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The Genetic Privacy of Presidential Candidates

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In the wake of the often bitter presidential election, with its emphasis on negative campaigning and intermittent controversies over the release of candidates' health information, it is not too soon to begin planning for the next presidential campaign. By then, advances in genomics will make it more likely that DNA will be collected and analyzed to assess genetic risk information that could be used for or, more likely, against presidential candidates.

Since 1972, when George McGovern was forced to replace his vice-presidential running mate, Thomas Eagleton, after it was revealed that he had been hospitalized for depression, the health status of presidential candidates has been seen by the press as fair game.¹ More recently, historians have discovered that some presidential candidates, including Franklin Roosevelt, Dwight Eisenhower, and John F. Kennedy, misled the public about their health status and that illness may have adversely affected their ability to perform their duties.

In this year's election, Senator John McCain, who had released extensive medical records in 1999, released an additional 1100 pages of records but gave reporters only a few hours to review them. President-Elect Barack Obama released an undated one-page "medical summary" to the press. News organizations pressed for more details, in the belief that the public has a right to know about a candidate's risk of future disease as an important indication of fitness for office. Although the presence of a disease or health condition is the most salient factor in the prediction of future health, medicine's ability to define levels of risk for individuals is expanding to include family history (a proxy for genetic predispositions to many diseases) and genetic markers.

Family history was used by the McCain campaign, which highlighted the energy and mental sharpness of McCain's 95-year-old mother, in an attempt to counter the notion that McCain's age might be associated with diminished vigor or cognitive function. Little was said about the death of his father and grandfather of heart attacks at 70 and 61 years of age, respectively. By the same token, the Obama campaign remained silent about the death of Obama's grandfather from prostate cancer, which indicates that Obama's own risk is higher than average.

During future campaigns, presidential candidates could release information about parts of their own genomes in order to highlight what might be considered a favorable ethnic background or, if they have already had a disease such as cancer, to highlight the absence of genes that confer a risk of recurrence. But in a climate of negative personal and political

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messages, it is more likely that persons or groups opposing a candidate will release such information, hoping to harm his or her chances for election or reelection.

Obtaining DNA, even from a president, would not be very difficult. Sufficient DNA for amplification and analysis can be obtained from loose hairs, coffee cups, discarded utensils, or even a handshake. A genome scan assessing hundreds of thousands or more single-nucleotide polymorphisms (SNPs) in such a sample could be performed with a commercially available microarray, or “SNP chip.” Some SNP variants are known to be associated with clinical diseases, and a tremendous number of new markers are being discovered and reported, although the contradictory evidence regarding some of these associations and the limited strength of many of them makes interpretation problematic. Would analysis of genetic markers have given us useful information about McCain or Obama, for example, that would have clarified the implications of their family histories of heart disease and prostate cancer?

Some genes have been found to have significant associations with coronary artery disease — most notably, a locus on the 9p21 region of chromosome 9. Three regions in the 8q21 region of chromosome 8 have been reproducibly linked to prostate cancer. Both 9p21 and 8q21 are non-coding regions of the genome, meaning that there are no actual genes there that code for protein products, but there may be nearby genes that are important, or there may be regulatory sequences in these regions that are important in the expression of other genes. These associations have been replicated in several populations and are probably valid on a population basis, but their value in providing risk information about a given person is severely limited. The relative risks associated with the implicated SNP variants at either of these loci would be less than 2, and there are legitimate questions about whether this degree of increased risk is meaningful on the individual level. But in the world of inflammatory accusations and smears that characterize presidential politics, it would be easy to engage in what might be called “genetic McCarthyism” by implying that an increased risk of disease is more substantial than it really is.

Some associations between common diseases and gene markers are reasonably well established. However, there is a constant stream of less well validated markers being linked to psychiatric conditions (still the most stigmatizing for presidential contenders) or even personality traits, which could be used to raise doubts about a candidate in the minds of an uninformed public. For example, the risk of bipolar disorder is reportedly increased by a gene encoding diacylglycerol kinase eta (DGKH) and decreased by a particular allele at the SNP rs420259, but both of these findings have had limited replication and involve modest effect sizes. Still, in the next presidential campaign, someone might publish a candidate’s genome and focus on a marker that has been linked to a psychiatric condition, regardless of how unproven the association is.

Though current genome scans can reveal 1 million SNPs, sequencing is required to reveal many known mutations and copy-number variants that may be associated with mostly rare diseases. Sequencing an entire human genome has thus far been an elaborate and costly undertaking, but technological advances are rapidly increasing the speed and decreasing the cost. To date, only two people, Craig Venter and James Watson, have had substantial portions of their genomes published, and their cases illustrate the latitude for interpretation and the potential for distortion. In his autobiography, Venter noted that he had one copy of the apolipoprotein E (APOE) ε4 allele conferring an increased risk of Alzheimer’s disease, a variant of the gene for complement factor H that has been linked to an increased risk of macular degeneration, and longer forms of the serotonin-transporter gene 5-HTTLPR that might make him more resilient against depression.² Watson asked that his APOE results be redacted, but his published genome indicates homozygosity for two devastating diseases,

type 1B Usher's syndrome and Cockayne's syndrome, neither of which the 80-year-old Watson has.³ As these examples show, sequence information may produce results that are emotionally charged, easily overinterpreted, or simply wrong by virtue of technical errors, low sequence coverage, or low-complexity sequencing.⁴

For the foreseeable future, the examination of thousands of genes in any genome is likely to result in large numbers of false positive findings, along with "incidental" findings of dubious clinical value.⁵ Thus, when sequence information about individual genomes becomes available, we will have to contend not only with the statistical issues of replication, effect size, and attributable risk but also with the specter of genetic information that is wrong or misleading.

Genetic information is easy to misinterpret and to misrepresent. Nonetheless, its scientific patina will encourage presidential campaigns to use it to reinforce existing prejudices. Therefore, we think future presidential candidates should resist calls to disclose their own genetic information. We recommend that they also pledge that their campaigns will not attempt to obtain or release genomic information about their opponents. Genetics experts, whether partisan or neutral, must be prepared to speak with the press to explain the nature of genomic information if and when it becomes public. Though it might be tempting to enact laws that would make it a federal crime to sequence a candidate's DNA without consent, we believe that restraint by the candidates, coupled with education of the public, will be a more reasonable approach as we enter a medical future based at least in part on personalized genomics.

Using genetic information to disparage opponents has no place in presidential campaigns. Nonetheless, the threat of genetic McCarthyism provides us with an opportunity to engage in a public dialogue about the limitations and complexities of using genomic information for decisions about life and health — including voting for our president.

References

1. Annas GJ. The health of the president and presidential candidates. *N Engl J Med* 1995;333:945–9. [PubMed: 7666895]
2. Venter, JC. *A life decoded: my genome, my life*. New York: Viking; 2007.
3. Wheeler DA, Srinivasan M, Egholm M, et al. The complete genome of an individual by massively parallel DNA sequencing. *Nature* 2008;452:872–6. [PubMed: 18421352]
4. Ng PC, Levy S, Huang JS, et al. Genetic variation in an individual human exome. *PLoS Genet* 2008;4(8):e1000160. [PubMed: 18704161]
5. Kohane IS, Masys DR, Altman RB. The incidentaloma: a threat to genomic medicine. *JAMA* 2006;296:212–5. [Erratum, *JAMA* 2006; 296:1466.]. [PubMed: 16835427]