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ESTIMATION OF NONPATERNITY FOR X-LINKED TRAIT

To the Editor: Wiener [1] has noted a paradox in the figures given by Chakraborty et al. [2] for the probability of excluding paternity using X-linked systems. The answer given by Schull [3] that Wiener's arguments are all conditional upon the knowledge of the sex of the child is not correct. To set the record straight, we give the correct results here together with the appropriate extension for glucose-6-phosphate dehydrogenase (G6PD) when three alleles are recognized. In each case we give the probability that paternity can be excluded for a random mother-child pair and a random alleged father under the following assumptions: (1) half the children are of each sex, and (2) equilibrium genotypic frequencies under random mating have been reached in the population.

If an X-linked dominant gene has frequency p and the recessive gene has frequency $q = 1 - p$, then, as noted by Wiener, the probability of excluding paternity is $\frac{1}{2}q^2(1 - q^2)$; this is the appropriate probability for Xg, Xm, and for G6PD if electrophoretic typing is not available. If the female heterozygote is distinguishable from both homozygotes (as in a codominant system), the probability is $pq(1 - pq)$ whether the system is autosomal or X-linked. In the case of G6PD, three polymorphic alleles can be recognized in blacks on electrophoretic typing: B, A, and $A-$. The phenosets are $\{BB\}$, $\{BA, BA-\}$, $\{AA, AA-\}$, and $\{A-A-\}$; thus, unlike the ABO blood group, this is not a factor-union system [4]. For this X-linked system the probability of excluding paternity is $p(1 - p^3) - 2p^2(1 - p) + \frac{1}{2}qr^2(1 + r)$, where p, q, and r are the gene frequencies of B, A, and $A-$, respectively.

Table 1 shows the probabilities of excluding paternity that are found when these

TABLE 1

System	Alleles	Probability of Nonpaternity		
		Black	White	Japanese
Xg	Xg^a, Xg	.0807 (.1615)	.0483 (.0965)	.0672 (.1344)
Xm	Xm^a, Xm	.1220 (.1757)	.1250 (.1625)	...
G6PD	G, g	.0069 (.0932)	...	...
	$B, A, A-$	.1758	...	...

NOTE.—Figures in parentheses are those given by Chakraborty et al. [2].

formulas are used. We use the same gene frequencies as Chakraborty et al. [2] except for the triallelic G6PD case, which they did not consider; for that we use $p = .66$, $q = .22$, and $r = .12$. The probabilities given by Chakraborty et al. [2] for Xg can be obtained by ignoring the fact that half the children are males; that is, their result is conditional upon all the children being females. In the cases of Xm and G6PD, their probabilities can be obtained by (incorrectly) assuming the heterozygotes to be distinguishable as in codominant systems.

It should be noted that none of these particular results have any material effect on the general conclusions reported by Chakraborty et al. [2], and Schull's [3] second answer, namely that their figures were only intended to be guides, is perfectly acceptable.

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HALF CHROMATID MUTATIONS MAY EXPLAIN INCONTINENTIA PIGMENTI IN MALES

To the Editor: The paper of Gartler and Francke [1] on half chromatid mutations appears to offer an explanation for the occurrence of incontinentia pigmenti of Bloch Sulzberger type in males. The condition is probably due to an X-linked dominant gene lethal in hemizygous males. A total of 355 cases have been described in females, six in males. Those seen in males are in no way phenotypically different from those in females. The pattern of the skin affection is similar to the pattern exhibited by the heterozygous condition of some X-linked genes in mice, hamsters, cattle, and cats. The occurrence of a similar pattern in male children with incontinentia pigmenti has been puzzling. It might be attributed to early somatic mutation, but if so, smaller sectorial involvement rather than a generalized