



David M. Umbach, Ph.D.

Education:

Ph.D. 1992 Biometry Cornell University
M.S. 1983 Ecology Penn State University
B.A. 1970 Physics & Philosophy Yale University

In the last 10 years, advances in molecular biology have profoundly changed how epidemiologists study environmental influences on disease. Today the notion that genes and exposures work together in causing disease is fundamental to research in this area. By measuring environmental exposures and genotyping subjects at particular loci, researchers can study how genetic polymorphisms modulate exposure effects, thereby uncovering clues to etiologic mechanisms and helping to identify susceptible subgroups in the general population.

My primary methodological interest is in developing new statistical tools for detecting and characterizing such gene-environment interactions through epidemiologic studies. Currently, I am investigating what assumptions are needed to yield valid tests and risk estimates with study designs that use affected and unaffected probands and their parents. My current work in this area assumes that the parent-offspring triads in the sample are independent. Often, however, data on affected probands may come from previously-identified extended pedigrees; multiple triads formed from each pedigree will not generally be independent. In the future, I will develop approaches for analyzing parent-offspring triads that arise from extended pedigrees.

I also actively collaborate with NIEHS researchers on a variety of studies. Topics include: lead and amyotrophic lateral sclerosis, birthweight distributions, hormonal effects of soy formula in infants, genetic susceptibility to bladder cancer, and the relationship of hepatitis B virus, aflatoxin exposure, and p53 mutations in hepatocellular carcinoma.

Relevant Publications:

Weinberg CR, Umbach DM. Choosing a retrospective design to assess joint genetic and environmental contributions to risk. *American Journal of Epidemiology* (in press).

Umbach DM, Weinberg CR. Using case-parent triads to study the joint effects of genotype and exposure. *American Journal of Human Genetics* 66:251-261, 2000.

Umbach DM, Weinberg CR. Designing and analyzing case-control studies to exploit independence of genotype and exposure. *Statistics in Medicine* 16:1731-1743, 1997.

Blazer DG, Umbach DM, Bostick RM, Taylor JA. Vitamin D receptor polymorphisms and prostate cancer. *Molecular Carcinogenesis* 27:18-23, 2000.

Taylor JA, Umbach DM, Stephens E, Castranio T, Paulson D, Robertson C, Mohler J, Bell DA. The role of N-acetylation polymorphisms in smoking-associated bladder cancer: evidence of a gene-gene-exposure 3-way interaction. *Cancer Research* 58: 3603-3610, 1998.

Longnecker MP, Kamel F, Umbach DM, Munsat TL, Shefner JM, Lansdell LW, Sandler DP. Dietary intake of calcium, magnesium, and antioxidants in relation to risk of amyotrophic lateral sclerosis. *Neuroepidemiology* (in press).

Kamel F, Umbach DM, Munsat TL, Shefner JM, Sandler DP. Association of cigarette smoking with amyotrophic lateral sclerosis. *Neuroepidemiology* 18:194-202, 1999

Umbach, D.M. Unit conversion as a source of misclassification in U.S. birthweight data. *American Journal of Public Health* 90:127-129, 2000.

Weinberg, C.R., and D.M. Umbach. Using pooled exposure assessment to improve efficiency in case-control studies. *Biometrics* 55:152-160, 1999.

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