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1983–1987	A.B., Physics Princeton University, Princeton, NJ
1987–1990	M.A., Ph.D. in Physics University of California, Berkeley, CA
1991	Visiting Scientist, NEC Research Institute, Princeton, NJ
1991–1992	Member of the School of Natural Science, Institute for Advanced Study, Princeton, NJ
1992–1993	NSF-NATO Postdoctoral Fellow, Oxford University, Oxford, England
1993–1998	Research Scientist, Whitehead Institute for Biomedical Research, Cambridge, MA
1998–present	Associate Member, Fred Hutchinson Cancer Research Center, Seattle, WA
1998–present	Affiliate Associate Professor, Department of Genetics, University of Washington, Seattle, WA
1999–present	Affiliate Associate Professor, Department of Molecular Biotechnology, University of Washington, Seattle, WA

Honors

1983–1987	National Merit Scholar
1987	Kusaka Memorial Prize in Physics, Princeton University
1987	Sigma Xi
1987	Phi Beta Kappa
1987	Highest Honors in Physics, Princeton University
1987–1990	Fannie and John Hertz Graduate Fellow
1991–1992	Fellow of Forbes College, Princeton University
1992–1993	NSF-NATO Postdoctoral Fellow
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Single-nucleotide polymorphisms in human genetics

Recently, attention has focused on the use of whole-genome linkage disequilibrium (LD) studies to map common disease genes. Such studies would employ a dense map of single-nucleotide polymorphisms (SNPs) to detect association between a marker and disease based on LD between the marker and a disease-risk gene variant. Construction of SNP maps is currently underway. One key question is the required marker density of such maps. I have used population simulations to estimate the extent of LD around common gene variants in the general human population as well as in isolated populations. Two main conclusions have emerged from these investigations. First, a useful level of LD is unlikely to extend beyond an average distance of approximately 3 kb in the general population, which implies that at least 500,000 SNPs will be required for whole-genome LD mapping studies in samples drawn from this population. Second, the extent of LD is similar in isolated populations unless either their history meets a set of specific criteria, such as a very narrow founding bottleneck and slow early growth, or the frequency of the variant is low (<5%). Another important question concerns the choice of strategy for SNP discovery. I will discuss the impact of different SNP discovery strategies on the properties of the resulting catalogue of SNPs.