Nash, David. Department of Genetics, University of Alberta, Edmonton, Alberta, Canada. The equivalence of Hairless alleles of reportedly different penetrance.

It has been suggested (Nash, 1965) that the Hairless (H, 69.5, chromosome III) mutant is effectively an inoperative (amorphic) gene. Its recessive lethality and dosage effects in triploids (Gowen 1933) lead to this conclusion. There exists a number of alleles (independent occurrences) of H, some of which have been described as "strong" or "weak" alleles. The presence of great variability in genetic background could account for these differences. However, should qualitatively different alleles exist, the amorphic nature of the gene would be in doubt.

As a simple test of the similarity (or dissimilarity) of some of the various alleles, several independent mutants (H^1, H^2, H^3) have been placed in different inbred genetic backgrounds by a chromosome substitution procedure which produces flies with only their Y chromosomes and the third chromosome carrying H unsubstituted. In addition some flies carry heterozygous fourth chromosomes not derived from the inbred lines.

Table 1

SITE*	Edinburgh			Oregon			Florida		
	H^1	H^2	H^3	H^1	H^2	H^3	H^1	H^2	H^3
	P E	P E	P E	P E	P E	P E	P E	P E	P E
1	46(99)	18(100)	37(100)	29(100)	20(100)	12(100)	1(100)	0(-)	1(100)
2	15(67)	33(100)	35(100)	29(94)	72(97)	31(92)	1(100)	8(100)	2(100)
3	100(83)	100(87)	100(82)	100(20)	100(27)	99(23)	100(8)	100(20)	100(4)
4	73(3)	72(0)	51(5)	81(9)	74(28)	62(15)	40(0)	17(0)	23(0)
5	23(0)	5(0)	22(0)	32(0)	27(0)	32(3)	20(0)	10(0)	20(0)
6	20(0)	7(0)	29(0)	26(0)	15(0)	40(0)	37(0)	22(0)	44(0)

The inbred lines were obtained from the Department of Genetics, University of Birmingham, England.

P = Penetrance = Percentage of bristles absent from site.
E = Expressivity = Percent of affected sites lacking a vestige (see Nash, 1965).

*Bristle sites
1 = Ocellar
1 = Anterior Orbital
3 = Postvertical
4 = Posterior Orbital
5 = Posterior Vertical
6 = Anterior Vertical

Compared with the large differences contributed by background modifiers in different backgrounds, there is so little difference between the effects of the three alleles upon head macrochaetae in the same background that it is concluded that the three alleles are identical to each other in morphological effects. It is notable that, where differences are found within a given background, they do not consistently support Plunkett's (1926) original description of a more extreme effect for H^2.

These differences (between the alleles in a given background) are quite likely due to the residual dissimilarity of genetic background. This possibility has been demonstrated using two stocks (A and B) carrying H^1 of known identical origin and separated by inbreeding for some thirty generations. The same substitution procedure was used as before.

Table 2

SITE*	Edinburgh		Oregon		Florida	
	A	B	A	B	A	B
	P E	P E	P E	P E	P E	P E
1	55(100)	83(100)	40(98)	37(100)	0(-)	1(100)
2	74(98)	48(95)	92(100)	85(97)	17(100)	3(75)
3	100(93)	100(99)	100(58)	100(68)	100(9)	99(28)
4	63(15)	50(7)	90(48)	73(43)	37(0)	40(0)
5	24(0)	75(0)	34(0)	29(0)	44(0)	12(0)
6	27(0)	16(0)	31(0)	19(0)	51(0)	14(0)

See Table 1 for notes.

Differences between stock A and B and between them and H^1 in Table 1 are as great as the differences between alleles in Table 1.

A critical test for the inactivity of H mutants could be performed using a cytologically identifiable deficiency of the $^{+H}$ region. There is, as yet, no suitable deficiency available to me.

References:

Gowen, J. W. (1933). Amer. Naturalist 67, 178.
Nash, D. (1965). Genet. Res., Camb. 6, 175.
Plunkett, C. R. (1926). J. Exp. Zool. 46, 181.

Schalet, A. University of Connecticut, Storrs, Connecticut. Additional data concerning genes in the proximal region of the X chromosome of Drosophila melanogaster.

The following information is presented as a supplement to the research note of Schalet and Finnerty (DIS-43:128). Correction: Lethal A7 of Kaplan and Lethal N-30 of Himoe are alleles. A7/N-30 is lethal.

Additions: Lethal A7 and lethal N-30. Males carrying either of these lethals and a $y^+Yma\text{-}1^{126}$ chromosome, in which the deletion that eliminated the $ma\text{-}1^+$ region from the Y involved a break close to the normal allele of the lethal locus, often exhibit malformations of the eyes, legs, wings and antennae. In extreme cases a leg may be branched or completely duplicated, an antennae or arista may be duplicated or triplicated. Males carrying these lethals and a $y^+Yma\text{-}1^{106}$ chromosome or males carrying a $y^+Yma\text{-}1^{126}$ chromosome and a more proximately located X chromosome lesion, including the loci from lethal DCB1-35c thru bb, fail to show these abnormalities. When either lethal A7 or N-30 is made heterozygous with a $y\ bb^{1158}$ chromosome (Oak Ridge), some females show malformations.

Little fly (lf) of Fahmy (DIS-33) located at 68.1 according to Lindsley and Grell is allelic to lethal t2-14a of Kaplan. lf/t2-14a is viable and lf in phenotype.

Lethal LV7 of Kaplan. LV7/Y males and LV7/deficiency females occasionally survive. They are phenotypically identical with the description given for uncoordinated (unc) found by Fahmy and placed at 65.9 by Lindsley and Grell.

Relative positions of sw and mel. In the previous note mel was tentatively placed between sw and ma-1. This position is made more secure on the basis of obtaining a single crossover between sw and mel. Females of the genotype y v sw $ma\text{-}1^2$/y mel $1(1)20\cdot Dp(1)sc^{V1}y^+$ were crossed to Y/y v f mel males and incubated at 24-25 degrees. From eggs laid during the first six days, 280/10,711 (2.6%) of the female offspring represented crossovers between mel and y^+. This frequency is consistent with the earlier data: sw--ma-1 (0.5%); ma-1--su-f (2.2%); and su-f--y^+ (0.06%). At 24-25 degrees only about 15% of the sw males eclose and all show the expected wing and/or eye abnormalities. If the correct order is sw mel ma-1, then single crossovers between sw and lethal(1)20 can produce three classes of viable males with respect to the mel and ma-1 markers: 1) $ma\text{-}1^2$ 1) mel $ma\text{-}1^2$ and 3) mel. Since some ma-1 offspring segregating from non-ma-1 mothers are subject to a maternal effect, all non-sw male offspring were crossed to y v f ma-1 attached-X females to confirm the presence or absence of ma-1. The two mel classes can be distinguished from the mel^+ class by the body color and wing phenotype of mel, and the "dull red eye color" of the mel class is distinguishable from the mel $ma\text{-}1^2$ class which exhibits a "brownish red eye color" similar to the phenotype of $ma\text{-}1^2$. (The eye color of the v mel combination is usually quite similar to v alone and readily separated from the v mel ma-1 combination which appears very much like v ma-1.)

There were 65 y sw^+ male offspring but only 1 was mel^+ and it also proved to be $ma\text{-}1^2$. Of the remaining males, 10 failed to breed, ten were mel $ma\text{-}1^2$ and 44 were mel $ma\text{-}1^+$. If the parental males had produced equal numbers of functional X and Y bearing sperm then the 65 sw^+ males would represent a crossover frequency between sw and lethal(1)20 of about 1.2%. From the earlier positioning of lethal(1)20, less than 0.1% to the left of su-f, this is about one half the expected frequency. This reduction is probably not due to lowered viability of y mel or y mel mal^2 males, since the total of 280 recombinant females included 156 that were y mel in phenotype and only 124 that were wild-type in appearance. By taking 2.6% as a better approximation of the crossing over between sw and lethal(1)20, mel is located closer to sw, and approximately 1/11 of the map distance of 0.5 units between sw and ma-1.