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Statistical methods for causal inference in genomics.

How do genes regulate the expression of one another? Is it possible to learn the regulatory (or causal) relationships directly from vast amounts of observational genomic data? Mendelian Randomization (MR) makes tackling these questions possible. MR views genetic variants (SNPs, indels, and copy number variation) in a natural population as perturbations randomly performed by Nature, and provides a reasonable and potentially powerful assumption for studying causal relationships among genes. We develop machine learning and Bayesian methods in order to learn gene regulatory networks from genotype and expression data, which have directed edges that indicate causal relationships between pairs of genes. In methodology development, we also deal with several other challenges, such as controlling the false discovery rate, as well as reducing the impact of outliers. I will illustrate the performance of our methods on simulation data as well as The Cancer Genome Atlas (TCGA) data on breast cancer patients. (Received February 27, 2017)