

- satellites used to define haplotypes associated with 75 CFTR mutations from the UK on 437 CF chromosomes. *Hum Mutat* 8:229–235
- Kerem B, Rommens JM, Buchanan JA, Markiewicz D, Cox TK, Chakravarti A, Buchwald M, et al (1989) Identification of the cystic fibrosis gene: genetic analysis. *Science* 245: 1073–1080
- Macek M Jr, Macek M Sr, Krebsová A, Nash E, Hamosh A, Reis A, Varon-Mateeva R, et al (1997a) Possible association of the allele status of the CS.7/*Hha*I polymorphism 5' of the CFTR gene with postnatal female survival. *Hum Genet* 99: 565–572
- Macek M Jr, Mackova A, Hamosh A, Hilman BC, Selden RF40, Lucotte G, Friedman KJ, et al (1997b) Identification of common cystic fibrosis mutations in African-Americans with cystic fibrosis increases the detection rate to 75%. *Am J Hum Genet* 60:1122–1127
- Morral N, Bertranpetit J, Estivill X, Nunes V, Casals T, Giménez J, Reis A, et al (1994) The origin of the major cystic fibrosis mutation (delta F508) in European populations. *Nat Genet* 7:169–175
- Morral N, Dörk T, Llevadot R, Dziadek V, Mercier B, Ferec C, Costes B, et al (1996) Haplotype analysis of 94 cystic fibrosis mutations with seven polymorphic CFTR DNA markers. *Hum Mutat* 8:149–159
- Morral N, Estivill X (1992) Multiplex PCR amplification of three microsatellites within the CFTR gene. *Genomics* 13: 1362–1364
- Morral N, Nunes V, Casals T, Chillón M, Giménez J, Bertranpetit J, Estivill X (1993) Microsatellite haplotypes for cystic fibrosis: mutation frameworks and evolutionary tracers. *Hum Mol Genet* 2:1015–1022
- Ramsay M, Williamson R, Estivill X, Wainwright BJ, Ho M, Halford S, Kere J, et al (1993) Haplotype analysis to determine the position of a mutation among closely linked DNA markers. *Hum Mol Genet* 2:1007–1014
- Romeo G, Devoto M, Galiotta LJV (1989) Why is the cystic fibrosis gene so frequent? *Hum Genet* 84:1–5
- Russo MP, Romeo G, Devoto M, Barbujani G, Cabrini G, Giunta A, D'Alcamo E, et al (1995) Analysis of linkage disequilibrium between different cystic fibrosis mutations and three intragenic microsatellites in the Italian population. *Hum Mutat* 5:23–27
- Sereth H, Shoshani T, Bashan N, Kerem B (1993) Extended haplotype analysis of cystic fibrosis mutations and its implications for the selective advantage hypothesis. *Hum Genet* 92:289–295
- Traeger-Synodinos J, Kanavakis E, Tzetis M, Kattamis A, Kattamis C (1993) Characterization of nondeletion  $\alpha$ -thalassemia mutations in the Greek population. *Am J Hematol* 44:162–167
- Tzetis M, Kanavakis E, Antoniadis T, Adam G, Kattamis C (1997) Characterization of more than 85% of cystic fibrosis alleles in the Greek population, including five novel mutations. *Hum Genet* 99:121–125
- Welsh MJ, Tsui LC, Boat TF, Beaudet A (1995) Cystic fibrosis. In: Scriver CR, Beaudet AL, Sly WS, Valle D (eds) *The metabolic and molecular bases of inherited disease*, 7th ed. McGraw-Hill, New York, pp 3799–3876
- Williams C, Williamson R, Coutelle C, Loeffler F, Smith J, Ivinson A (1988) Same-day, first-trimester antenatal diagnosis for cystic fibrosis by gene amplification. *Lancet* 2: 102–103
- Zielenski J, Markiewicz D, Rininsland F, Rommens J, Tsui L-C (1991) A cluster of highly polymorphic dinucleotide repeats in intron 17b of the cystic fibrosis transmembrane conductance regulator (CFTR) gene. *Am J Hum Genet* 49: 1256–1262

Address for correspondence and reprints: Dr. Thilo Dörk, Institute of Human Genetics, Medical School Hannover, D-30625 Hannover, Germany. E-mail: doerk.thilo@mh-hannover.de

© 1998 by The American Society of Human Genetics. All rights reserved. 0002-9297/98/6302-0043\$02.00

*Am. J. Hum. Genet.* 63:662–663, 1998

### Media Portrayals of Genetics

*To the Editor:*

The article by Condit et al. (1998) demonstrates some of the limitations of quantitative analysis. The authors select from *Reader's Guide* articles listed under “heredity” in various time periods. Not surprisingly, such articles consistently attribute characteristics to genes. When the 50 articles selected from the eugenic period attribute human characteristics to heredity at almost the same rate as those selected from the 1990s, the authors conclude that nothing has changed. Predictably, they find that the “degree of determinism” (which they calculate to the fifth decimal) is consistent over 90 years of profound scientific and social change.

The paper is an example of the problem of trying to quantitate what is most compellingly understood in qualitative terms. Our study of the gene in popular culture (Nelkin and Lindee 1995), a target of Condit et al.'s paper, was not a quantitative study for the precise reason that the counting of such ambiguous and heterogeneous materials provides little insight into the public meaning of science. We focused on qualitative changes in a much broader literature, to suggest that the gene has acquired new powers as a guide to social policy. In the 1990s, the cultural meanings attached to the gene are shaping employment practices, educational policies, and decisions in the courts. The serious issues raised by the high-profile gene deserve more serious analysis.

DOROTHY NELKIN AND M. SUSAN LINDEE

*Department of Sociology*  
*New York University*  
*New York*

## References

- Condit CM, Ofulue N, Sheedy KM (1998) Determinism and mass-media portrayals of genetics. *Am J Hum Genet* 62: 979–984
- Nelkin D, Lindee SM (1995) *The DNA mystique: the gene as a cultural icon*. WH Freeman, New York

Address for correspondence and reprints: Dr. Dorothy Nelkin, Department of Sociology, New York University, 269 Mercer Street, Room 404, New York, NY 10003. E-mail: Dorothy.Nelkin@nyu.edu

© 1998 by The American Society of Human Genetics. All rights reserved. 0002-9297/98/6301-0044\$02.00

*Am. J. Hum. Genet.* 63:663, 1998

## Reply to Nelkin and Lindee

*To the Editor:*

The letter by Nelkin and Lindee, like their book (Nelkin and Lindee 1995), aptly demonstrates that qualitative methods can be at least as reductionistic as quantitative methods. Their reduction of our multiple historically sensitive index headings to the single heading of “heredity” is a misleading oversimplification. Furthermore, their claim that our article concluded that “nothing has changed” is false. Our study did show that contemporary public discourse about heredity, based as that discourse is in the accounts provided by molecular genetics and medical genetics, is not significantly more deterministic than were earlier accounts of human heredity. That, however, is not equivalent to a statement that there has been no change. In fact, our study demonstrates that contemporary presentations of genetics are more likely to assign different levels of genetic influences to different conditions. Contemporary accounts are also less likely to attribute genetic causation to simplistic behavioral characteristics. Moreover, our study demonstrates that in all periods the majority of popular representations do not attribute human characteristics solely to genetics but, rather, explicitly recognize that genes are only one factor in human outcomes.

Both quantitative and qualitative methods have useful contributions to make toward an understanding of the social implications of genetic science. To draw conclusions about the relative proportions of various types of discursive elements appearing in various venues requires that one make a quantitative assessment, no matter how informally. Formalizing one’s quantitative method by employing multiple coders and randomized article selection is useful for checking the researcher’s preconceptions by providing counterforces to the well-known tendencies toward selective perception of discourse. Cer-

tainly the quantitative findings of our study helped to modify our own preconceptions and to produce a more detailed, complex, and accurate qualitative account of the public discourse about biological heredity.

The qualitative portion of our study also indicates that reductionistic claims about increased determinism, of the sort made by Nelkin and Lindee (1995), fail to capture the complexities of the changes in public discussions about human heredity. Public accounts of the biological mechanisms of inheritance have shifted across the four eras in this century, from explanations centered on “germplasm” to “genes” to “DNA” to the “genome.” Accompanying these shifts have been changes in models of the relationship between genetic material and various environmental inputs. These models have posited increasingly fluid relationships between genetics and other forces across time, beginning with a model of the gene as boundary setter, moving to a model of DNA as a starting point, and, most recently, featuring models of genome and environment as coactive contributors to a normatively judged outcome. Space (not methodological choice) does not allow a full elaboration of these models and their complex relationships to other parts of the public discourse. Because of the enormous delay times in academic book publishing, we will be happy to make available, to anyone who requests it and pays postage and photocopying costs, the manuscript describing these features.

Nelkin and Lindee are correct that the new scientific information about genetics and the accompanying technological capabilities raise serious social questions, and their role in raising those questions has been valuable. However, these questions are best answered by approaches employing multiple methodologies and multiple perspectives.

CELESTE M. CONDIT

*Department of Speech Communication  
University of Georgia  
Athens*

## Reference

- Nelkin D, Lindee S (1995) *The DNA mystique: the gene as a cultural icon*. WH Freeman, New York

Address for correspondence and reprints: Dr. Dorothy Nelkin, Department of Sociology, New York University, 269 Mercer Street, Room 404, New York, NY 10003. E-mail: Dorothy.Nelkin@nyu.edu

© 1998 by The American Society of Human Genetics. All rights reserved. 0002-9297/98/6302-0045\$02.00