in testis determination and/or in spermatogenesis, but progress has been severely hampered by the absence of an adequate biochemical characterization of the antigens. We have been following an alternative approach by making cell membrane preparations from mouse liver, carrying out high-resolution PAGE separations, and searching for protein bands which are present in the male but are not apparent in the female. Such proteins might be antigenic in the female, and hence also detectable as male-specific antigens, but this has yet to be demonstrated. We have identified a complex of such male-specific proteins with acidic iso-electric points around pH 4·65. There is considerable variation between standard inbred strains, both in the pattern of bands which are present in the male and in which of these bands appear male-specific. There is no evidence from two consomic lines carrying different *Y*-chromosomes for an effect of the *Y*-chromosome on the pattern of the male-specific bands. Both *Sxr* and *Sxr'* sex-reversed *XX* males yield the same male specific bands as normal males, on both the outbred and C57BL/Mcl genetic backgrounds. This demonstrates that the male-specific proteins visualized in these experiments do not correspond directly to the H-Y antigenic specificity detected by cytotoxic T-cells, which has been reported to be absent in *XX Sxr'* males.

Recombinant congenic strains – a new tool for analysis of traits controlled by more than one gene

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The genetic control of a number of important phenomena, including resistance and susceptibility to development of various tumours, is multifactorial. This often poses unsurmountable problems in identification of the genes involved in these processes. The system of recombinant inbred strains (RIS) developed by Bailey, *Transplantation* **11**, 325, 1971, is a very useful aid in linkage analysis of traits controlled by a single gene. With multigenic traits, however, where additive and non-additive interactions of different participating genes determine the resulting phenotype, the phenotypes observed in a series of RIS often form a continuous range. In addition, very similar phenotypes may be caused by quite different genotypes. Therefore the association between the phenotype and the genotype, which is essential for the genetic interpretation of the results obtained with the RIS, is obscured or disrupted, and no relevant information about linkage can be obtained. To overcome these difficulties, a new genetic tool, *the recombinant congenic strains* (RCS, Demant & Harty, *Immunogenet.* **24**, 416, 1986) has been developed. They represent a genetic system consisting of a number of strains produced from two parental inbred strains by limited backcrossing and subsequent brother–sister mating. In contrast to the RIS, which contain even and randomized mixtures (50%) of the genes of the two parental strains, the RCS contain small portions (approx. 12%) of the genes of one parental strain spread among the genes of the other parental strain. This should allow the fixation of individual genes of a multigenic complex in separate RCS, where they can be identified and analysed individually. Several series of RCS are presently under production and preliminary typings of several non-linked enzyme polymorphisms indicate that the segregation of the genes is compatible with the expectation. The potential application of the RCS and the place of the RCS among other genetic tools are discussed.

Are biochemical and morphological measures of genetic distance between inbred strains of mice correlated?

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Wayne & O'Brien (*J. Mammal*, **67**, 441, 1986) studied the genetic distance between 15 inbred strains of mice based on 36–78 biochemical loci, and compared these with the Mahalanobis distances based on 11 mandible measurements, and the elapsed time of divergence of those strains with a common ancestry. They concluded that '... structural gene and morphometric variation of mandible traits are uncoupled ...' and that 'Genetic distance was highly correlated with elapsed time of divergence of the strains ...' while '... morphometric distance ... was not significantly correlated with elapsed time'. Unfortunately, their data were deficient in closely related strains, and they failed to use a linear scale of measurement. The strong association with elapsed time was probably an artifact in the selection of the data. Re-analysis of their data, and analysis of extensive new data suggests that the correlation between genetic and morphometric distances is strong, ranging from about 0·45 to 0·72 ($P < 0·01$) depending on the number and choice of morphometric measurement and the scales of measurement of all variables.

The expression of steroid sulphatase in male and female mice

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In humans, the steroid sulphatase locus (*STS*) has been shown to be subject to *X*-inactivation, although this may not be complete (Migeon *et al.*, *Nature* **299**, 838–840, 1982). Conversely, the mouse homologue, *Sts*, has been reported to escape *X*-inactivation and also to have active *X* and *Y* loci (Keitges *et al.*, Nature (**315**, 226–227, 1985). The published data on the *X*-inactivation of *Sts* and its expression differ not only between species but also between different mouse experiments (see Wiberg & Fredga, *Hum. Genet.* **77**, 6-11, 1987). Our studies on C3H/HeH and 101/H mice and their F1 hybrid show the STS levels in males to be nearly twice as high as those of females (*XY*:*XX* ratio 1·7:1). If it is assumed that the *X* and *Y* linked genes have equal activities these data clearly suggest that the *X*-borne locus in females is subject to *X*-inactivation. Such an assumption appears valid as we find males derived from reciprocal crosses between C3H/HeH and the *STS*-deficient C3H/An substrain to have similar *STS* levels. Further, we find *XO* females to have activities nearly as high as their *XX* siblings, again indicating that the *Sts* locus is subject to *X*-inactivation. The somewhat higher *STS* levels in *XX* females (*XX*:*XO* ratio 1·4:1) might suggest that the locus is only partially inactivated, as indicated in man, but other factors may be responsible.

Expression of allelic structural genes of *Pgk-1* in mice heterozygous and homozygous at sites in vicinity of *Pgk-1*

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'Chromosome imprinting' (CI) causes preferential expression of the maternally derived *X*-chromosome in extraembryonal lineages which are formed *before the occurrence* of lyonization. Another 'parental source effect' (PE) also taking place rather early but quite different from CI is associated with events *occurring at* lyonization. The phenotypic expression of *X*-linked allelic indicators is modified by PE. Consequently it was attributed to accompanying heterozygosity at the *X*-linked *Xce*-locus (references in *Gen. Res. Camb.* **47**, 43–48, 1985). To be demonstrated convincingly, PE, far from 'flip-flop' switching as CI, requires an exact quantification of indicators, parental congenic background and a larger body of data. In order to meet such requirements reciprocal matings started by T. J. Nielsen at Aarhus were continued in Munich using three parentally

homozygous and hemizygous pairs of *Pgk-1*- and *Xce*-alleles as maternal (Xm) and as paternal (Xp) contribution to doubly heterozygous offspring: (A) $Pgk\text{-}1^{b}$, Xce^{a}, (B) $Pgk\text{-}1^{a}$, Xce^{c} and (C) $Pgk\text{-}1^{b'}$, $Xce^{c'}$. The C3H/Aa line are of type (A) whereas (B) and (C) were derived from feral males caught near Aarhus and inbred into C3H/Aa. Measured means x, noted as %PGK-1B red cell activity of reciprocal crosses Xm/Xp, were: (I) = (A)/(B), $x = 30{\cdot}1\,\%$, $n = 37$, S.E.M. = 1·3 %; (II) = (B)/(A), $x = 36{\cdot}8\,\%$, $n = 51$, S.E.M. = 1·7 %. T-test (I) = (II): $t = 2{\cdot}6$, $P = 0{\cdot}01$ establishes PE in Xce^{c}/Xce^{a} females. But substitution of Xce^{a} by $Xce^{c'}$ in (III) = (B)/(C), $x = 52{\cdot}4\,\%$, $n = 99$, S.E.M. = 0·9 % and (IV) = (C)/(B), $x = 54{\cdot}6\,\%$, $n = 100$, S.E.M. = 1·1 % show insignificant PE at $Xce^{c}/Xce^{c'}$. Causes of non-random lyonization, PE and skewed PGK-patterns of oocytes found in London and Munich will be discussed.

Structural and organizational relationships of mouse homeobox genes and their predicted proteins

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Our laboratory has isolated and characterized several homeobox genes which form part of a complex (*Hox-2*) on mouse chromosome 11. Cosmids and cDNA clones were sequenced and analysed to determine the structure of two genes, *Hox 2.1* and *2.6*, in the complex. The predicted protein sequences show substantial identity to other mouse homeobox genes located on chromosome 6. Therefore based on the sequence similarities, intergenic distances, exon structure and predicted protein sizes it appears that a major portion of these two complexes arose by duplication and divergence from a common clustered ancestor. The degree of identity between the potential homologues is not uniformly distributed throughout the entire proteins, but is restricted to specific domains, in addition to the homeobox. These domains are also conserved between different species in that the *Hox 2.1* and *Hox 2.6* genes have a strong identity to a pair of clustered *Xenopus* homeobox genes in specific domains. The patterns of expression in the mouse embryo for the related members is similar, but not identical, and the regions of identity do extend outside of the protein coding region into potential regulatory elements. The data suggest that there has been strong selective pressure to conserve domains which may functionally interact with or complement the homeobox domain, thus generating specificity for any role in DNA binding or protein interactions for homeobox genes.

Haematological aspects of Harwell steel mice

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A complementation map of the steel locus is proposed based on pleiotropic effects noted in homozygotes and double heterozygotes of alleles recently identified independently by Cattanach, Peters and Searle. The alleles can be grouped as 'complete' or 'partial' mutation of a complex locus. Factors considered included macrocytosis of red blood corpuscles and capability to manifest endogenous haematopoietic splenic colonies derived from pluripotent stem cells (CFU-s) scored by a standard test (McCulloch–Till). The now classic Sl/Sl^{d} of The Jackson Laboratory is grossly defective in this ability due, it is supposed, to a fault in the supporting stroma. Of the viable Harwell compounds investigated, Sl^{17H} as homozygote or double heterozygote with any of the 'complete' group is also defective. Sl^{con} and Sl^{pan}, the other two in the 'partial group', compounded with other alleles are as competent as +/+. The kinetics of the fall and rise of CFU-s with time after 5 Gy of X-rays, studied by the reverse Till–McCulloch exogenous test, shows that Sl^{17H}/Sl^{17H} is slower in recovering from its nadir at 1 day exponentially to reach its norm, about 3 times lower than the norm for +/+, which in turn is lower than the norm for homozygous Sl^{con} and Sl^{pan}. Spleen weights vs. time fall precipitously to a minimum at 10 days whence rates of recovery are of similar form to those of CFU-s. Histology confirms that haematopoiesis is abundant in all types inspected. We conclude that 'environment' varies with the genotype and that for haematopoiesis in the spleen visible colony formation is not essential.

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t-complex inversions visualized by in situ hybridization

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The recombination suppressing effect of the mouse *t*-complex is known to be due to the presence of two inversions. The proximal inversion covers the region from brachyury, *T*, to *Tcp-1*, and the distal inversion extends from a point proximal to *tf* to beyond *Tla*. In order both to find the position and extent on chromosome 17 of the *t*-complex itself, and to attempt to locate the two inversions, in situ hybridization to the chromosome was carried out, in animals with or without a *t*-complex, and with the chromosome 17's made distinguishable by the presence of a Robertsonian or reciprocal translocation. The probes used were for the *Tcp-1* locus (proximal inversion), the *Qa-2,3* region (distal inversion) and the *C3* locus (outside the *t*-complex). Both inversions were recognizable by a shift in grain distribution after in situ hybridization. The centre of the grain distribution for *Tcp-1* was in band A1–A2 in *t*-chromosomes and in A3 in normal chromosomes (previous data); for *Qa-2,3* the grain distribution centres were in A3 in *t*-chromosomes and in band C in normal chromosomes. The *t*-complex must therefore extend at least from the A1–A2 band junction to band C, and is thus somewhat larger than previously thought. The locus of complement component 3 (*C3*), genetically several cM distal to the *t*-complex, was in band E1.

Quantitative criteria of gonadal differentiation

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Criteria of gonadal differentiation have traditionally been based on histology. According to Jost, testicular differentiation in mammals begins with the appearance of the primordial Sertoli cell, which in the rat is first seen between 13 and 14 days post-fertilization. However, a significant difference in size between *XX* and *XY* gonads has been observed in embryos aged from 12 days onwards, and other quantitative differences between early *XX* and *XY* embryos have been described in rats, mice and humans. These data challenge the view that the development of male and female embryos is identical prior to the appearance of histological differences in the gonads, and raise the question as to when sexual differentiation can be said to begin. This leads to a consideration regarding the preconditions required for the formation of Sertoli cells in mammals. The model proposed by Eicher & Washburn specifies that the testicular pathway must differentiate earlier than the ovarian pathway, implying a precocious development of the *XY* gonadal rudiment. This suggests that the first difference between testicular and ovarian pathways is expressed in their rate of development, and that the function of sex-determining genes may be to ensure the correct rate for each sex. The available data suggest that *XY* embryos may develop faster than *XX* embryos at various stages of embryogenesis, and that this difference is enhanced in the somatic cells of the gonadal rudiment prior to the differentiation of Sertoli cells.

Glucocorticoid hormone effects on MHC control of carcinogen-induced lung tumorigenesis in mice

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Genes from the Major Histocompatibility Complex (H-2) are involved in tumorigenesis in the mouse. We have shown that the H-2 influence on carcinogen-induced lung tumours is not confined to tumour incidence only but comprises also their histological type and growth rate. It has been reported that the glucocorticoid hormone (GC) mediated control of differentiation of foetal lung is also under H-2 influence. We decided to study the relationship between GC action on foetal lung, the H-2 genotype and lung tumorigenesis. Mice from the strains B10.A and B10, sharing all genes except those of the H-2 complex and known to differ in (1) their susceptibility to lung carcinogenesis and (2) GC mediated control of foetal lung differentiation, were prenatally treated with either GC plus the carcinogen ethylnitrosourea (ENU) or with ENU alone. Assay of tumour development

showed that (1) for the alveolor type of lung tumour both treatments result in a highly significant difference in incidence between strains; (2) for the papillary type of lung tumour the GC/ENU treatment (vs. ENU alone) results in a higher number of tumours in strain B10, whereas in strain B10.A the reverse was found. An analogous strain specific effect of GC treatment on papillary lung tumour size is indicated as well. Thus in these H-2 congenic strains GC treatment has a differential effect on prenatally induced lung tumours: the H-2 linked influence on alveolar lung tumorigenesis remains unaltered, whereas for papillary tumours an H-2 haplotype specific increase or decrease in tumour number (and probably also size) was found.

Ornithine transcarbamylase (*Otc*) activity in mice carrying *X*-autosome translocations

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The presence of a single active *X* chromosome in mammalian cells of both sexes acts to equalize the expression of *X*-linked genes, but results in effective monosomy for these genes. As monosomy is detrimental for mammals, Migeon *et al.* (*Nature* **299**, 838, 1982; *Cell* **29**, 595, 1982) have suggested that transcriptional activity on the single active *X* of female, as well as male, mammals may be enhanced, resulting in a balance between autosomal and *X*-chromosome output. If this is so, then the effect might be mediated through the inactivation centre. Two mouse *X*-autosome translocations, T(*X*;4)37H and T(*X*;11)38H have been used to test this hypothesis. In T37H the locus of ornithine transcarbamylase, *Otc*, has become separated from the inactivation centre and is active in all cells, whereas in T38H, *Otc* remains linked to the inactivation centre and undergoes random inactivation. Thus in T37H, *Otc* gene activity might be half of that in a normal active *X*, whereas in T38H the gene activity is expected to be the same as in a normal active *X*. *Otc* activity has been measured in heterozygotes for T37H and T38H, and for both translocations the gene activity is the same as in a normal active *X*. Thus, either gene activity is not enhanced on an active *X* compared to autosomes, or the effect is not mediated through the inactivation centre.

Genomic imprinting in the mouse: the role of chromosome 7

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When certain translocation heterozygotes are intercrossed, unbalanced gametes with duplications and deficiencies of specific chromosome segments cannot undergo normal complementation but generate lethal or otherwise abnormal zygotes. Factors in the segments concerned seem to be important in genomic imprinting, which fails if these factors are not transmitted through both maternal and paternal pathways. Two translocations, T(7;15)9H and T(7;18)50H, and several genetic markers have been used to investigate failure of complementation involving chromosome 7. This occurs with maternal duplication/paternal deficiency of two separate regions on the chromosome, one proximal to the T9H breakpoint at 7B3, the other distal to it and probably also distal to the albino locus. In both cases there is marked retardation, with postnatal and late foetal death respectively. Complementation also fails when both homologues of the segment distal to T50H (breakpoint 7E3–7F2) are transmitted by the father (paternal duplication/maternal deficiency). The factor concerned may be the same as the more distal maternal one but its effects are more severe, with earlier intrauterine loss. Thus phenotypic consequences of solely maternal or paternal inheritance of these critical regions agree well with the results of nuclear transplantation experiments.

Mutation rates in Peru mice

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Mice trapped in Peru and bred in Cambridge have a spontaneous rate of mutation and chromosome damage much higher than that in laboratory mice (*J. Hered.* **76**, 221–278, 1985). Mice from the third trapping, Peru–Coppock, inbred for 21 generations, have been selected for seven generations for chromosomal damage, reaching 5-fold the initial rate (*MNL* **71**, 18, 1984). These are now minimally inbred as Peru–Wallace (*MNL* **78**, 40, 1987). A specific locus test is under way using Harwell's PT stock (with 7 recessives) and Peru–Wallace mice. So far one possible recessive (under study) and 3 dominants have been seen in 3369 mice. In 1984 I predicted to this Group that I expected to breed 7200 mice before seeing any mutant using Peru–Coppock mice. So selection has been successful. This prediction excluded the rate of residuals from the forebears of the trapped mice. With this same exclusion, estimates of the number of mutants per locus per 10^{-6} gametes are:

Laboratory (Ibid)	dominants	0·54
	recessives	0·67
Peru–Coppock	dominants + recessives	7·65
Peru–Wallace	dominants + recessives	296·82

A breakdown of all data, using comparable mutants, shows that the rate of production of mutants per gamete (not per locus) is nearly 10 times the laboratory rate in Peru–Coppock mice and nearly 100 times the laboratory rate in Peru–Wallace, for recessives; it is 50 times and 1000 times respectively, for dominants.

Personal note: Facilities for keeping Peru mice in Cambridge are inadequate for maintaining the gene-pool. Only small numbers have been requested elsewhere. It is not known how much of the gene pool controls the rate of mutation. There is urgent need for enough stocks of Peru–Wallace to be established elsewhere, and they should be minimally inbred rather than sibmated.

Biochemical evidence for sharing of gene products in spermatogenesis

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We have raised antibodies to *t*-complex polypeptide-1(TCP-1) and show by indirect immunofluorescence staining that the protein is associated with the Golgi apparatus in various tissue culture cell lines. In spermatids, where TCP-1 is most highly expressed, there is staining associated with the developing acrosome. The T^{hp} mutation is a large deletion ($\sim$ 3 cM).in the proximal region of chromosome 17 which includes the *Tcp-1* gene cluster. Half the spermatids of $T^{hp}/+$ males are deleted for the *Tcp-1* gene and yet we find that all the spermatids isolated from such males stain positively with the TCP-1 antibodies. This result has implications for mechanisms of gene product sharing in the syncytium during spermatogenesis.

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