The Role of Gene-Environment Interaction in the Formation of Risk Attitudes

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Abstract

Risk preferences are an important feature of every individual's decision-making process, which has been treated as an exogenous and fixed parameter of economic models for a long time. However, recent empirical economic literature suggests that risk aversion is in fact an endogenous variable that may change throughout life. Despite recent efforts to find factors that explain the formation of risk-preference, the empirical evidence is inconclusive and does not provide a clear picture of its architecture. I investigate risk-preference formation using a novel model class of gene-environment interactions and using a risk preference measure directly linked to the labour market. This allows me to study the relationship between genetic endowment, previous experiences of changes in the unemployment rate, and risk preferences related to labour market uncertainty. This is the first study to shed light on the complementary role of socio-economic factors and genetic endowment in the formation of risk-preference. The aim of the paper is to deepen our understanding of the risk-preference formation process in a way which was not possible in previous studies that focused only on the socio-economic dimension of the problem. The results show that only individuals with low genetic predispositions for risk tolerance are affected by the changes in their unemployment-rate histories, whereas individuals with high genetic predispositions for risk tolerance are not significantly affected. Hence, this paper presents evidence that adverse economic situations accentuate the inequality in risk preferences that originates from initial genetic endowment. This may ultimately lead to an increase in inequality in health, wealth, income and other outcomes related to risk preferences.

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Introduction

Attitudes towards risk (risk attitudes) fundamentally shape decisions. A risk aversion parameter is present in many economic models that aim to explain, for example, investment decisions (e.g. Brunnermeier and Nagel, 2008). Furthermore, recent empirical research has shown that measurements of fundamental risk aversion parameters are related to many important outcomes including migration (Jaeger et al., 2010), self-employment status (De Blasio et al., 2018), health outcomes (Dohmen et al., 2011), and many others.

Most current economic models assume that risk preferences are exogenous and fixed. However, the current empirical risk-aversion literature has presented evidence that changes in environments may influence preference for risk taking[3]. Specifically, the literature shows that, among other factors, changes in wealth, financial crises, and natural disasters lead to changes in risk attitudes (e.g. Page et al., 2014, Cameron and Shah, 2015, Cassar et al., 2017, De Blasio et al., 2018, Hanaoka et al., 2018). However, the empirical evidence is inconclusive and sometimes contradictory, which suggests that the relationship between environments and risk attitudes is more complex [4]. At the same time recent advances in behavioral genetics and genoeconomics document that individual heterogeneity in risk-taking behavior is influenced to some extent by genetic endowment (Cesarini et al., 2009, Benjamin et al., 2012). However, this strand of the literature does not take into account the economic dimension of the problem, or the possibility that the genetic and the economic sides may interact, which may skew their conclusions (Heckman, 2007, Mostafavi et al., 2020, Houmark et al., 2020).

To shine new light on the discussion and provide a possible explanation for the mixed evidence of the risk-aversion literature in empirical economics, this study brings the two existing strands of literature together. It does so by investigating how risk attitudes are formed by earlier life experiences and by genes, and by the interaction of the two factors. If the genetic and the economic (or environmental) sides do interact, then not taking them both into account may lead to a skewed and incomplete explanation and picture of the architecture of risk attitudes.

Hence, the aim of this study is to help to understand the mixed evidence from the empirical risk-aversion literature and help illuminate the overall risk-attitudes formation problem. Additionally, the study addresses to what extent genetic endowment influences risk attitudes. Although there are several gene-environment interaction studies that have investigated the genetic architecture of many behavioral outcomes (i.e. phenotypes), this is, to the best of my knowledge, the first study that investigates how environment moderates the relationship between genes and elicited risk tolerance.

[3]By environments I mean the broad area of surroundings the individual is exposed to. These may include social factors, natural factors, and others. Including environments into which individuals self-select.

[4]For a comprehensive discussion of the topic see Chuang and Schechter (2015).

I use data from the Health and Retirement Study (HRS), which includes genetic information about the respondents and income lottery questions that elicit individual risk attitudes. As a measure of risk attitudes I create a risk-tolerance measure, which is the inverse of risk aversion. The most attractive feature of the HRS risk tolerance measure is that the elicited risk tolerance is directly linked to the labor market by its construction. This is especially important for this paper because it links individual exposure to increase in the unemployment rate to risk tolerance measure that captures risk preferences that are related to labour market outcomes. The downside of previous literature, which investigate risk attitudes formation, is that they often use a general measure of risk aversion or risk tolerance. Although in economics risk aversion is often represented by one parameter, it is likely that individuals posses different risk preference in different aspects of their lives. For instance a CEO may exert an exceptional level of risk tolerance when it comes to firm investments into research and development but at the same time the same CEO may behave very risk averse when driving a car or during a hiking trip. It is of course possible that what drives the difference in the CEO behaviour in this example is just their beliefs about the distribution of the possible outcomes but a reasonable alternative explanation is that individuals have different risk tolerance parameter for different risky situations. This paper is among the first that link specific changes in the environment (unemployment rate) to a risk tolerance measure that is constructed based on risky situations that are related to the environment.

To measure the genetic endowment, I follow the literature and construct a single index measure called a polygenic score (PGS) (see e.g. Janssens et al., 2006, Belsky and Harden, 2019), which captures individual genetic predisposition for risk taking. As a measure of earlier life experiences I use individual life experiences on the labor market. To overcome the potential endogeneity arising from individual selection into experiences based on their risk tolerance, I exploit the variation across birth cohorts.

Specifically, I merge the HRS data with the national unemployment rate from the Bureau of Labor and Statistics and create a variable that captures the US nationwide unemployment rate growth that each individual faced from the year they were born until the year of the survey. Moreover, following the seminal work of Malmendier and Nagel (2011) I allow for each historical unemployment growth rate to have a different weight, which is captured by a single parameter. This approach allows investigation of which life experiences are the most formative. The other benefit of this approach is that it allows the model to control for age and time fixed effects while estimating the role of life experiences based on the birth cohort variation.

The effects of life experiences related to economic conditions on risk attitudes are subject to many studies. These life experiences can include financial crises, changes in wealth, GDP growth, and adverse development of stock markets. However, similarly to other domains the empirical evidence is mixed. For instance, in closely related papers,

Malmendier and Nagel (2011) and Malmendier et al. (2011) suggest that experiencing unfavorable development of the stock market and financial crisis lead to higher risk aversion.

Another closely related paper by Levin and Vidart (2020) finds no evidence for the experience of GDP growth on risk attitudes but does find evidence that the volatility of GDP growth has a positive effect on risk averse behavior. In contrast, a substantial part of the literature suggests that the main source of heterogeneity in risk attitudes is due to persistent differences between individuals and that risk attitudes do not change in response to changes in income, unemployment status, or wealth (e.g. Sahm, 2012, Brunnermeier and Nagel, 2008).

The goal of this study is to investigate whether individuals with different genetic predispositions differ in their responsiveness to adverse economic shocks. The individual heterogeneity in responsiveness may be one source of the inconclusive evidence, because if the effect of an environment is moderated by genotype, then the estimates of the treatment effects of the environment depend on the genetic composition of the sample. More importantly, the current empirical models are not able to identify the effects of adverse economic conditions for individuals who were endowed with lower genetic predispositions for risk taking. The model I investigate may further illuminate this heterogeneity, which is important in order to better understand how adverse economic conditions affect inequality in risky behavior.

The results document that genetic predisposition for risk tolerance positively affects the elicited risk tolerance in my sample, and this effect does not disappear even if I control for past experiences. In terms of magnitude, the estimate of the effect of genes is similar to those for females, veteran status, and education. I do not find any evidence that life experiences have a dire impact on elicited risk tolerance. However, I do find evidence that the effect of life experiences varies with genotype. Specifically, I find that individuals at and below the median of the distribution of the standardized polygenic score for risk tolerance (low PGS) are substantially affected by past experiences, while individuals above the median of the PGS distribution for risk tolerance (high PGS) are not responsive to past development of growth in the unemployment rate. The low PGS individuals are, on average, willing to sacrifice around 30 percentage points less of their income in order to play the risky option when they experience a one percentage point increase in unemployment growth than high PGS individuals.

Furthermore, I show that the results do not change substantively when I change the specification of the PGS group to quartiles. In this case the two bottom-quartile groups are significantly less risk tolerant than the highest PGS group (above the third quartile). Hence, my results show how genes and the environment jointly shape outcomes, implying that adverse life experiences can amplify genetic differences This may lead to an increase in the inequality in risk tolerance, which can ultimately be reflected in health, income and

many other outcomes related to risk tolerance. Thus, the results help us to understand how changes in environments may further alter the differences between individual risk tolerance that arise due to genetic endowment.

The rest of the paper is structured as follows. Section 1 describes the background of genetic data analysis and related challenges. Section 2 describes the data and the main variables of interest and presents robustness checks. Section 3 presents the model and our identification strategy. Section 4 presents our findings and discusses their implications. Finally, section 5 concludes.

1 Genetic Markers in Economic Research

For many decades, scientists have discussed the respective roles of genes and environments in the formation of human outcomes. This debate is often summarised by the nature vs nurture dichotomy. However, recent evidence shows that this debate is obsolete and imprecise. Instead, new models have been proposed that capture a more nuanced relationship between outcomes, genes and socio-economic variables. These models allow for genes and environments to interact in the formation of important human outcomes (phenotypes) (Turkheimer et al., 2003, Rutter, 2006, Heckman, 2007, Biroli, 2015, Houmark et al., 2020). Although the notion of gene-environment interaction has been discussed in the past, it was not then feasible to investigate their importance in empirical models due to the high cost of obtaining genetic data.

Recent decline in the price of genetic data collection and advances in genetics have lead to projects called genome-wide-asscociation studies (GWAS) (Okbay and Rietveld, 2015, Locke et al., 2015, Linnér et al., 2018, Lee et al., 2018). These are hypothesis-free studies that aim to find robust relationships between genetic markers called single-nucleotide polymorphisms (SNPs) and a phenotype (Schmitz and Conley, 2017a). This and the recent availability of genetic data in many socio-economic surveys has opened doors to investigate the questions that were previously not feasible to explore.

The new empirical studies have started to provide more insights into how genes and environments complement or substitute each other in the vast areas of outcomes, which include smoking behavior (e.g. Schmitz and Conley, 2016b), education (e.g. Conley et al., 2015, Schmitz and Conley, 2017b), obesity (e.g. Biroli, 2015, Schmitz and Conley, 2016a, Barcellos et al., 2018), or skills (Houmark et al., 2020). This paper is one of the first attempts to investigate how genes moderate the relationship between an environment and elicited risk tolerance.

One of the main challenges of incorporating genetic data into social-science research is their large dimensionality. Chabris et al. (2015) show that all behavioral traits are polygenic in their nature, which means that most outcomes are affected by many genetic markers with small effect sizes. Fortunately, external analyses called GWAS usually work

with samples of hundreds of thousands or millions of individuals, so they are suitably powerful to estimate robust relatioships between individual SNPs and outcomes. The results of GWAS are used by practitioners who work with survey data to construct an index called a polygenic score (PGS)(see e.g. Janssens et al., 2006, Belsky and Harden, 2019). Many survey data now provide rich information about the respondents but lack the number of observations necessary in order to find robust relationships between high-dimensional SNPs and outcomes.

The attractiveness of a polygenic score is that it is a single index that captures individual genetic predispositions for a given trait. Hence, using a polygenic score instead of all the SNPs in empirical models, substantially decreases the dimensionality of the models (from several thousand to only one variable). The basic ingredients of the PGS are the SNPs from the survey and SNP association coefficients from the GWAS. Humans possess a total of 23 pairs of chromosomes. This means that we have 2 versions of each SNP (one per chromosome). Consequently, SNPs can take on only three possible values: 0,1, or 2. The concrete realization of the SNP variable depends on how many risky alleles a person has at a given SNP[5]. The PGS index is then commonly used as a variable that captures genetic endowment or genetic predispositions for a given outcome and can be interacted with a measure of an environment in the gene-environment (GxE) models.

2 Data

This paper uses data from the Health and Retirement Study (HRS). The HRS is a nationally representative sample of the elderly US population over the age of 50. It was launched in 1992 and its participants have been surveyed every two years since then. Each wave of interviews contains information about approximately 20 000 individuals. Additionally, every six years a new cohort of participants is introduced to the survey. This paper uses information about individuals belonging to the following cohorts: "Children of the Depression" cohort (CODA), born 1924-1930, "HRS" cohort (HRS), born 1931-1941, "War Babies" cohort (WB), born 1942-1947, "Early Baby Boomers" cohort (EBB),born 1948-1953, and "Mid Baby Boomers" cohort (MBB), born 1954-1959. The purpose of the HRS survey is to monitor the changes in economic conditions, health, and cognition in the aging population in the US (Sonnega and Weir, 2014). This paper only studies individuals born between 1929 and 1959. I do not include individuals born before 1929 because the imputed data on the unemployment rate from the Bureau of Labor and Statistics ends in 1929.

I employ an easy-to-use version of the publicly available HRS data, which is available

[5]A risky allele represents a specific realisation of a SNP that increases the chance or amount of an outcome. For more information see **?**

at RAND HRS [6], together with information about individual genotype from the HRS and nationwide unemployment-rate data from the Bureau of Labor Statistics. The Bureau of Labor and Statistics provides information about the US unemployment rate only from 1947. In order to obtain precedent unemployment rates, I link the information from Lebergott (1948) about the measured unemployment rates to the updated information about the imputed unemployment rate from 1929-1946 [7].

2.1 Analytical Sample

The HRS collected DNA samples from respondents during interviews in 2006, 2008, and 2010. The DNA was extracted from saliva and genotyping was conducted using the Illumina Omni2.5 Beadchip. Before the analysis, standard quality checks were applied to the genotype data. These checks include the SNP Hardy-Weinberg Equilibrium (HWE) p-value: $p < 0.0001$, the SNP missing rate $\leq$1%, individual missing rates $\leq$ 10%, and minor allele frequency $\geq$ 1%.

Besides the quality control mentioned above, this paper also uses information about the ancestry group provided by the HRS to select only individuals from European ancestry. Hence, the final sample consists only of individuals of European descent. This is because, as mentioned for example in Tishkoff et al. (2009), individuals with African ancestry form a group of genetically diverse populations. This diversity is reflected in different linkage disequilibrium patterns and different minor allele frequencies across populations. Consequently, these differences alter the relationship between a phenotype and SNPs that was established by a GWAS performed on a population of European descent. If the GWAS results were applied to construct a genetic risk score, also known as a polygenic score, in a sample of individuals from different ancestry groups, the resulting index of genetic risk would be noisy and lose a great deal of its predictive power.

In 1998 respondents from the birth cohorts used in the analysis were asked risk attitudes related questions. In 2000, one in twelve individuals were randomly selected to answer the questions. In 2004 and 2006 only individuals younger than 65 years were asked the questions, and in 2002 only individuals from the EBB cohort were asked these questions (Bugliari et al., 2016). Hence the final sample consists of 5243 individuals observed across 5 interview waves, which amounts to a total of 9937 observations. Table 1 summarizes the main variables of interest.

[6]The RAND HRS Data (Version P, 2016) was developed by the RAND company with funding from the National Institute on Aging and the Social Security Administration, Santa Monica.

[7]The imputation study is available in the publications section of the Bureau of Labor and Statistics

Table 1: Summary Statistics

	All	
	mean	sd
Risk Attitudes		
Gamble Questions	0.165	0.201
Genes		
PGS(standardized)	0.000	1.000
Demographics		
Educ(years)	13.591	2.390
Age	57.512	5.333
Household Income (yearly. thousands of USD)	89.849	189.191
Sex	0.593	0.491
Behavior		
Smoking Now	0.185	0.388
Smoking Ever	0.577	0.494
Self-employed	0.192	0.394
Risky Assets (thousands of USD)	69.371	452.622
Observations		

2.2 Measurement of the variables of interest

A. Risk Aversion measure

Unlike the traditional approach in economics, where preferences are perceived as fixed starting points and not malleable throughout life, a modern approach relaxes the fixed nature of preferences and rather perceives them as malleable psychological traits (e.g. Loewenstein and Angner, 2003). For instance, Becker et al. (2012) show that economic preferences measures are likely to be complements of measures of the big five psychological traits in explaining real behavior. Although many economic studies treat preferences as malleable outcomes, it is still not clear which mechanisms lead to heterogeneity in individual risk aversion and to changes in it. Moreover, there is lack of consensus in the empirical literature about the effects of changes in various environments on risk aversion. One reason for the contradictory results is the imperfect measurement of risk aversion. Often the risk-aversion-survey measures also capture other factors such as expectations about the future, beliefs, status quo biases, and others. Moreover, most papers use a general measure of risk preferences and do not take into account that individuals can have different risk aversion levels for different life situations. Therefore, to properly study risk aversion (or risk tolerance), it is necessary to have a clean measure of risk-taking behavior that captures appropriate risk that is linked to the underlying model. In this study I use income-gamble responses, which allow me to estimate the effect of life experiences on a risk attitude measure net of expectations and other potential confounding factors. This is

because the income gamble questions explicitly state the probabilities of every scenario. Moreover, the income gamble lottery questions capture risk preferences related to the labour market, which is the relevant type of risk preference in my model.

The main outcome variable of interest is a measurement of risk tolerance[8], which is derived from the responses to hypothetical HRS income lottery questions. The individual's risk attitudes are then induced from the responses. From 1992 until 2006 the HRS questionnaire included hypothetical lottery questions that asked participants to choose between a job with a certain income and one that has a 50% chance of doubling the individual's income and a 50% chance of cutting it by a certain amount. The risk attitudes can be induced from the switching point, where the respondents switch from the risky option to the safe option. From 1992 until 1996 the safe option meant staying in their current hypothetical job or switching to a risky job. In 1998 both alternatives were presented as new job offers and the questions included two additional categories. For the 1992-1996 waves, the specific wording of the question is:

"Suppose that you are the only income earner in the family, and you 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?"

If the individual accepts the new job then a new question is presented with a higher potential income cut. If the individual declines the new job a new question is presented with a lower potential income cut. In the 1998-2006 the questions were changed to:

"Suppose that you are the only income earner in the family. Your doctor recommends that you move because of allergies, and you have to choose between two possible jobs. The first would guarantee your current total family income for life. The second is possibly better paying, but the income is also less certain. There is a 50-50 chance the second job would double your total lifetime income and a 50-50 chance that it would cut it by a third. Which job would you take – the first job or the second job?"

If the safe option is chosen, then the individual is asked to choose again between the two jobs but this time the income cut is reduced to 20%. If the individual still prefers the safe option, he or she is presented with a new question where the potential income cut is reduced even further to 10%. If the individual chooses the risky option in the first question, then the potential income cut in the next question is increased to a half. If the individual chooses the risky option again then the potential cut is increased further to 75%.

This paper uses only information from the 1998-2006 waves because the questions are

[8]Risk tolerance is the inverse of risk aversion.

framed as a choice between two new hypothetical jobs, while the older version was framed as a choice between new and current hypothetical jobs. Hence, the older version is likely to capture both risk aversion and status quo bias (Kimball et al., 2008). This paper aims to obtain the cleanest available measurement of risk aversion that is available and hence uses only the later version of the questions.

The main outcome variable is constructed from the responses presented in table 2. From table 2 it follows that the values of the risk-attitudes variable coincide with the point at which the individual switches from the safe to the risky option (e.g. individuals with value 0.33 accept the safe option if the income cut is higher than one third and when it is one third, they switch to the risky option). Hence, the higher the value of this variable, the higher the degree of risk tolerance.

Although the income-gamble questions provide a clean measure of risk preferences, the measure may still contain other factors including e.g. beliefs (Levin and Vidart, 2020). Hence, it may be the case that the income gable questions are still an imperfect measure of risk-related behavior. However, Dohmen et al. (2011) show that the income gamble questions that are often used in surveys to elicit risk aversion are correlated with real risk taking behavior across many different dimensions. Furthermore, the authors validated similar survey questions to mine with experiments and showed them to be valid measures of risk attitudes. To present more evidence on this matter, I perform simple tests to examine to what extent the HRS risk attitudes measure is related to real outcomes. Table 3 shows correlations between risky investment decisions and hypothetical lottery outcomes. Table 3 shows that the income gamble questions are significantly related to actual behavior.

Table 2: Risk Tolerance measures

Value of the Outcome	Income Cut Accepted	Income cut Rejected
0.75	75%	-
0.5	50%	75%
0.33	33%	50%
0.2	20%	33%
0.1	10%	20%
0	-	10%

B. Life Experiences

The aim of this paper is to investigate how life experience, together with genetic endowment, shape risk tolerance. The risk attitudes measure that we introduce above is induced from gamble lotteries regarding hypothetical job offers. Therefore, it is natural to choose a life-experience measure that is related to the labour market. One class of possible environmental variables would be individual job histories. However, these measures can suffer from endogeneity because, for example, more risk-averse individuals may select

Table 3: Correlation of Risk Tolerance and Various Real Life Outcomes

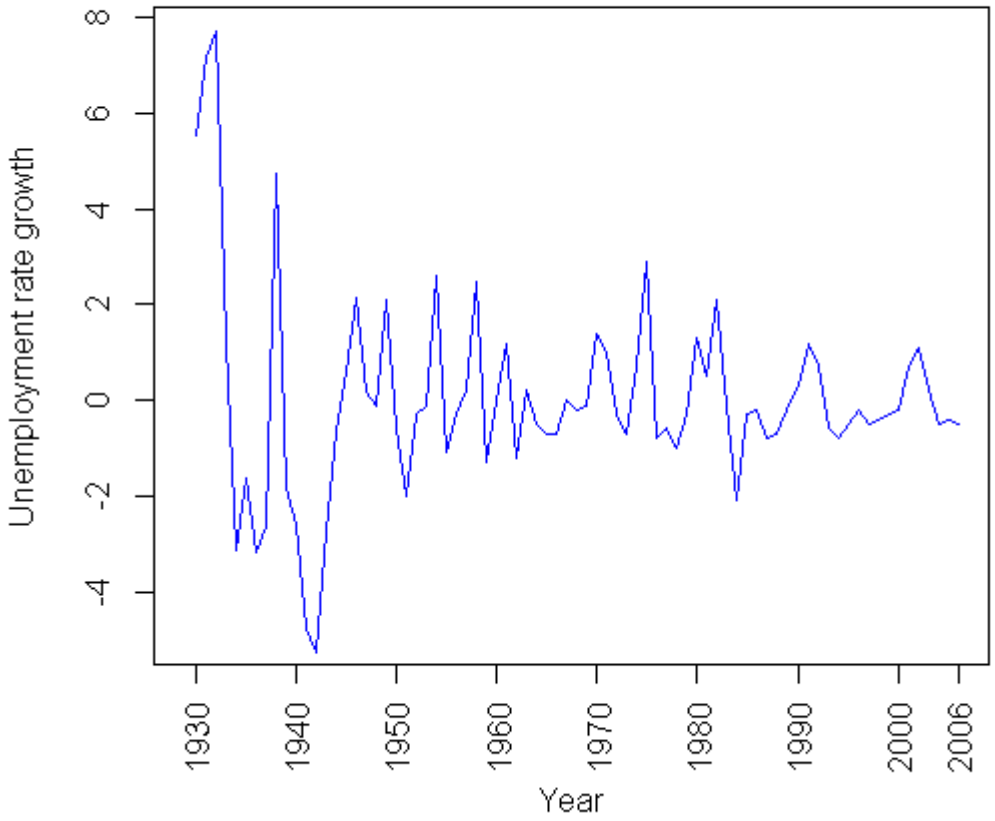
	Risk Tolerance
Intercept	0.042**
	(0.020)
Female	-0.053***
	(0.007)
Veteran	-0.022**
	(0.009)
Risky Assets	0.000
	(0.000)
Income	0.000
	(0.000)
Education Years	0.010***
	(0.001)
Ever Smoked	0.012*
	(0.006)
Smoke Now	0.003
	(0.008)
Self-employed	0.066***
	(0.008)
Nobs	9937
Adjusted R squared	0.048

Note: *Individual level clustered standard errors in parenthesis.* $***p < 0.01, **p < 0.05, *p < 0.1$.

themselves into jobs with lower risk of being laid off and consequently experience fewer spells of unemployment. To deal with the bias emerging from the selection of individuals into labor market environments based on their risk attitudes, I use an aggregate measure of changes in unemployment rate as a life experience measure. To construct the environmental measure, I merge the data on the unemployment rate from the Bureau of Labor and Statistics with the HRS Data. Including the imputed data, I have information about the US unemployment rate from 1929 to 2006, which was the year of the last wave of the survey that contained the income-gamble questions. Figure 1 shows the development of the unemployment-rate growth in the US in this time period. Note that the paper focuses on changes in the unemployment rate rather than on its levels. This is because evidence from behavioral economics suggests that individuals are more susceptible to changes in their environments than to levels (e.g. Kahneman, 1979, Malmendier and Nagel, 2011).

I link the aggregate unemployment measure to every individual, such that the life experience of each individual is captured as the history of aggregate changes in the unemployment rate that they experienced since birth. Thus, the main source of variation in the life-experience measure comes from the differences in birth years. The resemblance

Figure 1: Growth of the US Unemployment Rate



of this approach to cohort studies implies several potential challenges as the main issue with cohort studies is the linear dependence of year, age, and cohort effects. Furthermore, omitting one of the effects may confound the estimate of the remaining two. For example Dohmen et al. (2017) find that risk aversion increases with age, which is by definition correlated with birth year and the year of an interview. Similarly, the time of the interview may matter for the response. Some periods may, for example be less volatile than others, which could be projected into a higher degree of risk tolerance for everyone. Therefore, in order to separate the cohort effects from the time and age effects, it is necessary to control for age and time in the analysis. However, this is not feasible as the three variables are perfectly collinear (Heckman and Robb, 1985).

Another challenge linked to the historical unemployment rates is the dependence of the effect of life experiences on the stage of the life cycle the individual is currently in. For example, a significant increase in the unemployment rate may have a different effect for a forty-year old worker with a secured job than for a young adult who is entering the labor market. It follows that the experience measure should not be a simple average of growth

in past unemployment rates, but the specification should rather capture the possibility that the unemployment rate shocks may have different effects in different periods of life. One extreme specification would be to allow for each year of realized unemployment rate growth to have a different effect. This specification is difficult to implement because not only would it imply a high-dimensional empirical model, but also the number of parameters to estimate would differ for each individual.

To address the two challenges mentioned in the previous two paragraphs, I use the methodology developed by Malmendier and Nagel (2011). The idea is to build a single-parameter function of age, life experiences, and how long ago the experience was realized. This function is essentially a weighted average of past experiences where the weight depends on the age of the respondent, how long ago the experience was realized, and a parameter λ that allows for different weighting schemes:

$$A_{i,t}(\lambda) = \sum_{k=1}^{age_{i,t-1}} w_{i,t}(\lambda, k) * \Delta Unemployment_{i,t-k}$$

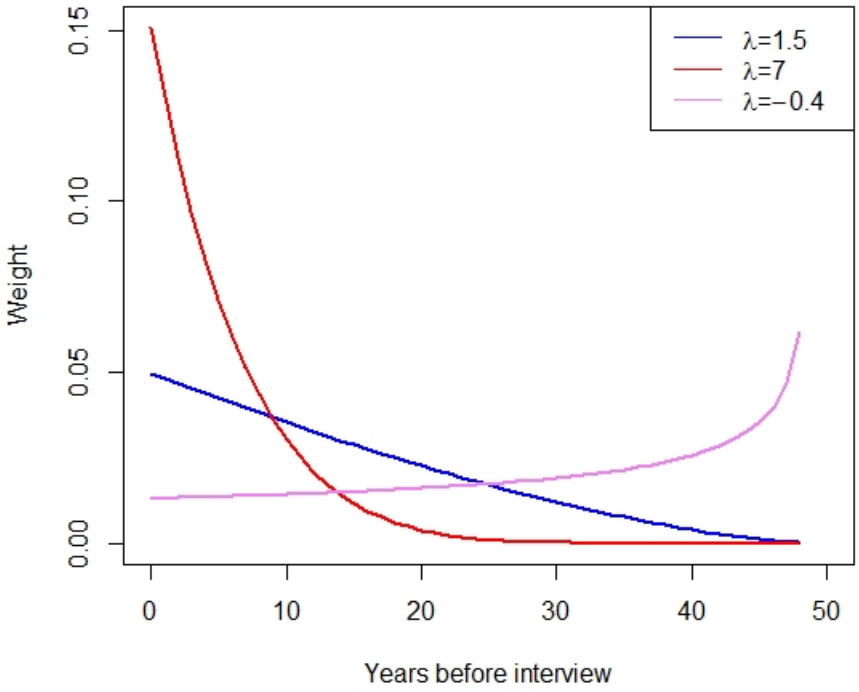
$$Where \quad w_{i,t} = \left[\frac{(age_{i,t} - k)^{\lambda}}{\sum_{k}^{age_{i,t-1}} (age_{i,t} - k)^{\lambda}} \right] \tag{1}$$

Equation (1) can flexibly capture the differential importance of historical growth in unemployment rates without imposing too complex model that may be difficult to estimate. The weight assigned to each past experience is captured by a parameter λ that can be estimated from the data and serves as a discount rate of past experiences. If $\lambda < 0$, then the weight is monotonically increasing and convex. Such a profile would imply that early life experiences matter more than more-recent ones. If $\lambda = 0$, then each past experience would obtain equal weight and equation (1) would become a simple average of past experiences. If $\lambda > 0$, then the weight is monotonically decreasing and concave, which means that the more-recent experiences matter more than the past ones. Finally, if $\lambda = 1$, then the weight is linearly decreasing. To better illustrate the logic behind the parameter λ, in figure 2 I show the weights as a function of time lags for different lambdas for a representative fifty-year-old individual.

Finally, because the expression in equation (1) is a function of the unemployment rate and a nonlinear function of age, it is possible to include controls for age and time in the main specification, as I show in section 3.

C. Polygenic Score As described in section 1 most of the outcomes (phenotypes) of interest to social scientists are in their nature polygenic. Hence, it is not generally feasible to estimate the effect of the individual genes in survey data because of the lack of power in these data sets. Instead, I adopt the standard approach from the behavioral genetic literature that studies the effects of gene-environment interactions and construct a single index measure that captures individual genetic risk, also known as a polygenic score.

Figure 2: Weights with different values for λ



To construct the polygenic score, I use the summary statistic from the recent GWAS conducted by Karlsson Linnér et al. (2019), together with information about genes from the HRS. I then construct the polygenic score as follows

$$PGS_i = \sum_j^J \beta_j SNP_{i,j} \tag{2}$$

where i stands for individual and j stands for a SNP. The β coefficients are taken from the GWAS summary statistic.

The discovery sample of the GWAS did not include the HRS data and was performed on individuals of European descent. I do not use any trimming methods for the score construction. Thus, the polygenic score includes all the SNPs that are in the HRS and overlap with the SNPs from the GWAS. Moreover, in the analysis, I work with a standardized polygenic score. Table 4 presents evidence on the predictive power of the HRS polygenic score on both income-gamble questions and for some real-life outcomes. Each row of table 4 shows a coefficient on the polygenic score from a regression of an outcome on the polygenic score and the first ten principal components of the genetic-relatedness matrix, which controls for population stratification (Price et al., 2006).

Table 4: PGS on Risk Tolerance and Real Life Outcomes

Outcome:	PGS(standardized) Coefficient:
Lottery	0,008**
	(0.003)
Self Employed	0.018**
	(0.008)
Education (years)	0.05
	(0.04)
Ever Smoked	0.03***
	(0.009)
Smoke Now	0.013**
	(0.006)
Nobs	9937

Note: *Individual level clustered standard errors in parenthesis.* $***p < 0.01, **p < 0.05, *p < 0.1$.

3 Empirical Model

In the analysis I explore the effects of life experiences, and polygenic score, and their interaction, on the responses to income gamble questions. To model the life experiences, I adopt the method developed by Malmendier and Nagel (2011) (described in more detail in section 2.2). This section describes the heterogeneity in individual perception of the past within the framework of the established model described above. There are many potential reasons why individuals may remember their past differently, for example, they may idealize their past (Schutz, 1962). Alternatively, the heterogeneity in how individuals react to their experiences may come from their genetic endowment. This paper investigates whether individuals who have a higher genetic predisposition for risk tolerance are resilient when it comes to adverse developments in the labor market, while individuals who are genetically predisposed to be less risk tolerant are more careful and more responsive to their past experiences. To investigate the heterogeneity in responsiveness to life experiences by genotype, I extend Malmendier and Nagel's (2011) current model by introducing a measure of genetic predisposition for risk tolerance and its interaction with the past-experiences measure. The general form of the model is described by equation (3).

$$\text{Risk}_{i,t} = \alpha_1 + \gamma_1 A_{i,t}(\lambda) + \sum_g^G \delta_{1,g} \mathbb{1}_{PGS_g}(PGS_i) + \sum_g^G \theta_{1,g} A_{i,t}(\lambda) \times \mathbb{1}_{PGS_g}(PGS_i) + X_{i,t}\beta_1 + \psi_{1,a} + \mu_{1,t} + \epsilon_{i,t} \tag{3}$$

where $A_{i,t}(\lambda)$ is the measure of life experiences defined by equation (1). Because in the case of this paper $A_{i,t}$ captures the individual unemployment rate history, in what follows I address this measure as "Unemployment" or "Unemployment history". $\mathbb{1}_{PGS_g}(PGS_i)$

stands for the polygenic score dummy variable, which is equal to 1 if individual i belongs to a specific part of the distribution of polygenic score PGS_g and 0 otherwise. Finally, X stands for other covariates, including the first 10 principal components of the genetic-relatedness matrix, to control for population stratification. $\psi_{1,a}$ and $\psi_{2,a}$ stand for age fixed effects and $\mu_{1,t}$ and $\mu_{2,t}$ stand for time fixed effects. Hence, the model allows me to study the effects of the cohort differences in unemployment-rate growth net of time and age effects. Finally, $Risk_{i,t}$ stands for the responses to the income-gamble questions. I estimate the model using nonlinear least squares. To capture the nonlinear relationship between the interaction term and the risk-attitudes outcome, I assign individuals into G genetic groups based on their polygenic score and estimate the main effect and the effect of the interaction with unemployment rate histories for every polygenic score group.

$$\mathbb{1}_{PGS_g}(PGS_i) = \begin{cases} 1, & \text{if } PGS_i \in PGS_g \\ 0, & \text{if } PGS_i \notin PGS_g \end{cases}$$

The step-function approach is meant to approximate the true conditional mean:

$$E[Y|PGS, A(\lambda)]$$

I choose to use the step function of the PGS because it fits the relationship better than a simple linear function of the PGS. Specifically, as I show in more detail in section 4, the relationship between the interaction of the PGS and unemployment rate history and risk attitudes is rather flat for high values of the PGS distribution and spikes upwards for lower values of the PGS distribution. To capture different possible functional forms, I consider 3 versions of model (3). First, I split the PGS by median into two PGS groups. Second, I split the PGS by tercile into 3 groups. Finally, I split the PGS by quartiles into 4 groups.

3.1 Identification and Basic Concepts

As stated above, the aim of the empirical model is to shed more light on how genetic endowment and life experiences shape human risk attitudes. To better understand the model a useful mental exercise is to link it to an ideal experiment. The simplest experiment I have in mind is to compare four hypothetical scenarios for individual risky behavior such that in all the scenarios individuals differ only in terms of their experience and genetic predisposition for risk tolerance. In the first scenario individuals would have low genetic predispositions for risk tolerance and experience adverse situations on labor markets[9]. In the second scenario the individuals would experience favorable situations

[9]Adverse situations on the labor market may be embodied by several factors including a higher amount of the individual's unemployment spells, and a bad match between an employer and an employee that may lead to income loss or unemployment. In general I think of an adverse situation as a series of

on labor-markets but have low genetic predispositions for risk tolerance. In the third scenario the individuals would experience adverse labor market development and have high genetic risk for risk tolerance. Finally, in the fourth scenario the individuals would have high genetic predispositions for risk tolerance and experience favorable situations on the labor market. It is useful to think of the model as an approximation to the ideal experiment because it simplifies the analysis of the potential shortcomings of the model and its strength.

Model 3 allows for the heterogeneous effect of past experiences by genotype. Hence, it provides more insight into discrepancies between the results from previous empirical literature. The core idea behind gene-environment interaction is that some traits are mainly affected if both suitable environments and suitable genes occur at the same time (see e.g. Rutter, 2006). It follows that some environments may affect outcomes only for people who are genetically predisposed to be susceptible to changes in those outcomes. Thus, one possible reason for the mixed evidence in the empirical literature that investigates the causes of risk aversion may be the heterogeneous treatment effect by genotype. So far the main goal of the empirical risk-aversion literature was to find an average effect on the treated (ATT) which is an average of the effects over all genetic groups:

$$ATT = E_{G|T}[E[Y_1 - Y_0|T = 1, G]|T = 1]$$

However, if the fixed nature of risk aversion is relaxed, it is possible to perceive it as a skill that can be harnessed. Therefore, as has been shown, for example, in Heckman (2007), Houmark et al. (2020), the environment side and the genetic side may dynamically complement each other. If true then omitting either of the two dimensions from the main specification may lead to a skewed and incomplete picture of the risk preference formation. The ATT estimates from the contemporaneous empirical literature may, to some extent, depend on the genetic composition of the sample if, for instance, only individuals with a certain genetic predisposition for risk tolerance are susceptible to the change in the environment, or if the effects have an opposite sign for individuals with different genetic predispositions. At the same time, the behavioral genetics literature, which at best considers the environmental channel and the genetic channel to contribute independently to the risk-aversion preference, misses the point of possible dynamic complementarity of the two dimensions. The proposed empirical model (3) aims to illuminate the complementary relationship between initial genetic predispositions, environment, and risk attitudes. In the rest of this section I discuss potential problems and constraints of the empirical model, which allows me to better define the realm of insights the model can provide.

A potential problem that may arise when estimating individual life experiences from

adverse shocks that can lead to either income losses and or unemployment

the survey data is reverse causality. In this setting, individuals may select themselves into environments based on their degree of risk tolerance and consequently differ in their life experiences. The empirical model overcomes this issue by focusing on the unemployment rate at a national level. Although it is possible that individuals may affect their environments I assume that no individual from the sample has sufficient power to affect the aggregate unemployment rate. The downside of this approach is that the model is only sensitive to nationwide historical changes in unemployment rates that affected everyone in the given cohort.

The nature of an empirical model that focuses on differences between cohorts implies that the effect of life experiences may include other factors that differ between cohorts. For instance Levin and Vidart (2020) argue that differences in smoking behavior across cohorts may lead to differences in death rates and therefore also to differences in potential risk tolerances between treated and control groups [10]. I include in the model other explanatory variables that may capture additional differences between cohorts. However, it is possible that some unobserved cohort differences are still captured by my measure of earlier life experiences.

Finally, given that this paper is a gene-environment interaction study, it also embodies the threats that arise from using genetic data as described in section 1. The main concern is the spurious correlation between genes and a phenotype due to population stratification, which is essentially a special case of omitted variable bias. To address the potential confounders that arise due to population stratification, I adopt the standard approach of the gene-environment interaction studies and include the first ten principal components in the regressions.

4 Results

In this section I document that genetic predispositions and past labor-market experiences contribute together to the formation of risk tolerance. Furthermore, I show that not taking into account the possibility of the complementary role of the two dimensions may lead to incorrect inference about the architecture of risk tolerance. To support this claim, I first consider a scenario that does not allow for the gene-environment interaction, thus reflecting the nature vs nurture dichotomy. Such a model allows life experiences and genetic predispositions to contribute independently to the risk tolerance measure. The results are shown in table 5. The results suggest that the polygenic score influences the responses to income-gamble questions. Specifically, a one standard deviation increase in the polygenic score leads to a 3.5 percentage points increase in the share of income the individual is willing to sacrifice in order to play the risky option in the lottery.

[10] I discuss the problem of differences in survival probabilities between birth cohorts in more detail in section 5

The coefficient of the past-life-experience measure is not statistically significant. Thus, the results based on a simple model that does not take into account gene-environment interactions suggest that past labor-market experiences play little role in forming risk tolerance compared to the genetic factor. The result that earlier life experiences do not contribute to risk attitudes, be it risk tolerance or risk aversion, is not uncommon in the empirical literature (Chuang and Schechter, 2015). At the same time the results in table 5 are in accordance with the traditional notion that risk preferences are predetermined and do not change over the lifetime. However, as I document in the rest of this section, this conclusion is imprecise as it does not allow for the more dynamic relationship between predetermined genetic predispositions and life experiences.

Table 5: Role of life experiences and the PGS

Outcome: Risk Tolerance	
Constant	-0.093
	(0.084)
Unemployment	-0.119
	(0.540)
λ	24.67
	(0.997)
PGS(standardized)	0.035
	(0.00)
Female	-0.053
	(0.000)
Veteran	-0.016
	(0.000)
Income	2.894×10^{-8}
	(0.000)
Education(YRS)	0.008
	(0.001)
MSE	0.039
Age FE	Yes
Year FE	Yes
Principal Components	10
Observations	9937

Note: p-values in parenthesis. To calculate the p-values I used clustered bootstrapping as described in Cameron and Miller (2015).

Following the discussion on the complementary role between genetic endowment and environments (or choices), I next estimate the gene-environment model 3, which captures the idea that genes and life experiences may complement or substitute each other in the

formation of risk attitudes. Table 6 shows the results for 2 PGS groups[11]. Individuals below the median polygenic score are labeled as a group with low genetic predisposition for risk tolerance and individuals with a score higher than the median genetic score are labeled as a group with high predispositions for risk tolerance.

Table 6: Life experiences shape risk attitudes for individuals with low genetic predispositions for risk tolerance

Gene-Environment Model	
PGS below median	-0.032
	(0.001)
PGS below median × Unemployment	-0.372
	(0.043)
λ	1.749
	(0.292)
Unemployment	1.027
	(0.412)
MSE	0.039
Age FE	Yes
Time FE	Yes
Principal Components	10
Observations	9937

Note: p-values in parenthesis. To calculate the p-values I used clustered bootstrapping as described in Cameron and Miller (2015).

The main effect of the low polygenic score group is negative and significant, which is consistent with the results from table 5. The results show that individuals with low-polygenic-score predispositions to risk tolerance are willing to sacrifice, by 3 percentage points, less of their income in order to play the lottery than the high polygenic score individuals. The coefficient of life experiences, which is captured by past unemployment rate changes, and the lambda coefficient are not statistically significant. However, the coefficient on the interaction term is both statistically significant and large in magnitude.

Taken together, the results provide a more detailed picture of the formation of risk attitudes than models that take into account only either the genetic or socio-economic part of the problem and do not allow for these two strands to interact. The results from the gene-environment interaction model document that individuals with lower genetic predispositions to risk tolerance who experienced unemployment growth are willing to risk less than their counterparts who also experienced high unemployment growth but belong to the group with high genetic predispositions for risk tolerance. The difference in responsiveness to an unfavorable past between the two genetic groups is a 37 percentage

[11]In section 5 I discuss additional model specifications and show that the main results hold across these.

points share of income they are willing to sacrifice in order to play the lottery.

The results imply that individuals with a low polygenic score for risk tolerance are willing to sacrifice less of their income in order to play a lottery when they experience an increase in the overall unemployment rate than the high polygenic score group. At the same time, individuals with a higher polygenic score are almost non-responsive to unfavorable past experiences. While low-polygenic-score individuals are, on average, willing to sacrifice less of their income, by 37 percentage points, when they experience a 1 percentage point increase in the unemployment rate, high polygenic score individuals seem to be affected less by the past experiences[12].

The last parameter of interest is λ, which captures the relative importance of more-recent experiences relative to more distant ones. Table 6 presents suggestive evidence that individuals discount past histories relatively more than more recent ones. However, the estimate is rather noisy, which is reflected in its large standard error.

It follows from the discussion above that the effect of past experiences on risk attitudes is present only for individuals who are strongly genetically predisposed to be risk averse. Individuals who have average or even high genetic predispositions for risky behavior have a "thick skin", so for them past experiences play little role when they decide if they should take a risk or not. An important implication of these results is that negative life experiences may increase inequality between individuals who were born with different genetic endowment. The inequality may arise because risk attitudes are related to many real-life outcomes. The results also help to clarify the inconsistency of previous studies that try to estimate the effects of changes in environments on risk attitudes. By allowing for the interaction between genetics and the environment, the model uncovers an important feature of the role of past life experiences on risk attitudes that would not be possible under a simpler version of the model that does not include both dimensions and their interaction.

5 Robustness Checks

This section discusses potential threats to identification of the coefficients of interest and provides tests of these concerns. Moreover, this section tests different model specifications to test for robustness of the results.

To address the robustness of the results, I examine additional possible PGS step-functions. For that purpose, I next consider two additional versions of model (3). First, I split the polygenic score distribution into terciles. The low genetic predisposition risk

[12]The difference in the responsiveness by the PGS group can be seen by adding the estimates of the past unemployment-rate effect and the interaction effect together. However, this simple arithmetic does not account for the confidence intervals. Therefore, I conclude that high polygenic score individuals are, on average, less responsive to the unfavorable past experiences.

group corresponds in this case to the observations below the first tercile; the average polygenic score group corresponds to observations between the first and the second tercile and the high polygenic score group corresponds to observations above the second tercile. Formally:

$$PGS_{Low} = \{PGS_i : PGS_i \leq Q(1/3)\}$$
$$PGS_{Average} = \{PGS_i : Q(1/3) < PGS_i < Q(2/3)\}$$
$$PGS_{High} = \{PGS_i : Q(2/3) \geq PGS_i\}$$

where $Q(p)$ is the corresponding quantile corresponding to probability p from $F[PGS]$. The finer structure helps to identify the nature of the relationship between the variables of interest. It also allows to further investigation of the relationship between polygenic score and risk tolerance [13].

The results for the PGS step function by terciles are presented in the first column of table 7. The results indicate that the low polygenic score group is less risk tolerant than the high polygenic score group. The average polygenic score group is more similar to the high polygenic score group in terms of risk tolerance. Table 7 demonstrates that there is neither statistical nor substantial difference between the average and high polygenic score groups. Thus, the results confirm the previous finding that the effect of polygenic score on risk tolerance is driven mainly by the low polygenic-score individuals. The interaction term between both the average and low polygenic score groups are both statistically insignificant due to low precision of the estimates. However, the point estimates of the two interaction terms suggest that the negative effect of past unemployment rate history is greater for the lowest PGS group, which is in accordance with the results based on the main specification presented in section 4. Individuals from the low PGS group are willing to sacrifice, by 25.6 percentage points on average, less of their income in order to play the lottery when they experience a percentage point increase in unemployment history.

Next, I consider the quartile based PGS step function. Formally:

$$PGS_{Low} = \{PGS_i : PGS_i \leq Q(1/4)\}$$
$$PGS_{LowAverage} = \{PGS_i : Q(1/4) < PGS_i < Q(1/2)\}$$
$$PGS_{HighAverage} = \{PGS_i : Q(1/2) < PGS_i < Q(3/4)\}$$
$$PGS_{High} = \{PGS_i : Q(3/4) \geq PGS_i\}$$

[13] To uncover the relationship precisely, we would need to have a dummy variable for each value of the polygenic score, but the score is continuous so this specification is not feasible.

The results for the model with PGS by quartiles are presented in column 2 of table 7. The pattern of the effects of the PGS score groups is again in accordance with previous findings. Specifically, individuals from the lowest PGS group (below the first quartile of PGS distribution) are, on average, willing to sacrifice 4 percentage points less of their income to play the lottery than the highest PGS group (above the third quartile of the PGS distribution). Individuals from the low-PGS group (between the first and second quartile of the PGS distribution) are, on average, willing to sacrifice 3 percentage points less of their income to play the lottery than the highest PGS group. The average PGS group (between second and third quartiles of the PGS distribution) is very similar to the highest PGS group in terms of risk tolerance. The coefficient for the average group presents evidence that the difference in the income the individuals are willing to sacrifice in order to play the lottery, compared to the highest PGS group, is -0.8 percentage points. The coefficient is statistically insignificant and substantially small compared to estimates of the lower PGS groups.

Table 7: Model Specification check: Different versions of the PGS step function

	(1)	(2)
Gene-Environment Model	PGS by tercile	PGS by quartiles
PGS below 1^{st} tercile	-0.032	
	(0.004)	
PGS between 1^{st} and 2^{nd} tercile	-0.006	
	(0.583)	
PGS below 1^{st} tercile × Unemployment	-0.256	
	(0.240)	
PGS between 1^{st} and 2^{nd} tercile × Unemployment	0.066	
	(0.812)	
PGS below 1^{st} quartile		-0.041
		(0.001)
PGS between 1^{st} and 2^{nd} quartile		-0.033
		(0.005)
PGS bewteen 2^{nd} and 3^{rd} quartile		-0.008
		(0.536)
PGS below 1^{st} quartile × Unemployment		-0.477
		(0.072)
PGS between1^{st} and 2^{nd} quartile × Unemployment		-0.436
		(0.080)
PGS between 2^{nd} and 3^{rd} quartile × Unemployment		-0.155
		(0.614)
λ	1.818	1.700
	(0.327)	(0.249)
Unemployment	0.836	1.116
	(0.531)	(0.382)
MSE	0.039	0.39
Age FE	Yes	Yes
Time FE		Yes
Yes		
Principal Components	10	10
Observations	9937	9937

Note: p-values in parenthesis. To calculate the p-values I used clustered bootstrapping as described in Cameron and Miller (2015).

The interaction effects present further evidence on the heterogeneous response to the unfavorable unemployment shocks by genotype. The effect of past unemployment histories is largest for the lowest PGS group (-0.47). The estimate suggests that individuals from the lowest PGS group (below the first quartile of the PGS distribution) are willing to sacrifice 47 percentage points less of their income to play the lottery, than the high PGS group when they have experienced a 1 percentage point increase in unemployment rate history. The interaction of the low-PGS group (between first and second quartile of the PGS distribution) suggests that individuals from this group are willing to sacrifice 43 percentage points less of their income on average to play the lottery, than the highest PGS group when they experienced a 1 percentage point increase in unemployment rate history. Both estimates are of substantial magnitude and similar to the interaction coefficient estimate from the main specification in table 6 (PGS groups by median). The interaction-effect estimates from the two low-PGS quartile groups are statistically significant on the ten percent significance level. This is mainly due to the low number of observations in both groups compared to the main specification where these two groups are merged into 1 (below median).

Nevertheless, together with the interaction effect estimate from column one, the results suggest that the interaction between the risk aversion PGS and unemployment rate history is negative and quite substantial in magnitude. Finally, the estimate of the interaction effect of the high average group (between the second and third quartiles of the PGS distribution) suggests that there is no significant difference between this group and the highest PGS group (above the third quartile of the PGS distribution). The discussion of different model specifications implies that individuals who are below the median of the PGS distribution are the most responsive to shocks in their unemployment histories, while those above the median are less so. Hence, the main specification includes the PGS step function by median.

Next I discuss a potential threat to identification that comes from the nature of the HRS data. Because the survey focuses on population aged 50+ and, as mentioned in section 2, this paper includes individuals born between 1929 and 1959, it is possible that some respondents may not have survived until the data collection phase. Domingue et al. (2016) show that the genotyped HRS sample differs in observables from the non genotyped sample. This suggests that the genotyped HRS sample suffers from a mortality related sample-selection problem. Moreover, Domingue et al. (2016) show that averages of various polygenic scores differ by birth cohort which indicates a genotype-based sample selection.

The mortality-based selection that may arise due to non-random deaths of HRS respondents across cohorts, may lead to a survival bias of the estimated GxE coefficient. The main concern is that more-risk-tolerant individuals may be more likely to die from their risky behavior. Consequently, the resulting bias of the GxE coefficient would be

positive. The other possible scenario is that the less-risk-tolerant individuals may be more likely to die prematurely[14]. In this case the bias of the GxE coefficient would be negative. To mitigate the survival bias, it is more appropriate to use variation across birth cohorts that are close to each other in terms of birth year. Table 8 presents a simple test of differential survival rates of individuals with different risk attitudes.

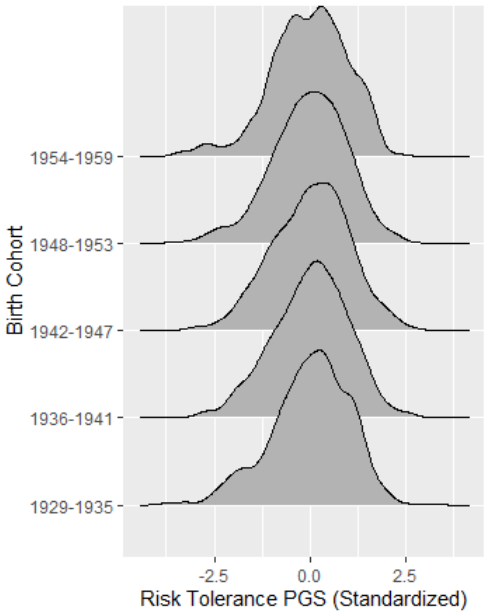
First, I address the concern that genetic predispositions for risk tolerance may lead to higher risk tolerance, which may lead to higher death rates. This claim would be reflected in different polygenic score distributions among the cohorts. Table 8 does not support this claim as the means and standard deviations of polygenic scores are rather stable across birth cohorts. Additionally, figure 3 presents a simple comparison between PGS distributions across cohorts and further supports the claim that the PGS distribution is rather stable across cohorts. Second, table 8 suggests that the risk tolerance, measured by the income-gamble questions, is also stable across cohorts.

Table 8: Cohort Comparison

	All		1929-1935		1936-1941		1942-1947		1948-1953		1954-1959	
	mean	sd	mean	sd	mean	sd	mean	sd	mean	sd	mean	sd
Risk Attitudes												
Gamble Questions	0.165	0.201	0.146	0.201	0.170	0.208	0.167	0.205	0.165	0.194	0.153	0.184
Genes												
PGS(standardized)	0.000	1.000	-0.003	1.030	0.016	0.990	0.019	1.009	-0.028	0.992	-0.017	0.991
Demographics												
Educ(years)	13.591	2.390	12.874	2.504	13.214	2.480	13.580	2.423	13.967	2.230	13.662	2.226
Age	57.512	5.333	67.199	1.923	62.486	1.931	57.986	3.673	53.905	2.548	48.401	2.822
Household Income (yearly. thousands of USD)	89.849	189.191	56.352	63.534	79.982	192.318	85.135	94.735	103.808	273.237	109.574	162.435
Sex	0.593	0.491	0.637	0.481	0.529	0.499	0.586	0.493	0.580	0.494	0.843	0.364
Behavior												
Smoking Now	0.185	0.388	0.121	0.326	0.158	0.365	0.192	0.394	0.193	0.395	0.239	0.427
Smoking Ever	0.577	0.494	0.610	0.488	0.613	0.487	0.603	0.489	0.524	0.500	0.541	0.499
Self-employed	0.192	0.394	0.275	0.447	0.237	0.426	0.195	0.397	0.172	0.378	0.143	0.350
Risky Assets (thousands of USD)	69.371	452.622	106.783	367.301	65.064	216.952	81.038	636.184	56.262	298.878	46.846	407.299
Observations												

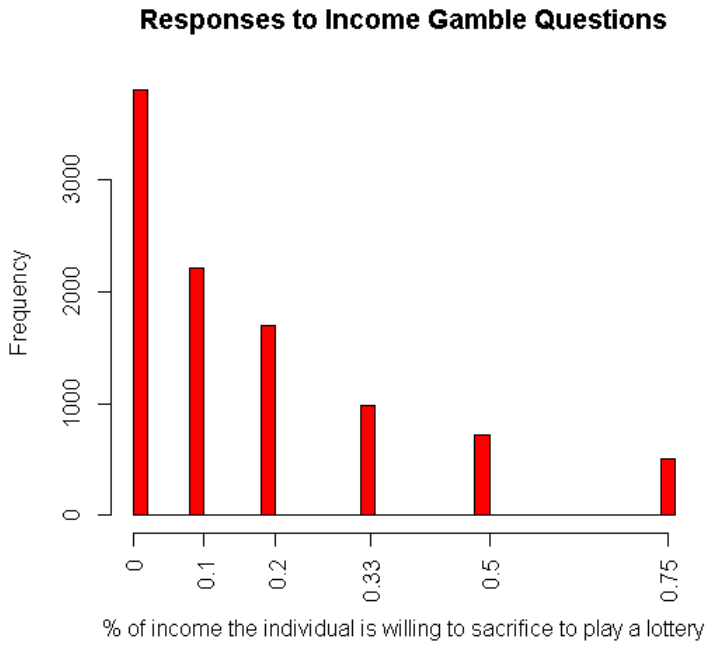
[14]For example they may achieve less wealth due to their low risk tolerance

Figure 3: Risk Tolerance PGS Distribution by Cohorts



Another potential problem may arise in situations when individuals do not wish to answer all the risk-aversion related questions or if they answer them fast, without thinking too much about the lottery questions. Specifically in the income-gamble question, the individuals may incline to always choose one of the extremes. Such a measurement would be too noisy to provide any meaningful information about individual risk tolerance. In figure 4 I provide evidence that this is not the case in the HRS survey.

Figure 4:



Conclusion

This paper investigates how adverse life experiences influence the inequality in risk tolerance that stems from genetic endowment. To identify the role of adverse experiences, I use data concerning the historical US unemployment rate. To capture the multidimensional problem of genetic predispositions for risk tolerance, I construct a polygenic score variable. Thus, the paper exploits the variation on the birth cohort level. I adopt a non-linear model developed by Malmendier and Nagel (2011) that allows me to estimate an effect of a cohort level variable while at the same time controlling for the age and time fixed effects.

Previous research shows that elicited risk tolerance is related to many real life outcomes, which means that heterogeneity in the elicited risk tolerance has consequences for inequality in health, income, or wealth. Consequently a substantial body of empirical economic research has investigated whether and how changes in the environment influence risk attitudes. Despite this effort, there is little concensus on the matter. Hence, the literature does not provide a clear message about whether preferences for risk taking can change during life and, if they do, in what direction. Parallel to the discussion in economics, there is a debate in behavioral genetics that investigates the genetic component of the variation in risk attitudes. However, this strand of literature does not take into account the potential interaction of genetic endowment with choices and environments, which makes the conclusion incomplete and skewed.

This paper combines the knowledge of both strands of the literature and aims to estimate a gene-environment model in the setting of risk attitudes, providing a more precise picture of the architecture of risk attitudes. Specifically, the paper investigates whether previous life experiences of changes in the unemployment rate affect risk tolerance and how this effect varies for individuals with high and low genetic predispositions for risk taking. Thus, the paper provides an important insight into the relationship between adverse economic conditions and risk attitudes.

The results show that individuals with low genetic predispositions for risk tolerance are, on average, 30 percentage points more susceptible to adverse shocks on the labor market than individuals with high genetic predispositions for risk tolerance. Thus, the paper demonstrates that individuals who have low genetic predisposition to risk tolerance became less risk-tolerant in response to adverse changes on the labor market. In contrast, individuals with high genetic predisposition to risk tolerance are not significantly by such changes. Hence, unfavorable development on the labor market may further accentuate the inequality in attitudes towards risk that arise because of differences in initial genetic endowment. The increased inequality in risk aversion may lead to a further increase in income inequality. The findings also suggest that, in order to uncover a more detailed and precise picture of risk attitudes formation, it is necessary to take into account both genetic

and socio-economic factors. Although these two factors may contribute independently to risk attitudes, an important feature uncovered by the paper is that they complement or substitute each other in the risk-attitudes-formation model.

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