

Table 1 Probability that M carries the band and X has a maternally derived band for each model in the reduced analysis

Model	Probability	Likelihood ratio
M unrelated to X (u)	qx	$L_u/L_m = 0.26^{25} = 2.4 \times 10^{-15}$
M aunt of X (a)	$q(1+x)/2$	$L_u/L_a = 0.413^{25} = 2.5 \times 10^{-10}$
M mother of X (m)	q	$L_a/L_m = 0.63^{25} = 9.6 \times 10^{-6}$

x , Band frequency, assumed to be the same for each band ($x=0.26$); q , allele frequency ($x=2q-q^2$).

Table 2 The number of bands (n_{ijk}) present in the sample with $i=(0)1$ if the band is (absent) present in M, $j=(0)1$ if the band is (absent) present in X, $k=0, 1, 2, 3$ is the number of sibs with the band, and computation of likelihood

No. of sibs	Band absent in X ($j=0$)				Band present in X ($j=1$)			
	0	1	2	3	0	1	2	3
Band absent in M ($i=0$)	-	10	4	0	0	7	9	2
Band present in M ($i=1$)	2	5	8	4	3	13	13	15

p_{ijk} , Probability corresponding to n_{ijk} . The log likelihood is computed conditional on the occurrence of the band in at least one individual, with summation excluding the 000 class: $\ln L = \sum_i \sum_j \sum_k n_{ijk} [\ln p_{ijk} - \ln p_{000}]$.

Table 3 Maximum likelihood estimates of allele frequency (q_{max}) for each model, together with corresponding log likelihoods ($\ln L$) and likelihood ratios in the full analysis, assuming B, S1, S2 and X all have the same father

Model	q_{max}	$\ln L$	Likelihood ratios
u	0.32	-256.78	$L_u/L_m = 2.1 \times 10^{-9}$
a	0.26	-247.05	$L_u/L_a = 5.9 \times 10^{-5}$
m	0.18	-236.78	$L_a/L_m = 3.5 \times 10^{-5}$

model. As more information about the frequencies and inheritance of the minisatellites becomes available, the models can be refined and the obvious power of the DNA-fingerprint technique utilized. Nevertheless, I do not believe that comparisons such as these give positive identification as suggested¹, only evidence that alternatives are highly unlikely.

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JEFFREYS ET AL. REPLY—We thank Professor Hill for his rigorous statistical analysis of the DNA fingerprint data of this immigration test-case. We are pleased that the new analysis gives ranges of values

for the likelihood ratios which are broadly similar to ours. We reiterate that even the new analysis includes a number of assumptions. The maximum likelihood values of q are based on only one family, and will be inaccurate. It is assumed that there is no variation in q between bands, and it is assumed that bands with the same mobility are always allelic and that bands with different mobilities never are.

We admit that we have not provided positive identification of the mother of X in a mathematical sense, since no probability calculation could do this. However, we believe that the probability of error in this case is so low that it would be legally appropriate to treat the test as providing positive identification.

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X;autosome translocations in females with Duchenne or Becker muscular dystrophy

RAY *et al.*¹ recently documented their efforts at isolating the gene for Duchenne muscular dystrophy by studying DNA cloned from the breakpoint of an X;21 translocation associated with Duchenne muscular dystrophy. However, the female patient² from whom this X-chromosomal material was derived³, clearly had Becker muscular dystrophy and not Duchenne dystrophy, as she was 20 years old and still ambulant. It thus seemed timely to critically review all the reported cases of

muscular dystrophy in females associated with an X;autosome translocation (Table 1) in order to try and designate them as Duchenne or Becker type.

Duchenne muscular dystrophy is an X-linked condition with onset in early childhood and a rapidly progressive course, so that the vast majority of affected boys lose the ability to walk by the age of 12 years⁴; Becker muscular dystrophy has an identical pattern of muscle involvement but follows a more benign course, and affected males will remain ambulant beyond 16 years of age and usually into adult life⁵. Inevitably there will also be occasional cases who may fall in the grey zone between the two and be a little milder than the usual Duchenne but not quite as mild as the usual Becker type and lose ability to walk between 13 and 16 years.

Careful attention has been paid to the diagnosis of male cases of either Duchenne or Becker dystrophy in the application of restriction fragment length polymorphism (RFLP) studies and detection of deletions. It would seem logical to pay the same attention to the initial diagnosis in the X;autosome translocation females from which the DNA sequences are cloned, rather than loosely referring to them all as Duchenne muscular dystrophy, as seems to have been the current practice.

Of the twelve cases documented to date, four⁶⁻⁹ conform closely to Duchenne dystrophy and a further four¹⁰⁻¹³ also seem to have a Duchenne severity but are still too young to be certain. One additional case¹⁴ is probably a Duchenne but was only published in abstract form with no mention of the age, which had to be surmised from the date of birth and timing of the abstract (approximately 14 years); she was said to be "unable to walk without assistance", which is also difficult to interpret. One case² conforms to Becker type and another¹⁵ also seems likely to be, but is too young (13 years) to be certain about ambulation beyond 16 years. The remaining case¹⁶ (published only in abstract form) has insufficient clinical data to draw any conclusions. Two of these cases are somewhat atypical, one¹⁰ having an associated dysmorphic syndrome and the other⁹ an associated Turner's syndrome.

Recent studies¹⁷⁻²⁰ have shown that the gene locus for Becker muscular dystrophy is very close to that for Duchenne and it seems likely that the two conditions are allelic. Studies with some of the probes (for example pERT 87.8)²¹ have shown deletions in approximately 9% (5/57) of cases of Duchenne muscular dystrophy but none with cases of Becker muscular dystrophy.

In the report of Ray *et al.*¹, only one Duchenne dystrophy patient out of 50 studied showed a deletion for their XJ-1 clone, and this was also the only case which showed a deletion for pERT 87. In