identified homosexual brother pairs who volunteered to take part are represented) and thus does not purport to represent other homosexual men. The study must therefore be widened to other homosexual and heterosexual men to help define the extent of the association.

Finally, this study has no bearing on female homosexuality. Hamer and colleagues are also pursuing a line of investigation aimed at defining similar correlations in this group. The male study benefited from an observed tendency for female transmission of the homosexual trait. This observation immediately suggested X-chromosome transmission and provided a rationale for screening homosexual brothers for common inheritance of X-chromosome sequences. Unfortunately, this option is not available for the study of a female trait, so Hamer and colleagues will initiate a genome-wide search for an equivalent female association.

No doubt this move will also promote an inappropriate response from much of the media. Just as over-dramatizing the supposed effects of the male homosexuality linkage serves no useful purpose, neither do arguments on the extent of genetic influences on homosexuality. In a recent commentary published in the *New York Times*, Ruth Hubbard (professor emeritus of biology, Harvard University) attempts to discredit the associations between homosexuality and genetics by pointing out the limitations of such study. This is a surprising thing to do, as the limitations were made abundantly clear by the authors. Hubbard seems to be worried that society at large will hear of the recent advances being made in the genetic studies of polygenic and multifactorial traits and assume that each step of each investigation is a *fait accompli* with an immediate potential diagnostic implication. But this disregards the unambiguous and proven method of calculating the statistical likelihood that an association is real as opposed to one that has come about by chance and simply reflects a sporadic chance occurrence. Thus when Hamer and colleagues report that the observed association between certain X-chromosome markers and homosexual behaviour is quantified as carrying a lod score of 4.0, this simply means that there is 1 in 10,000 chance that the observed association is due to a statistical anomaly rather than to a real (and genetic) effect in the group studied.

This result is not, as some commentators have suggested, a breakthrough in our understanding of sexuality, nor does it promise to eradicate discrimination against homosexuals or claim to be the basis of a new set of civil rights laws. It is an interesting and stimulating result that suggests that further investigation into the potentially inherited component of sexuality may advance understanding. It is also straight genetics and should be valued as such. □

Cardiomyopathy revisited

In the July's *Nature Genetics* editorial, the complexity and heterogeneity of cardiomyopathic disorders were discussed in the context of the professional basketball player and celebrity, Reggie Lewis. A dispute had arisen between senior cardiologists about the reason for the collapse of Lewis during a crucial play-off last April. The original diagnosis of hypertrophic cardiomyopathy (which threatened to end the athlete's career) was later questioned by Gilbert Mudge (Brigham and Women's Hospital) who suggested that Lewis had an essentially normal and healthy heart but was subject to a neurocardiogenic syncope, a relatively benign condition. Tragically, on July 27, Lewis died during mild exercise and just three months after his much publicized collapse. Early post-mortem reports describe an enlarged and extensively scarred heart, thus fueling the dispute. Certainly, these findings suggest that the second diagnosis of a neurological condition was at best incomplete and have unfortunately resulted in fans of Lewis making a target of Mudge. The whole episode serves to emphasize the complexity of diagnosing heart conditions but perhaps more important reinforces what we also know but often ignore — medicine is an incomplete science that often requires further collaborative study rather than open and public bickering over what are necessarily difficult diagnoses. Rather than continuing the unsightly and needless dispute over who was right and who was wrong, the eminent cardiologists involved (and others) would serve the community better by acknowledging the difficulties and educating a public that could be forgiven for believing that if sufficient funds are available to hire any number of leading physicians, the correct diagnosis can always be made for any condition. It cannot. □

References

1. Hamer, D.H. *Science* **261**, 321–327 (1993)