Table 2. **Results of inoculating F_3 plants of individual F_2 plants from the crosses of OX686 (*Rsv? Rsv?*) × L78-379 (*Rsv1 Rsv1*) and OX686 × OX670 (*Rsv2 Rsv2*) with the G1 or G4 strain of soybean mosaic virus**

F_3 classification[a]	F_2 genotype	Expected ratio	OX686 × L78-379 Observed no.	OX686 × L78-379 Expected no.	OX686 × OX670[b] Observed no.	OX686 × OX670[b] Expected no.
All R	*RsvRsv*[c]	4	19	20.25	15	14.25
R and STN	*Rsvrsv Rsv?Rsv?*	2	11	10.12	8	7.12
R, STN, and M	*Rsvrsv Rsv?rsv?*	4	20	20.25	16	14.25
R and M	*Rsvrsv rsv?rsv?*	2	9	10.12	7	7.12
All STN	*rsvrsv Rsv?Rsv?*	1	5	5.06	2	3.56
STN and M	*rsvrsv Rsv?rsv?*	2	12	10.12	6	7.12
All M	*rsvrsv rsv?rsv?*	1	5	5.06	3	3.56
χ^2				0 63		1.31
P				.99		95–.98

[a] R = resistant (no mosaic or necrosis); STN = stem-tip necrosis; M = mosaic (susceptible).

[b] OX686 STN (29 plants); OX670 R (10 plants).

[c] *Rsv1/rsv1* for first cross; *Rsv2/rsv2* for second cross.

segregation, indicating that *Rsv3* is not linked with *Rsv1* or *Rsv2*.

In the F_2 generation of the crosses of OX686 with Harosoy, there were 80 *Rsv3__ Rmd__*, 27 *Rsv3__ rmdrmd*, 27 *rsv3rsv3 Rmd__*, and 6 *rsv3rsv3 rmdrmd* plants. This indicates independent segregation for the two gene pairs that entered the cross in the coupling phase.

Discussion

Cho et al.[3] suggested that necrosis reaction and mosaic resistance in the cultivar 'Kwangkyo' (PI 406710) might be effects of the same gene for SMV resistance. The dominant Kwangkyo gene could be an *Rsv* gene similar to the *Rsv3* gene. Kwangkyo produces a necrotic reaction with strains G5, G6, and G7 and a symptomless reaction with strains G1, G2, G3, and G4,[4] whereas strains G1 through G7 produce some STN in OX686, with G1 and G4 showing a more severe reaction (J. C. Tu, unpublished results).

Local lesions often occur on the primary leaves of plants that develop stem-tip necrosis when they are inoculated with necrosis-causing strains of SMV[4] (J. C. Tu, unpublished results). This supports the concept that STN is a hypersensitive (resistant) reaction. Thus, if stem-tip necrosis is the result of an incompatible reaction between the soybean and an SMV strain, it should not be classified as a "susceptible" reaction. Considering STN as a resistant reaction affects the genetic interpretation. For example, Lim[8] and Kwon and Oh[7] considered STN to be a susceptible reaction, whereas Kiihl and Hartwig[6] rated STN plants as being resistant.

As noted by Cho and Goodman,[4] the use of *Rsv* genes to give resistance to mosaic carries the risk that SMV strains may occur which result in a stem-tip necrosis reaction and plant death. The epistatic effect of *Rsv1* and *Rsv2* on *Rsv3* we observed indicates that the use of two *Rsv* genes to provide mosaic resistance in a cultivar should reduce the risk of this happening.

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The Gene for the β Subunit of the Follicle-Stimulating Hormone Maps to Bovine Chromosome 15

R. Fries

Using a 1,500-bp cDNA specific for the β subunit of bovine follicle-stimulating hormone as a probe for in situ hybridization, I assigned the corresponding locus, *FSHB*, to bovine chromosome 15q24-qter. Previous assignments of the loci for β-globin (*HBB*) and parathyroid hormone (*PTH*) to chromosome 15, along with my assignment, indicate the evolutionarily conserved synteny of these loci on the short arm of human chromosome 11 and on bovine chromosome 15. The analysis of the distribution of the autoradiographic silver grains suggests that *FSHB* is more distally located than are *HBB* and *PTH*, contrary to the order of these loci on the short arm of human chromosome 11.

Attempts have been made to establish systematically the gene maps of domestic species of animals by identifying evolutionarily conserved segments of chromosomes.[1,2] We have shown the conserved linkage of the loci for parathyroid hormone (*PTH*) and β-globin (*HBB*) in cattle and have assigned the linkage group to bovine chromosome 15.[4] *PTH* and *HBB* map to the short arm of chromosome 11 (11p) in humans and to chromosome 7 in mice, respectively. From a comparative point of view, human 11p can be divided into at least two segments: a distal region containing *PTH* and *HBB*, corresponding to a part of murine chromosome 7, and a proximal region containing the loci for catalase (*CAT*) and the β-polypeptide of the follicle-stimulating hormone (*FSHB*), corresponding to a part of murine chromosome 2.[10] Genes from both regions map to chromosome D1 of the cat.[8] Because the bovine gene for the β-polypeptide of *FSHB* has been cloned, I attempted to determine its chromosomal position in cattle by in situ hybridization to delineate the extent of the conservation of human 11p on cattle chromosome 15. I present evidence that *FSHB* also maps to bovine chromosome 15.

Materials and Methods

I obtained chromosome spreads from fibroblast cell lines by the mitotic shake-off technique. The fibroblast cell lines had been established from fetal bovine tissue. Metaphase spreads were QFQ-banded and

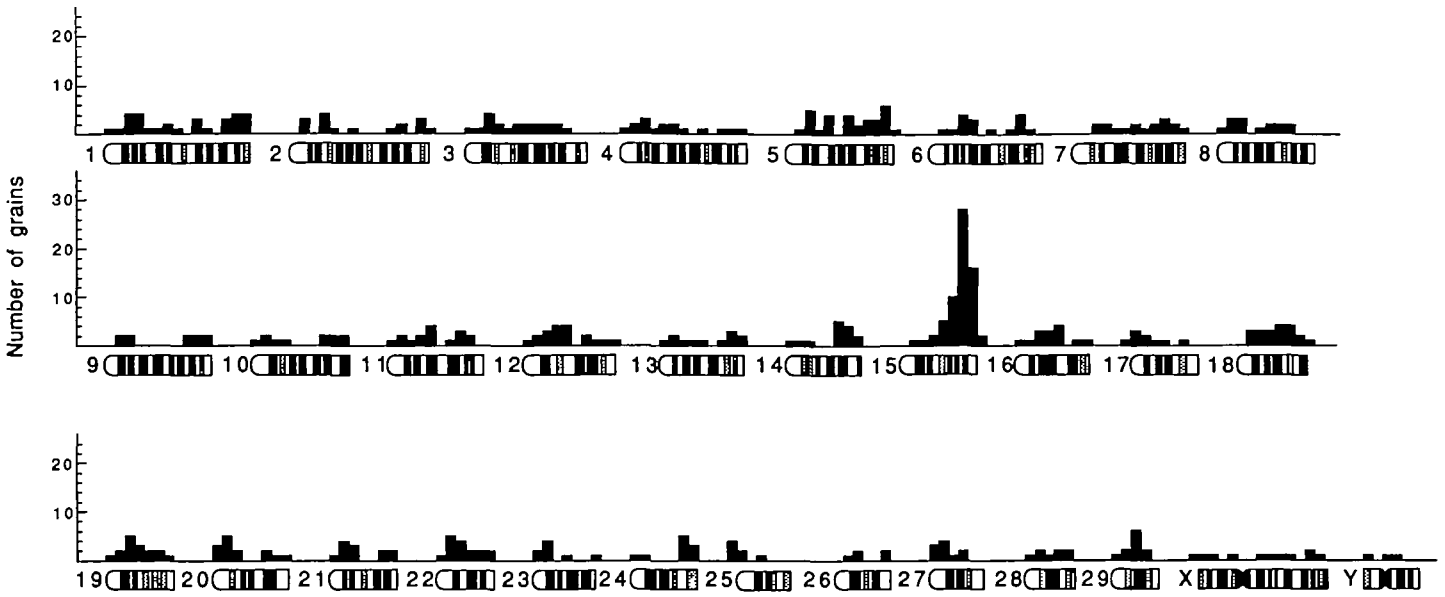

Figure 1. Histogram showing the distribution of autoradiographic silver grains from 102 metaphase spreads after in situ hybridization with an *FSHB*-specific sequence.

photographed prior to in situ hybridization, as described elsewhere.[3] The probe for the *FSHB* locus was a 1,500-bp bovine cDNA insert excised with *Hind*III and *Eco*RI from a recombinant pGEM3 plasmid.[6] The *FSHB*-specific sequence was labeled by the random priming method[1-3] to a specific activity of 2×10^8 dpm/μg DNA, using three tritiated nucleotides. The labeled DNA was separated from unincorporated nucleoside triphosphates with a Sephadex G-50 column and was recovered by ethanol precipitation in the presence of alkali-sheared salmon sperm DNA (10 μg). In situ hybridization was done according to the method of Harper and Saunders,[5] with modifications as described by Fries et al.[3] The concentration of the probe in the hybridization solution was 20 ng/ml. Autoradiography of slides was done for 14 to 20 days.

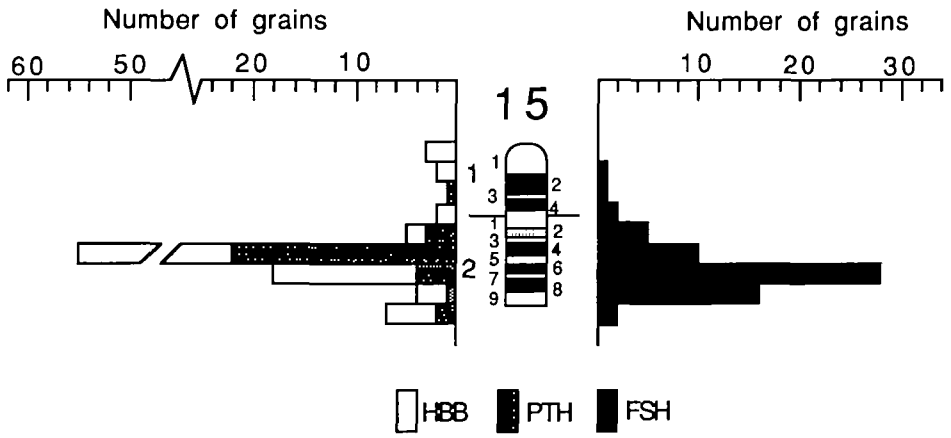

Figure 2. Composite histogram of silver grain distribution over chromosome 15 from three in situ hybridization experiments, using three probes. Data for *HBB* and *PTH* from Fries et al.[4]

Results

Figure 1 shows the distribution of autoradiographic silver grains after hybridization in 102 Giemsa-stained metaphase spreads. All spreads were QFQ-banded before in situ hybridization to allow chromosome identification. The relative position of silver grains along the Giemsa-stained chromosomes was estimated, the chromosomes were identified, and the grains were assigned to one of 324 units scaled to the average diameter of a silver grain (0.55 μm). The band idiogram shown in Figure 1 was prepared according to the Reading standard.[9] Of 606 silver grains touching a chromosome, 10.7% were associated with chromosome 15. This chromosome amounts to 3% of the haploid genome length. A total of 83.1% of the grains found on chromosome 15 were concentrated over bands q24-qter. This grain distribution indicates the location of *FSHB* within these bands, most probably in bands q26 and q27 (Figure 2).

Discussion

Using in situ hybridization with an *FSHB*-specific probe, I assigned *FSHB* to bovine chromosome 15. This assignment was partly expected because of our previous assignment of *PTH* and *HBB* to bovine chromosome 15[4] and because *HBB, PTH,* and *FSHB* are syntenic in humans. Thus, it is quite likely that the gene content of at least half of human 11p is conserved on cattle chromosome 15, with *HBB* mapping to the tip and *FSHB* mapping to about the middle of human 11p. *CAT,* another locus very close to *FSHB* in the central region of 11p, has recently been shown to be syntenic with *HBB* and *PTH* in cattle (J. Womack, personal communication). Comparative mapping therefore allows the provisional assignment of gene loci located between *HBB* and *FSH* on the human gene map to cattle chromosome 15.

With *FSHB, HBB,* and *PTH* having been regionally mapped to cattle chromosome 15, it is possible to draw conclusions about the position of the gene loci relative to the centromere. Figure 2 summarizes the silver grain distribution of the previous *PTH* and *HBB* assignments[4] and of my experiment. This silver grain distribution suggests a more distal location of *FSHB* relative to *PTH* and *HBB.* This order differs from the order of the same genes and the

centromere on human 11p, a fact that may be explained by the reshuffling of chromosome segments during evolution, as a result of which the orientation of gene loci to a centromere was not necessarily maintained.[7]

HBB/PTH and *FSHB/CAT* are syntenic in humans and cattle but not in mice.[10] Womack and Moll[11] found more linkage discordances between humans and mice than between humans and cattle. This study accords with their findings. It indicates another example of linkage conservation between cattle and humans that is not conserved between humans and mice.

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A New Black Tarsomere Allele in *Aedes aegypti* (Diptera: Culicidae) from the Island of Grand Comoros

M. Coetzee and N. van Schaik

We received a sample of wild larvae of *Aedes aegypti* (L.) from the Indian Ocean island of Grand Comoros. Several of the adults reared from the larvae had some degree of black scaling on hind tarsomere V, which is normally all white. The results of crosses of the new mutant with normal *A. aegypti* and a strain homozygous for the gene black tarsomere (*blt*) showed that the black scaling is controlled by a single autosomal gene that is allelic to *blt*. The new allele is dominant to black tarsomere but incompletely dominant to the wild type.

Ornamentation of the tarsomeres of *Aedes aegypti* (L.) normally involves large areas of black and white scaling, with hind tarsomere V being completely white (Figure 1a). Edwards[2] described a black-legged form of *A. aegypti* var. *atritarsis* from Accra, Gold Coast. This variety had a marked reduction of white scaling on all legs (Figure 1d). Subsequent work on the genetics of *A. aegypti* demonstrated that this trait is controlled by an autosomal recessive gene designated black tarsomere (*blt*).[1]

We received a sample of wild larvae from the island of Grand Comoros in 1985. A morphological variant with black and white scaling on hind tarsomere V was noted (Figure 1, b and c). All other legs were normal. We report the results of breeding experiments between the new mutant and normal *A. aegypti* and a strain homozygous for black tarsomere (*blt*).

Materials and Methods

We received 10 larvae of *A. aegypti* collected in the wild on the island of Grand

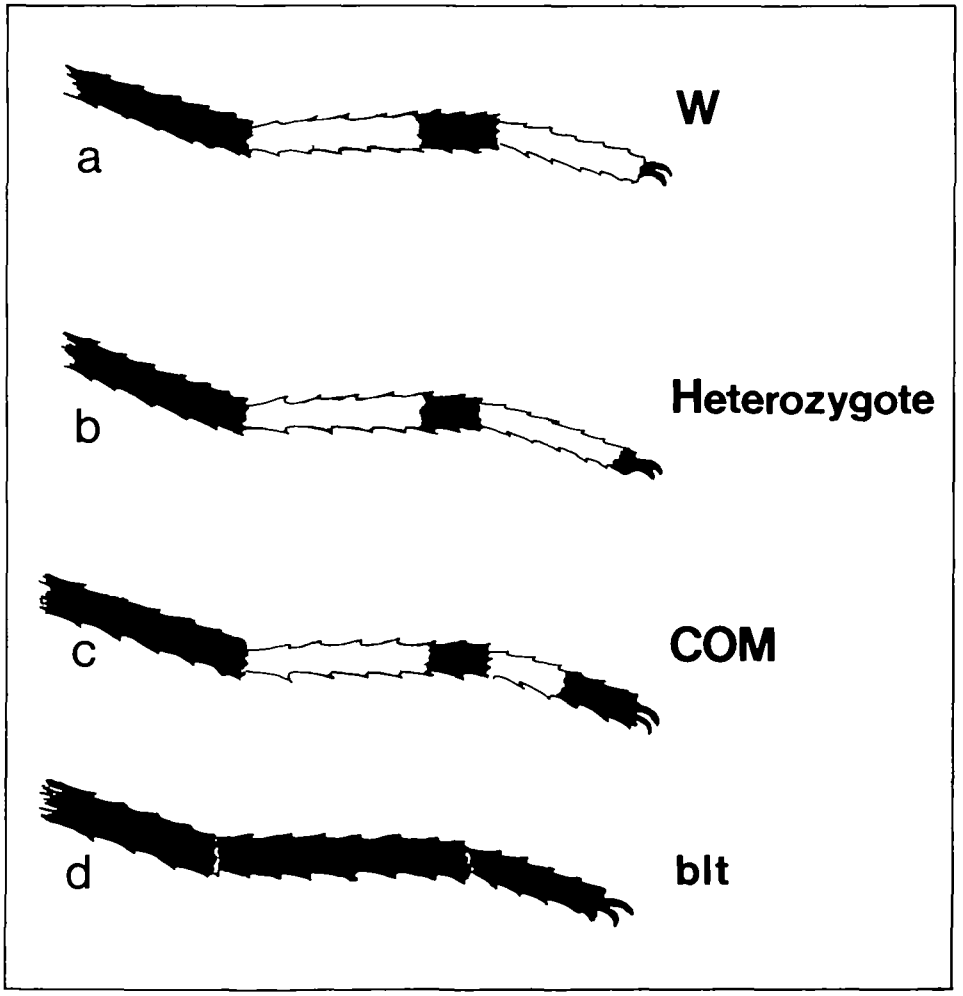

Figure 1. Diagram showing variation of black scaling on the hind tarsomere V of *A. aegypti*. (a) W = wild-type, all-white scaling on tarsomere V. (b) about six black scales at the tip, seen in the F_1 progeny when wild-type and COM were crossed. (c) COM = half-black tarsomere V. (d) *blt* = all-black tarsomeres.

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