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Characterizing the Genetic Influences on Risk Aversion

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Risk aversion has long been cited as an important factor in retirement decisions, investment behavior, and health. Some of the heterogeneity in individual risk tolerance is well understood, reflecting age gradients, wealth gradients, and similar effects, but much remains unexplained. This study explores genetic contributions to heterogeneity in risk aversion among older Americans. Using over 2 million genetic markers per individual from the U.S. Health and Retirement Study, I report results from a genome-wide association study (GWAS) on risk preferences using a sample of 10,455 adults. None of the single-nucleotide polymorphisms (SNPs) are found to be statistically significant determinants of risk preferences at levels stricter than 5×10^{-8}*. These results suggest that risk aversion is a complex trait that is highly polygenic. The analysis leads to upper bounds on the number of genetic effects that could exceed certain thresholds of significance and still remain undetected at the current sample size. The findings suggest that the known heritability in risk aversion is likely to be driven by large numbers of genetic variants, each with a small effect size.*

Introduction

Older Americans vary in their preparation for the financial burdens of retirement and old age. Some of this variation is due to differences in risk preferences, as well as differences in income, education, cognitive ability, and family background. Heterogeneity in risk aversion is still not fully understood despite thorough empirical research. Gender, age, and education levels are all predictors of risk aversion but often can explain only a small portion of the variation Americans display in risk aversion (Barksy et al. 1997). Risk preferences are known to be heritable (Cesarini et al. 2009b; Benjamin et al. 2012b). Substantial evidence from twin studies estimates that narrow-sense heritability ranges from 20 to 50 percent, implying that risk aversion is partly driven through variation in individual genetic structure.

The increasing incorporation of genetic data into social science surveys has provided new opportunities to correlate genetic variants to observed social and economic traits. I exploit this opportunity to examine the genetic nature of risk preferences, preferences that are fundamental to most individual-level demographic events, including financial and labor market decisions, health behaviors, migration, and marriage and fertility.

To date, most studies examining genetic influences on socio-behavioral traits such as educational attainment (Rietveld et al. 2013; Benjamin et al. 2012a), political participation (Hatemi et al. 2011), and self-employment (Van der Loos et al. 2013) have generated few positive findings, and efforts at replication have proven to be largely unsuccessful, with

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a few notable exceptions found in consortium efforts (Rietveld et al. 2013). A reasonable inference from these results is that the heritability of these traits arises from a large number of genetic variants—each with a relatively small effect—that current sample sizes are too underpowered to detect.

Still, an inquiry into risk preferences might hold more promise, as risk preferences may be more biologically proximate than previously studied traits. Risk preferences have been showed to be closely tied to hormones and neurotransmitters associated with the rewards system in the brain (Kuhnen and Chiao 2009; Campbell et al. 2010; Zhong et al. 2009). If risk preferences are indeed less polygenic, one could reasonably expect to identify significantly associated genetic variants.

Moreover, individual-level risk preference can vary substantially across different domains of risk and may depend on what elicitation technique is used to measure risk. There is no uniform battery of questions used to elicit risk preferences, as there is for IQ or the Big 5 personality traits. As such, risk preferences are not a good candidate to study through survey consortium efforts, since they are measured differently across different surveys.

Using the newly released genetic data from the U.S. Health and Retirement Study (HRS), this research runs a genome-wide association study of risk aversion using a large, nationally representative sample of over 10,000 respondents, the largest sample of this nature to date. In light of the evidence of substantial heritability of risk preference, this study seeks to answer the question: Is the heritable portion of risk aversion in individuals driven by a few genetic variants with large causal effects, or by a high number of variants, each providing small effects but contributing collectively to more pronounced individual variation in risk aversion?

To preview the findings, no single genetic variant was found to be significantly associated with measured risk preference at a significance threshold appropriate for these data. These results suggest that risk aversion is a complex trait that is highly polygenic. The analysis offered here is followed by detectability bound calculations that suggest that the known heritability in risk aversion is likely to be driven by a large number of genetic variants, each with a small effect size.

This article is organized as follows. Section 2 reviews the literature that provides compelling evidence of the heritability of risk preferences. Section 3 describes the data used and details the empirical strategy. Section 4 offers results, and Section 5 provides a concluding discussion.

Background

Substantial Heterogeneity in Risk Aversion Exists

Risk preferences vary markedly by gender (Schubert et al. 1999; Powell and Ansic 1997; Eckel and Grossman 2002), family background (Hartog et al. 2002), work characteristics (Van Praag and Cramer 2001), and educational attainment levels (Brunello 2002), as well as across different contexts of risk (Weber 2002; Soane and Chmiel 2005). These observed characteristics do not suffice to explain the differences in risk preferences, underscoring the importance of exploring genetic correlates to risk preferences.

Prior studies have established that differences in risk preferences are partially driven by genetic variation. A number of twin studies lend substantial support to the idea that risk preferences have a heritable component. A subsequent body of work has shown correlations between experimentally elicited risk preferences and variations in a few specific genes, most notably genes affecting dopamine and serotonin transporters (Kuhnen and

Chiao 2009; Dreber et al. 2011; Chew, Ebstein, and Zhong 2012). These findings provide evidence that risk preferences do have a genetic component, but they leave the source of most of the heritability unexplained.

The introduction of genome-wide data offers the potential for more precise empirical work in this area. Genome-wide data process large portions of the genome through the collection of hundreds of thousands or even millions of individual single-nucleotide polymorphisms (SNPs). An SNP is a DNA sequence variation occurring commonly within a population in which a single nucleotide in the genome has multiple forms, which can lead to phenotypic differences among individuals. (A comprehensive explanation of molecular biology written for social scientists can be found in Beauchamp et al. 2011.) Genome-wide studies allow researchers to test the association between an individually measured SNP in the genome and a specific outcome of interest.

Twin Studies Provide Estimates of Heritability of Risk Preferences

Evidence suggesting that risk aversion might be partially "hard-wired" arose from heritability estimates in twin studies comparing monozygotic and dizygotic twins. Four recent studies provide substantiation of the heritability of risk. One of the largest twin studies exploring risk preferences was conducted with a total of 1,875 twin pairs in Australia, comprising 867 pairs of identical twins and 1,008 pairs of nonidentical twins (Le et al. 2010). This study found that approximately 20 percent of the variation in risk is linked to genetic differences. Cesarini et al. (2009b) also found that genetic differences explain about 20 percent of individual-level variation in preferences for risk using a sample of 314 twins. Zhong et al. (2009) provide evidence that the heritability of economic risk attitude is as high as 57 percent, using a sample of 232 twin pairs in China. Zyphur et al. (2009), examining a subset of 200 male twin pairs from the Minnesota Twin Registry, attribute approximately 50 percent of the variation in risk preferences to genetic sources.

Similar results are found in twin studies using financial decision-making to measure risk preferences. A follow-up study by Cesarini et al. (2010) using the Swedish twin registry showed that 25 percent of variations in triskiness of portfolio allocation could be attributed to genetic variation. Using the same data, Barnea, Cronqvist, and Siegel (2010) found that a genetic factor explains about one-third of the variance in stock market participation and asset allocation and concluded that there are "innate differences in factors affecting stock market participation costs" (p. i).

Candidate Genes Point to Specific Areas on the Genome but Lack Precision of Strength of Effect

Candidate gene studies provide complementary evidence to the estimates of overall heritability provided by twin studies. They examine associations between survey measures and forms of a small number of genes chosen on the basis of prior knowledge about biological pathways. In work with candidate genes, handfuls rather than millions of statistical significance tests are at stake, so much weaker associations can be found to pass thresholds for statistical significance. For risk aversion, three of the most well-known candidate genetic variants in the biomedical literature have been found to generate positive results. These variants are not SNPs but another less common kind of genetic variation called "tandem repeats." These variations are not available in the HRS data, although it may be possible in future work to use proxies from the HRS data to investigate them.

First, there is a substantial literature on associations between the polymorphism *DRD4* (affecting dopamine receptors) and propensities for risk-taking. Examples include Kuhnen and Chiao (2009), Dreber et al. (2010), and Zhong et al. (2009), the latter of whom employ a lottery question similar to the one used in this study. Second, and similarly, studies have found the *5-HTTLPR* polymorphism (affecting serotonin receptors) to be associated with a number of dimensions of decision-making; carriers of the short allele are more likely to exhibit risk aversion. Additional examples in this area of research include Crisan et al. (2009), Homberg et al. (2008), Roiser et al. (2009), Stoltenberg and Vandever (2010), and van den Bos et al. (2009). Third, a polymorphism in the monoamine oxidase A gene (*MAOA*) has been studied in connection with financial risk-taking and responses to hypothetical lotteries (Frydman et al. 2011; Zhong et al. 2009). Various associations with risk aversion have likewise been reported in conjunction with nicotine receptors (Roe et al. 2009), oxycotin (Apicella et al. 2010), and testosterone (Zethraeus et al. 2009).

Data and Methods

This research ran a genome-wide association study to test the association of each individual SNP with the measure of risk aversion. Using this dataset, with more than a million SNPs and over 10,000 sample respondents, this study represents the largest GWAS on risk preferences to date.

Both phenotype and genotype data were drawn from the HRS, a longitudinal survey of a representative sample of Americans over the age of 50. The current sample is over 26,000 persons in 17,000 households, with a 2:1 oversample of African American and Hispanic populations. All spouses were also surveyed, regardless of age.

Genotype Data

The HRS recently released a set of genetic markers suitable for GWAS whereby it genotyped approximately 2.5 million SNPs from respondents. The total sample used for this GWAS is 10,455 individuals. The HRS has made available genotyped data for 12,595 respondents from the 2006 and 2008 survey waves, but both genotype and phenotype data were available for only 10,455 individuals.

Phenotype Data: Measure of Risk Aversion

This study used a measure of risk aversion first introduced in the 1992 survey wave by Barsky et al. (1997) that was subsequently included in five additional data waves. This measure categorizes risk aversion through responses to a series of hypothetical gambles on lifetime income, with the first question asking:

> Suppose that you are the only income earner in the family, and have a good job guaranteed to give you your current (family) income every year for life. You are given the opportunity to take a new and equally good job, with a 50-50 chance it will double your (family) income and a 50-50 chance that it will cut your (family) income by a third. Would you take the new job?

Depending on the initial response, follow-up questions were asked with lower or higher cuts to family income, allowing respondents to be categorized into one of four risk groups.

This question has been established as an accurate measure of risk preference. Since introduction, this measure of risk aversion has been used in a number of studies attempting to measure risk preferences (Smith et al. 2004; Schulhofer-Wohl 2007; Evans and Smith 2010) and has inspired similar measures in other surveys (Dohmen et al. 2005). A number of recent studies have attempted to validate Barksy et al.'s (1997) measure of risk preference and have concluded that hypothetical questions track closely, albeit imperfectly, with actual risk-taking behaviors (Dohmen et al. 2005; Guiso and Paiella 2004; Falk and Heckman 2009). This measure has been shown to be highly correlated with various measures of risky behavior, including smoking, failing to have insurance, and holding stocks rather than treasury bonds (Barsky et al. 1997).

Empirical Strategy

The main empirical strategy for this research was a genome-wide association study on the measure of risk aversion. Following usual practices (Sullivan and Purcell 2008), I applied four quality control measures to the sample of SNPs and respondents. First, individuals were dropped if the fraction of the SNPs in their array with missing data was greater than 0.05. Next, SNPs with a missing data frequency greater than 5 percent were deleted. Third, I eliminated SNPs for which the least common allele had an incidence smaller than 1 percent (called the "minor allele frequency"). Finally, I excluded SNPs that failed a test of Hardy–Weinberg equilibrium at the 10^{-4} level. Applying all filters left a total of 1,222,014 SNPs and 10,455 individuals for analysis.

I ran the following regressions using an ordinary least squares method:

$$\text{Risk Aversion} = \beta_0 + \beta_1 \cdot \text{SNP}_\text{S} + \text{PC} \cdot \beta_2 + \text{X} \cdot \beta_3 + \varepsilon \tag{1}$$

where Risk Aversion is a measure from 1to 4, with 4 being the most risk averse; SNP_S is the number of copies of the minor allele (0, 1, or 2) an individual has at SNP s; PC is a vector of the 10 top principal components of the genome of the sample to control for population stratification; and the vector X includes age measured in the same year as the last measure of risk aversion and gender. I ran a total of 1,222,014 regressions, one for each SNP, using the GenABEL library in R. I restricted my sample to respondents that have at least one response in any one of the six HRS waves in which this question was asked. In the case of multiple responses, the most recent response was used. Analysis was conducted to ensure that a Gaussian specification was appropriate for the outcome measure.

As is apparent from these specifications, the difficulty in interpreting the results of a GWAS comes from the repeated number of regressions that are run on the same sample of respondents. Because the sample of respondents is much smaller than the number of SNPs, many SNPs will inevitably turn out to be statistically significant at conventional levels simply because of sampling variation. As such, standard levels of significance for *p*-values are not appropriate for this technique. I followed standard practice and used a threshold of a *p*-value of less than 5×10^{-8} (McCarthy et al. 2008).

Results

Descriptive Analysis

Table 1 details the descriptive statistics of the respondent sample. The sample is nearly 60 percent female and, as is consistent with the sampling procedure, the majority of the

Table 1
Descriptive statistics of sample

	n	%
Gender		
Male	4,311	41
Female	6,144	59
Age		
0–49	503	5
50–54	1,941	19
55–59	3,258	31
60–64	3,483	33
65–69	707	7
70–79	536	5
80+	27	0
Risk aversion		
Least risk averse	1,189	11
Second-most risk averse	1,037	10
Third-most risk averse	1,482	14
Most risk averse	6,747	65
N	10,455	

Source: Author's tabulations of HRS respondent survey and genome-wide association data.
Note: These tabulations refer to the genotyped sample before filtering for quality controls.

sample is between the ages of 50 and 65. Because spouses are included, the youngest sample member is 25. The oldest person in the sample is 92. Approximately 60 percent of respondents are categorized into the fourth group of risk, which is the most risk averse. The other respondents are fairly evenly distributed across the remaining three categories, with just over 10 percent being the least risk averse.

The risk aversion question was asked in survey waves 1 and 4 through 8, with the number of respondents in each wave ranging from 748 in Wave 5 to 5,451 in Wave 1. As seen in Table 2, not surprisingly, risk aversion tends to increase with age, although not by a marked amount, since the sample consists primarily of persons over the age of 50 years. Also in line with the literature on risk aversion, there are slightly higher shares of females categorized as most risk averse. Finally, because I pooled responses across different waves, I ran sensitivity tests to ensure that there were no systematic differences in responses across waves.

Results of GWAS Analysis

In Table 3, I report results for the 20 SNPs that attain the highest statistical significance for the specification in Equation 1. The results provided in Table 3 are based on the specification that controls for age and gender, as well as for population stratification, through the inclusion of the top ten principal components. The first column gives the name of each SNP with the chromosome on which it is located. In the second column, I report the effect size of the SNP. In the third column, I report the *p*-value for each SNP. None of the approximately 1.2 million of the SNPs reaches the conventional significance threshold of 5×10^{-8}. In fact,

Table 2
Risk aversion by control factors (for test sample)

	Risk aversion measure			
	Least risk averse	Second-most risk averse	Third-most risk averse	Most risk averse
Sex				
Male	436 (15%)	345 (12%)	410 (14%)	1,790 (60%)
Female	384 (9%)	399 (9%)	633 (15%)	2,948 (68%)
Age category				
0–49	37 (11%)	40 (12%)	55 (16%)	213 (62%)
50–54	167 (12%)	157 (12%)	226 (17%)	800 (59%)
55–59	242 (10%)	219 (9%)	299 (13%)	1,547 (67%)
60–64	280 (11%)	251 (10%)	355 (14%)	1,567 (64%)
65–69	52 (11%)	45 (9%)	70 (14%)	325 (66%)
70–79	40 (11%)	31 (8%)	36 (9%)	272 (72%)
80+	2 (11%)	1 (5%)	2 (11%)	14 (74%)
Most recent wave with data				
1994	229 (12%)	208 (11%)	210 (11%)	1,259 (66%)
2000	65 (9%)	66 (10%)	84 (12%)	471 (69%)
2002	35 (12%)	19 (7%)	36 (13%)	193 (68%)
2004	148 (12%)	118 (10%)	184 (15%)	749 (62%)
2006	11 (18%)	7 (11%)	8 (13%)	36 (58%)
2008	332 (10%)	326 (10%)	521 (16%)	2,030 (63%)

Source: Author's tabulations of HRS respondent survey and genome-wide association data.

none of the SNPs reaches a significance of 10^{-7}, although 7 of the top 20 SNPs reach a significance level of 10^{-6}.

The fourth column reports the minor allele for each SNP. Columns 5 and 6 represent the chromosome and the position on the chromosome where the SNP is found. The collection of SNPs from the top-20 list are spread over nine chromosomes. Six of the SNPs are concentrated near each other on chromosome 5, and there is another concentration of SNPs on chromosome 12. Otherwise, the rest of the SNPs are scattered across a number of chromosomes, suggesting that risk aversion is not driven by a small number of SNPs with large effects, but rather that there may be low-level genetic activity across different parts of the genome. Given the evidence of heritability from previous work, it is likely that a larger sample might reveal SNPs that meet this level of significance. Further work on these bounds is described in the detectability bounds section.

Figure 1 shows the plot of the top-ranked SNPs analyzed against the $-\log_{10}$ of the *p*-value. This version of a "Manhattan plot," as it is often called, only displays the top of the "skyline"—the subset of SNPs with $-\log_{10}$ (*p*-value) greater than 4. In other words, this plot includes only the top 113 SNPs; most of the SNPs lie below this threshold and therefore would be at the bottom of this plot if the Y-axis were extended to zero. I've truncated the axis to put the top-ranked SNPs into context relative to significance. As is evident, none of the SNPs makes its way up to the significance threshold of 10^{-8}, but there are a large number of SNPs that "rise above," with *p*-values in the range of 10^{-6}. With this sample size, it is impossible to distinguish between SNPs with actual effects and statistical

Table 3
Top 20 SNPs from GWAS run

SNP	Effect size	*p*-value	Allele	Chromosome	Position
kgp4560299	0.14694087	3.15E-06	GA	5	435256
kgp1703572	0.14580118	3.87E-06	AG	5	435273
rs818185	0.07931359	4.02E-06	GA	2	10648857
kgp5696875	−0.079052	4.73E-06	AG	19	9102742
rs10099909	−0.13249798	5.01E-06	AG	8	11287371
rs186493	0.08415274	6.47E-06	AC	16	3289331
kgp7673403	−0.18416093	8.39E-06	AG	12	58918789
kgp4043337	0.09221066	1.06E-05	AG	5	478655
rs7657627	−0.08238064	1.30E-05	AG	4	16412051
rs7762279	−0.13126482	1.48E-05	AG	6	32755290
rs957792	0.13700457	1.54E-05	GA	5	429989
kgp9153906	0.07763928	1.54E-05	CA	19	7924957
kgp910500	0.07500762	1.70E-05	GA	12	61497958
kgp12522368	0.07538613	1.86E-05	AG	12	61537215
kgp6975417	0.11141721	1.94E-05	GA	5	496730
kgp5076136	−0.34611196	2.02E-05	AG	12	22951899
kgp9495611	−0.28826115	2.09E-05	AG	14	67446965
rs9471770	−0.10335409	2.11E-05	AC	6	42081642
rs2079134	0.08690109	2.23E-05	AG	4	106006036
kgp9614205	0.13442179	2.34E-05	AG	5	429031

Source: HRS respondent survey and genome-wide data.

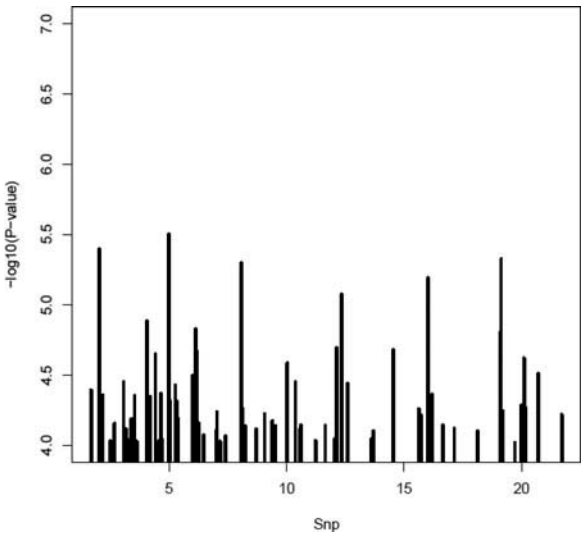

Figure 1. Manhattan plot for top-ranked SNPs associated with risk aversion.

noise. As sample size grows and our statistical tools become more powerful, the genetic components of risk aversion may reveal themselves.

Detection Bounds

The results from the GWAS are consistent with a picture in which large numbers of small genetic effects combine to account for the known heritability of risk aversion. There might be a few moderately large causal effects hiding within the random noise produced by sampling error for this sample of 10,455 respondents. However, given the polygenic nature of the trait, there will not be too many variants, nor will their effect sizes be too large. The following analysis allows some quantification of the terms *too many* and *too large*. I start by calculating the statistical power function for a GWAS test statistic against a family of alternative hypotheses.

Of course, there are a number of different ways of constructing families of alternative hypotheses. Here I adopt a simple approach. I develop a standard of what is meant by *large* by considering the *p*-statistics for SNPs that show up in the tail of the distribution with estimated *p*-values smaller than 10^{-4}; I'll call this group *B*. These *p*-values are the ones based on the chi-square test on one degree of freedom, appropriate to an additive genetic model, as already shown in Figure 1. The strength of effect for SNP_i is measured on a log scale by

$$G_i = -\log_{10}(p_i)$$

Under the null hypothesis, the true causal effects for all SNPs are zero, so all the variation seen is due to sampling error. Thus, I would expect one in 10,000 SNPs to appear in the batch *B*. Indeed they do. The expected number of SNPs to appear in this batch is 122; I observe 107 in the data.

Under the null hypothesis, G_i are independent exponential random variables. In GWAS data, SNPs are often correlated with each other and therefore are not independent, a situation referred to as *linkage disequilibrium*. However, since the quality control measures eliminate a great number of SNPs in linkage disequilibrium using a stringent threshold, I feel comfortable asserting the assumption of independence. If I were to use natural logarithms, the numbers would be exponential variables with unit means. Since I am using logarithms to the base 10, they are exponential random variables with a mean of $1/\log(10)$. The observed mean for SNPs in batch *B* is 4.329, very close to the expected mean of $4 + 1/\log(10)$, or 4.434. To determine the standard for *large*, I take effects whose true strengths measured on the G-value scale are as large as the observed mean for SNPs in *B*, namely, 4.329. For every choice of $k = \{5, 6 \ldots 15\}$, I consider an alternative hypothesis that *k* SNPs have true effects this large and that all the others have true effects of zero. All SNPs are subject to the sampling error found in my sample, making observed estimates vary around the corresponding true values. This construction produces a family of alternative hypotheses H_k indexed by *k*.

The customary GWAS test statistic for a test of the null hypothesis of no causal effects (all noise) is $\max(G_i)$. As I have discussed, the customary rejection region for the test is $R_1 = \max(G > 7\text{-}\log_{10}(5))$, based on the traditional *p*-value criteria set by the Wellcome Trust Case Control Consortium. With my data, I would reject the null hypothesis with any rejection region inside the region $R_2 = \max(G > 5.501)$, which is the maximum value in my data.

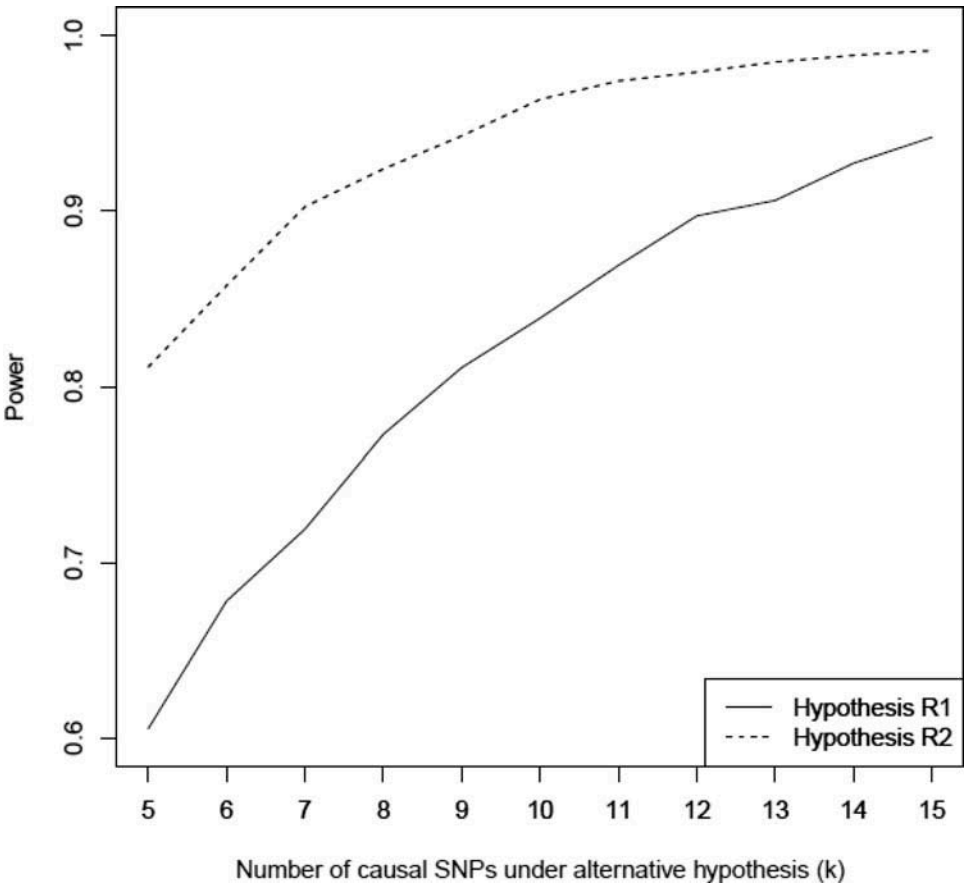

Figure 2. Power calculation against alternative hypotheses.

Figure 2 shows the statistical power functions for tests R_1 and R_2 against the family of alternative hypothesis H_k as a function of the posited possible number of causal alleles k. We see that for k bigger than 10, the customary test R_1 has power greater than 92.74 percent. For test R_2, the power is greater than 98.9 percent for any k bigger than 10, and greater than 90.26 percent for k bigger than 3.

We see that it is unlikely that we would be rejecting the null hypothesis if there were more than a dozen or so SNPs with true causal effects on risk aversion as strong as the apparent effects in the batch we have described.

Higher sample sizes would allow us to strengthen these bounds. But our power function calculations already point strongly to a highly polygenic character for the heritable component of risk aversion. Very recent advances in this statistical technique will allow continued work in detectability bounds for the increasingly long list of highly polygenic traits (Wu et al. forthcoming).

Discussion

As the results have shown, the analysis reveals that risk aversion is likely to be highly polygenetic in nature and not driven by a few genetic variants with large causal effects. The

results of this GWAS using a large, nationally representative survey show no associations of single SNPs that are significant at the conventional threshold of genome-wide significance required for the sample size of 10,455. The interpretation of these results is that the heritability of risk aversion must be driven by large numbers of genetic variants with causal effect sizes small enough that they cannot be detected with the current sample size.

I confirm these results through some analysis of detectability bounds to eliminate a family of alternative hypotheses in which a few large causal SNPs drive genetic variation in risk aversion. My findings suggest that much of the "missing heritability"—the gulf between the cumulative explanatory power of specific common variants identified to date and the overall heritability estimated through twin studies—reflects the fact that risk preferences have a complicated genetic nature that requires still-larger sample sizes to identify.

These findings add to a body of accumulating evidence from studies exploring a number of economic, political, and social preferences (Beauchamp et al. 2011; Benjamin et al. 2012b; Fowler and Dawes 2013) that suggest that the effects of *common* genetic variants explored through candidate gene studies on complex outcomes are small. The introduction of genome-wide data allows the inclusion of genetic variants that are less common and previously unexplored and, as a result, allows for a much more precise understanding of the nature of the preferences that underlie economic and social parameters. A study exploring a similar measure of risk aversion using a sample size of 2,900 (Benjamin et al. 2012b) showed no significant SNPs associated with risk aversion; my study has expanded this scope by exploring whether some of these genetic variants would come to light using a sample size more than three times larger. Continued investments in larger sample sizes might reveal these genetic variants. Recent efforts at combining a number of different datasets through data consortium efforts, leading to respondent sample sizes of 100,000 or more, have demonstrated limited success (Rietveld et al. 2013), but these consortium efforts are most appropriate for outcomes that we have a uniform way of measuring; risk preferences do not fall into this category.

The findings of this study do suggest that the construct of risk might be a broad phenotype, with several latent factors. A potential further direction of this researh could involve reproducing a GWAS analysis on these factors in order to possibly reveal genetic influences with a more precisely measured phenotype.

As large cohorts of older workers are moving into retirement, and as retirement savings are driven in growing shares by private savings, there is a growing imperative to understand the unexplained heterogeneity in individual risk aversion. The recent introduction of genetic material into social science surveys presents a unique opportunity to capture sources of variation that until recently have been nearly impossible to measure. Two individuals who are identical in terms of income, education, wealth, and age may still make very different portfolio investment choices; these results suggest that these individuals may have different genetic endowments that influence those choices.

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