Under the conditions of these experiments, the physical treatment that a virus vaccine undergoes during the various production stages of virus growth, inactivation and addition of preservative, was shown to be deleterious to the tissue culture cells from which the virus was produced; using a highly sensitive test system we were unable to demonstrate surviving cells. We were unable to demonstrate any breakdown product capable of producing tumours.

From these experiments it seems that the preparation of an inactivated vaccine from virus grown in these cells would carry no risk of inducing malignant tumour growth in hamsters and hence would also carry no risk for a heterologous host, in which there would, moreover, be an immunological barrier.

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GENETICS

Linkage in Man: the Inv and the Lp Serum Type Systems

The new serum type system Lp[1] is a useful genetic marker system, the frequency of the Lp^a gene being approximately 0·18 in Caucasians[2,3] and test reagents unrestrictedly available. Concerning the serum type system Inv (refs. 4 and 5), test reagents have been relatively difficult to obtain, but are now increasingly available[5–8]. The established Inv^a or Inv^{1a} gene frequencies, 0·06–0·10 in Caucasians and considerably higher in other populations, would indicate common use of this marker system in the future. This pre-supposes the elucidation of the genetics of this system, including analysis of linkage relationships. This communication provides data on the Inv–Lp linkage relation.

The material comprised 87 unrelated parents and 20 children from Norwegian families tested for Lp(a) and Inv(a). Of the unrelated individuals 67 were derived from a family material considered earlier as regards Lp(a) by Mohr and Berg[2], and 20 derived from a material concerning *Epidermolysis bullosa* (Gedde-Dahl, in preparation).

Anti-Inv(a) Math. serum and anti-*D* Roehm serum were kindly provided by Dr. A. G. Steinberg, while anti-Lp(a) serum was of our own production (K. B.). The Inv typing was performed as outlined by Steinberg[5] with minor modifications, and the Lp typing as described by Berg[9].

Among the 87 unrelated individuals 13·79 per cent (12 individuals) were found to be Inv(a$^+$) and 33·33 per cent (29 individuals) Lp(a$^+$) (Table 1). Four matings of the type Inv(a$^+$)Lp(a$^+$) × Inv(a$^-$)Lp(a$^-$) were observed among the 40 pairs of parents where both parents were tested. As appears from Table 2, recombination between the Inv and Lp loci must have taken place in each of these families. The Fisher-Finney method[10] gave values of 73·225 units, a linkage score of $-7·233$ and a test value of $-0·845$. The most likely recombination frequency is 50 per cent, that is free recombination. By the Morton-Smith method[11], linkage between the Lp and Inv loci may be excluded with a probability of 0·985, and linkage below 30 per cent with a probability of 0·996. It may be noted that practically all the information, 66 units by the Fisher-Finney method, was derived from a single 12 sib family. In all four families the double heterozygote parent was the mother.

Table 1. Phenotype Distribution of 87 Unrelated Parents

	Inv(a$^+$)	Inv(a$^-$)
Lp(a$^+$)	4	25
Lp(a$^-$)	8	50

Table 2. Phenotype Distribution of Children in Four Families of Mating Type Inv(a$^+$)Lp(a$^+$) × Inv(a$^-$)Lp(a$^-$)

Family	No. of children	Inv(a$^+$) Lp (a$^+$)	Inv(a$^-$) Lp(a$^-$)	Inv(a$^-$) Lp(a$^+$)	Inv(a$^+$) Lp(a$^-$)
Eb XXV	12	2	3	3	4
678	3	1	1	1	
688	3	1	1		1
Eb LXIII	2		1		1

Berg and Mohr[12] found the phenotype Lp(a$^+$) in 34·08 per cent of 314 unrelated Norwegians. 67 of these, namely 23 Lp(a$^+$) and 44 Lp(a$^-$) individuals, are included in the present material. The frequency of the phenotype Inv(a$^+$) has not previously been estimated for the Norwegian population. The distribution in the present material, however, does not differ significantly from our own finding of 14 Inv(a$^+$) individuals among 143 Norwegians not included here (χ^2 : 0·86, $0·5 > P > 0·3$). Both materials combined gave a phenotype frequency of Inv(a$^+$) of 11·30 per cent. These results of Inv(a) will be included in a later publication.

From the gene frequencies of Lp^a 0·1877[2] and Inv^a 0·059 an expected number of 0·8 double backcross matings among 40 random Norwegian matings may be calculated. Actually, 2 such matings were observed among 33 from an unselected family material, and 2 out of 7 in families selected by *Epidermolysis bullosa* and by the number of children.

It appears that the linkage relations of the Inv locus have not previously been analysed beyond an exclusion of linkage closer than 30 per cent recombination between the Inv and Gm loci[13].

Work on the relations between the Lp locus and the loci of the ABO, MNS, Rh, Le, Lu, P, Fy, Jk, K, Hp, Gc, Gm, and Ag systems has not revealed any linkage, but information on some of the relations was scarce[2,9].

The calculations were made by Mr. Dan Wöien, of the University Institute of Mathematics, Oslo, by means of an IBM electronic computer 1620 II.

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Artificial Insemination of Dystrophic Mice with Mixtures of Spermatozoa

Muscular dystrophy in mice (*Dystrophia muscularis*, symbol *dy*) is transmitted as an autosomal recessive[1,2] and is linked to Steel (*Sl*) and Ames waltzer (*av*) in linkage group IV[3–5]. Mice of genotype *dy*/*dy* have pronounced weakness of the hind limbs and die as young adults. Dystrophic male mice do not breed, and dystrophic females may have one or sometimes two litters early in their reproductive life, when mated to normal males. The disease can be recognized in mice 2–3 weeks old and has a characteristic manifestation, described in detail by Michelson *et al.*[1] and Loosli *et al.*[6].

In a previous report[7], litters consisting entirely of dystrophic (*dy*/*dy*) mice were obtained by inseminating dystrophic F_1 hybrid females (F_1 hybrid from 129/Re-*dy*/+ × *C57BL*/6-*dy*/+) (ref. 8) with spermatozoa from F_1 hybrid males of the same genotype. The number of dystrophic females producing litters following artificial insemination approaches that observed with natural