of the year gave the same symptoms; (e) on *L. esculentum* × *L. pimpinellifolium* the symptoms were identical both in the inoculation from the vine and from diseased *I. holstani*.

From *I. holstani* the isolate has so far been transmitted to tobacco (varieties White Burley and Samsun) and to *Petunia*, by sap and by *Myzodes persicae*; to *Nicotiana glutinosa*, *Datura stramonium*, *Vigna sinensis* and *I. holstani* by sap. The percentage infection in the transmission from these species to the same species or to the other species that gave positive results in the inoculation from *I. holstani*, is higher than in the transmission from *I. holstani*.

We are trying to transmit the isolates from the herbaceous plants to grape vine. For this work we use symptomless grape vines, selected during three years and belonging to varieties that appeared to be very receptive to the 'infectious degeneration' in previous experiments on transmission by grafting from vine to vine.

Other work in progress is the identification of the isolates.

No rod-shaped virus particles were seen in a series of observations, using the electron microscope, with exudates obtained by Johnson's method and with drops prepared with Brandes's dipping method both with diseased grape vines (leaves, shoots and roots) and with infected herbaceous plants.

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GENETICS

Gene Action in the *X*-chromosome of the Mouse (*Mus musculus* L.)

Ohno and Hauschka[1] showed that in female mice one chromosome of mammary carcinoma cells and of normal diploid cells of the ovary, mammary gland and liver was heteropyknotic. They interpreted this chromosome as an *X*-chromosome and suggested that the so-called sex chromatin was composed of one heteropyknotic *X*-chromosome. They left open the question whether the heteropyknosis was shown by the paternal *X*-chromosome only, or the chromosome from either parent indifferently.

The present communication suggests that the evidence of mouse genetics indicates: (1) that the heteropyknotic *X*-chromosome can be either paternal or maternal in origin, in different cells of the same animal; (2) that it is genetically inactivated.

The evidence has two main parts. First, the normal phenotype of *XO* females in the mouse[2] shows that only one active *X*-chromosome is necessary for normal development, including sexual development. The second piece of evidence concerns the mosaic phenotype of female mice heterozygous for some sex-linked mutants. All sex-linked mutants so far known affecting coat colour cause a 'mottled' or 'dappled' phenotype, with patches of normal and mutant colour, in females heterozygous for them. At least six mutations to genes of this type have been reported, under the names mottled[3,4], brindled[3], tortoiseshell[5], dappled[6], and 26*K* [2]. They have been thought to be allelic with one another, but since no fertile males can be obtained from any except, in rare cases, brindled, direct tests of allelism have usually not been possible. In addition, a similar phenotype, described as 'variegated', is seen in females heterozygous for coat colour mutants translocated on to the *X*-chromosome[7,8].

It is here suggested that this mosaic phenotype is due to the inactivation of one or other *X*-chromosome early in embryonic development. If this is true, pigment cells descended from cells in which the chromosome carrying the mutant gene was inactivated will give rise to a normal-coloured patch and those in which the chromosome carrying the normal gene was inactivated will give rise to a mutant-coloured patch. There may be patches of intermediate colour due to cell-mingling in development. The stripes of the coat of female mice heterozygous for the gene tabby, *Ta*, which affects hair structure, would have a similar type of origin. Falconer[9] reported that the black regions of the coat of heterozygotes had a hair structure resembling that of the *Ta* hemizygotes and homozygotes, while the agouti regions had a normal structure.

Thus this hypothesis predicts that for all sex-linked genes of the mouse in which the phenotype is due to localized gene action the heterozygote will have a mosaic appearance, and that there will be a similar effect when autosomal genes are translocated to the *X*-chromosome. When the phenotype is not due to localized gene action various types of result are possible. Unless the gene action is restricted to the descendants of a very small number of cells at the time of inactivation, these original cells will, except in very rare instances, include both types. Therefore, the phenotype may be intermediate between the normal and hemizygote types, or the presence of any normal cells may be enough to ensure a normal phenotype, or the observed expression may vary as the proportion of normal and mutant cells varies, leading to incomplete penetrance in heterozygotes. The gene bent-tail, *Bn* [10], may fit into this category, having 95 per cent penetrance and variable expression in heterozygotes. Jimpy, *jp*, is recessive, suggesting that the presence of some normal cells is enough to ensure a normal phenotype, but Phillips[11] reported one anomalous female which showed the jimpy phenotype. Since it showed the heterozygous phenotype for *Ta* this animal cannot be interpreted as an *XO* female; it is possible that it represents an example of the rare instance when by chance all the cells responsible for the jimpy phenotype had the normal gene inactivated.

The genetic evidence does not indicate at what stage of embryonic development the inactivation of one *X*-chromosome occurs. In embryos of the cat, monkey and man sex-chromatin is first found in nuclei of the late blastocyst stage[12,13]. Inactivation of one *X* at a similar stage of the mouse embryo would be compatible with the observations. Since an *XO* female is normally fertile it is not necessary to postulate that both *X*-chromosomes remain functional until the formation of the gonads.

The sex-chromatin is thought to be formed from one *X*-chromosome also in the rat, *Rattus norvegicus*[14], and in the opossum, *Didelphis virginiana*[15]. If this should prove to be the case in all mammals, then all female mammals heterozygous for sex-linked mutant genes would be expected to show the same phenomena

as those in the mouse. The coat of the tortoiseshell cat, being a mosaic of the black and yellow colours of the two homozygous types, fulfils this expectation.

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Genetic Basis for Graft-against-Host Immunological Reactions between Two Inbred Lines of Chickens

IT has been established that the enlargement of the embryonic spleen which follows the injection of adult chicken blood into chick embryos is due, at least in part, to a proliferation of cells derived from the injected blood[1,2]. Cock and Simonsen[3] have shown that virtually no splenic enlargement occurs when the blood-donor and embryonic recipients are members of the same inbred line of chickens. The phenomenon of splenic enlargement seems to be fundamentally immunological in nature, and due to donor cells proliferating in response to those host antigens which differ from any in the donor.

It should be possible, by injecting blood from adult birds from one parental line into F_2-generation and back-cross embryos between two inbred lines, to analyse the antigenic difference of the other parental line. Assuming that the antigens of the parental lines are dominantly determined and that they segregate in crosses between the lines in a Mendelian fashion, then a proportion of F_2-generation embryos, and of embryos of the back-cross to the parent of the blood-donating line, will be expected to lack those genes which determine antigens occurring exclusively in the non-blood-donating line. The proportion of embryos which lack these genes will be $(\frac{1}{4})^n$ in the F_2-generation and $(\frac{1}{2})^n$ in the back-cross, where n is the number of pairs of genes involved. Since splenomegaly will occur only when the recipient embryo possesses antigens foreign to the donor cells, these are also the proportions of embryos in the respective crosses which will show no splenic enlargement. All F_1 hybrids and embryos of the back-cross to the parent of the non-blood-donating line will receive the genes which determine antigens peculiar to the non-blood-donating line and all these embryos will therefore show splenic enlargement. Thus, an estimate of the value of n can be obtained by observing the proportion of F_2 and back-cross embryos which show no splenic enlargement. The genetic basis for this method is essentially similar to that used in analysing histo-compatibility differences between inbred strains of mice using tumour transplantation[4], and skin transplantation[5].

The method outlined above has been used to observe antigenic differences between the Reaseheath C- and I-inbred lines of White Leghorns[6]. Both lines have been brother–sister mated annually for more than twenty generations. Chick embryos were injected intravenously at 15 days of incubation with 0·1 ml. of citrated blood from I-line cocks, and killed 4 days later and their spleens weighed. The embryos injected were: C-line embryos, I-line embryos, the F_2-generation ($CI \times CI$ and $CI \times IC$), and the back-crosses ($C \times CI$ and $C \times IC$, $I \times CI$ and $I \times IC$). In designating the crosses, the male parent is stated first. Two I-line cocks were used as blood-donors to the F_2 and back-cross embryos and experiments with each donor were performed twice. Only a small number of C embryos were available for injection, but the marked splenic enlargements obtained indicate that C tissues are antigenic to I cells. A small number of I-line embryos injected with I-blood showed no splenomegaly. So far, we have had no F_1-hybrids to test, but Cock and Simonsen[3] have obtained splenic enlargement after injecting I-blood into newly hatched $C \times I$ chicks. The patterns of spleen weights obtained after injecting I-blood into F_2 and back-cross embryos were similar in each of the four experiments, and the results have been pooled in Fig. 1. The proportions of spleens in the different crosses showing no enlargement are shown in Table 1, and these results are compared with the theoretical frequencies expected for 1, 2 and 3 pairs of dominant genes determining antigens peculiar to the C-line. The proportions best fit the expectancy for one pair of genes, and the results suggest, therefore, that the C-line carries one antigen (capable of stimulating splenic enlargement) which is absent from the I-line. The results also fit the expectancy if the C-line possesses one dominantly determined antigen and one recessively determined antigen foreign to the I-cells. In this case, the proportion of unenlarged spleens in the F_2-generation would be 18·75 per cent (the proportion falls in a series $(\frac{3}{4})^{n_1} \times (\frac{1}{4})^n$, where n_1 is the number of pairs of recessive genes and n is the number of pairs of dominant genes). The proportion of unenlarged spleens in the back-crosses would remain unchanged. However, until we have other evidence for the

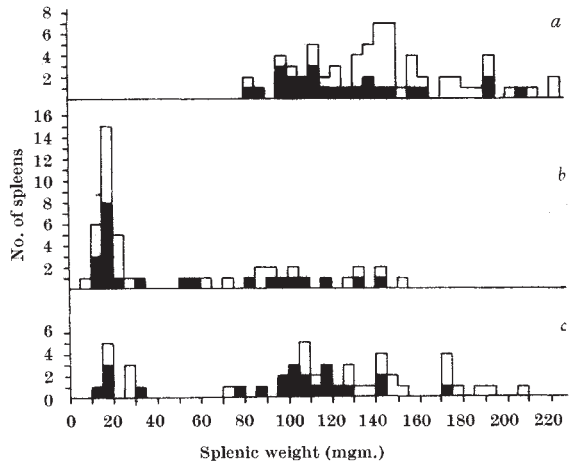

Fig. 1. Distribution of spleen weights obtained after injecting I-line cock-blood into F_2-generation and back-cross embryos. Solid squares, male spleens; open squares, female spleens. (*a*) $C \times CI$ and $C \times IC$ embryos; (*b*) $I \times CI$ and $I \times IC$ embryos; (*c*) $CI \times CI$ and $CI \times IC$ embryos