Economic Preferences, Genes, and Childhood Disadvantage

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Abstract

Motivated by the Scarr–Rowe hypothesis—which suggests that the expression of genetic potential depends on early-life conditions—this paper investigates how a genetic predisposition toward success in education interacts with the childhood environment to shape economic preferences. Using incentivized experimental data from a representative sample of English adults over the age of 50, we find that a higher genetic propensity for educational success is associated with lower risk aversion and greater patience—but only among those who experienced more advantaged childhood environments. Among participants who experienced an adverse early-life environment, risk aversion increases with genetic resources, whereas the positive relationship between genetic resources and patience is significantly attenuated. These findings suggest that early-life conditions can channel genetic resources in ways that are contextually adaptive. Concurrently, the results indicate that environmental inequality shapes the realization of genetic potential in ways that reinforce patterns of social immobility, thereby intensifying concerns regarding the systematic underutilization of potential arising from constraints on household resources.

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The phrase 'nature and nurture' is a convenient jingle of words, for it separates under two distinct heads the innumerable elements of which personality is composed. Nature is all that a man brings with himself into the world; nurture is every influence without that affects him after his birth — Sir Francis Galton (1874, p.12)

1. Introduction

Individual differences in cognitive development are shaped not only by genetic endowments but also by childhood environments (Bornstein & Putnick, 2012; Metcalfe, Harvey & Laws, 2013; von Stumm, 2012). The Scarr–Rowe hypothesis posits that genetic potential for cognitive ability is more fully realized in supportive, resource-rich settings (Rowe, Jacobson, & Van den Oord, 1999; Scarr-Salapatek, 1971; Tucker-Drob, Briley & Harden, 2013). That is, in the absence of environmental stressors in childhood (e.g., poverty, lack of educational access), cognitive genetic predispositions have a stronger predictive effect on cognitive phenotypes in later life. Empirical support from both twin and molecular genetic studies shows that genetic influences on cognitive performance and educational attainment are amplified in higher socioeconomic contexts (Harden, Turkheimer, & Loehlin, 2007; Ronda et al., 2022; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). Such environments foster synaptic growth, enhance neural connectivity (Dickens & Flynn, 2001), improve micronutrient status (Benton, 2008), and reduce cortisol exposure (Hackman, Farah, & Meaney, 2010), while also increasing access to cognitively enriching experiences (Bradley & Corwyn, 2002).[1]

[1] Other studies have found support for the compensatory advantage hypothesis, in which the cognitive trajectories of individuals from privileged backgrounds are less influenced by an adverse genetic allocation (Bernardi, 2014). In this context, genetic predispositions are less predictive of cognitive outcomes in later life, potentially because high-SES (socioeconomic status) families may (consciously or unconsciously) offset their children's lower genetic

While most gene-by-environment research has focused on the expression of genetic cognitive potential in cognitive phenotypes—such as cognitive ability and educational attainment—comparatively little attention has been paid to how this potential manifests in other traits where cognitive skills play a central role. In this study, we focus on economic preferences—traits related to decision-making under risk and over time.

Understanding how individuals make decisions under uncertainty and over time is a cornerstone of economic theory, and the study of these decision-making preferences has become central to fields such as behavioral science, psychology, finance, and development economics (Frederick, Loewenstein, & O'Donoghue, 2002; Rabin, 2000). Economic preferences play a fundamental role in shaping individual behavior and long-term socioeconomic outcomes. For example, more patient individuals are less likely to engage in criminal activity (Åkerlund, Golsteyn, Grönqvist & Lindahl, 2016), and tend to achieve higher levels of educational attainment, occupational status, income, and wealth (Cadena & Keys, 2015; DellaVigna & Paserman, 2005; Golsteyn, Grönqvist & Lindahl, 2014; Ventura, 2003). Similarly, individual differences in risk preferences are associated with labor market performance, investment behavior, health outcomes, and addictive behaviors (Anderson & Mellor, 2008; Barsky et al., 1997; Bonin et al., 2007; Dohmen & Falk, 2011). Economic preferences also provide a compelling context for studying gene-by-environment interactions, given their well-established theoretical and empirical associations with cognitive ability (Dohmen et al., 2010) and early-life environments (Caner & Okten, 2010; Eckel et al., 2012; Falk et al., 2021; Shah, Mullainathan & Shafir, 2012; Sheehy-Skeffington, Sidanius & Price, 2016; Sheehy-Skeffington, 2020), which we discuss in more detail below.

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endowments through compensatory investments or supportive traits (Ghirardi et al., 2024; Holm, Hjorth-Trolle & Jæger, 2019).

As Frederick (2005) argued, “there is no good reason for ignoring the possibility that general intelligence or various more specific cognitive abilities are important causal determinants of decision making” (p. 25). A number of empirical studies have since focused on the relationship between cognitive abilities and decision-making under risk and over time, finding that individuals with higher cognitive ability tend to exhibit greater patience and lower risk aversion (Dohmen et al., 2010, 2018; Shamosh & Gray, 2008). Consistently, higher cognitive ability is also associated with many of the same socioeconomic outcomes as more patient and less risk-averse preferences—such as higher income, greater occupational success, and increased wealth accumulation (Hafer, 2017; Heckman & Vytlacil, 2001; Hanushek & Woessmann, 2008; Sala-i-Martin et al., 2004; Schmidt & Hunter, 2004; Strenze, 2007). Furthermore, research in psychology and behavioural economics provides many channels through which cognitive skills influence decision-making under risk and over time. For instance, cognitive limitations can lead to narrow bracketing (Abeler & Marklein, 2017; Oberrauch & Kaiser, 2022; Rabin & Weizsäcker, 2009; Warsitzka et al., 2022), where decision-makers focus only on certain aspects of the consequences of a choice while ignoring others (Read, Loewenstein & Rabin, 1999). Here, narrow bracketing can lead to risk aversion and myopic preferences if individuals, for instance, fail to recognize how risky or intertemporal choices integrate with lifetime wealth (Tversky & Kahneman, 1981; Read, Loewenstein & Rabin, 1999).

A further channel is provided by the two-system approach to decision-making from the ‘heuristics and biases’ literature (Kahneman & Frederick, 2005), which emphasizes the interplay between emotions and cognition in decision making. Here, risk aversion and myopic preferences are thought to be universal properties of System 1, the emotional system that operates with little or no effort and no sense of voluntary control (Tversky & Kahneman, 1996). Those low in cognitive skills are typically regarded as less able to override the emotional

system and engage the deliberative, logical and analytical system, System 2, where reasoning is risk neutral and patient (Benjamin, Brown & Shapiro, 2013; McClure et al., 2004; Shiv & Fedorikhin, 1999; Shiv et al., 2005; Stanovich & West, 2008).

Low-SES (socioeconomic status) childhood environments and early-life stress have also been shown to influence the formation of economic preferences (Caner & Okten, 2010; Eckel et al., 2012; Falk et al., 2021; Shah, Mullainathan & Shafir, 2012; Sheehy-Skeffington, Sidanius & Price, 2016; Sheehy-Skeffington, 2020). However, the literature has been unable to provide a consensus perspective. For instance, the life history theory (Roff, 1992) suggests that early exposure to stress or scarcity leads to a 'fast' life strategy—prioritizing immediate rewards and risk-taking in response to environmental unpredictability—while stable environments encourage a 'slow' strategy, favoring future rewards and risk aversion (Griskevicius et al., 2011; Griskevicius et al., 2013; Pepper & Nettle, 2017; Simpson et al., 2012). In contrast, the uncertainty management perspective (Amir & Jordan, 2017; Amir, Jordan & Rand, 2018) sees early stress as a signal of future danger, shaping preferences that help manage uncertainty, such as impatience and risk aversion. Empirical evidence supports both accounts: early-life stress is consistently linked to impatience, while findings on risk preferences are mixed—predicting both greater risk-taking (e.g., Simpson et al., 2012; Wu et al., 2020) and greater risk aversion (Amir, Jordan & Rand, 2018).[2]

[2] The relationship between poverty and deprivation is complicated. For instance, risk-taking is often assumed to be a behavioural problem associated with low-income groups, but usually in regard to behaviours such as unsafe sexual practices, drug use, and problem gambling (Sheehy-Skeffington & Rea, 2017) as opposed to behaviours regarding occupational choice and important investment decisions (Caner & Okten, 2010; Eckel et al., 2012). Further inconsistency comes from the contrasting perspectives on poverty leading to vulnerability to

How early-life conditions and environments interact with cognitive factors to shape economic preferences is an open question. However, existing theories from developmental psychology, psychobiology, cognitive neuroscience, and behavioral genetics offer important insights. As noted, according to the Scarr–Rowe hypothesis (Scarr-Salapatek, 1971; Rowe, Jacobson & Van den Oord, 1999; Tucker-Drob, Briley & Harden, 2013), genetic potential for cognitive ability is more likely to be expressed in enriched, advantaged environments. If phenotypic cognitive skills are a key input into economic preferences, as the literature suggests (Dohmen et al., 2010; Burks et al., 2009; Frederick, 2005), then cognitive polygenic potential may be more predictive of such preferences among individuals from more advantaged childhood backgrounds.

However, gene-by-environment influences on economic preferences need not operate exclusively through cognitive phenotypes. Consistent with the Experiential Canalization Framework (ECF; Blair & Raver, 2012; Gottlieb, 1991), early-life adversity may impose pervasive constraints that interfere with the developmental processes and pathways that typically allow genetic predispositions to shape behavior. For instance, childhood adversity is associated with heightened emotional reactivity, chronic stress, negative affect, and reduced emotional regulatory control (Evans & Kim, 2013; Gunnar, 2000; Gunnar & Quevedo, 2007; Kim et al., 2013; Potter, Bridger, Piotrowska & Drewelies, 2025; Raposa et al., 2014; Sheikh, 2018), which promote reliance on fast, intuitive (System 1) decision-making at the expense of deliberative (System 2) processes (Blanchette & Richards, 2010; Eysenck et al., 2007; Muraven & Baumeister, 2000; Oldrati et al., 2016; Shields, Sazma & Yonelinas, 2016; Shiv et al., 2005;

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resource loss (i.e., avoiding risk) or desperation (i.e., risk-seeking; Banerjee, 2004; de Courson, Frankenhuis & Nettle, 2025) and indeed, poverty-related concerns reducing the cognitive capacity to make prudent financial decisions (Mani et al., 2013).

Simonovic et al., 2017). In a similar way, negative affect and anxiety can influence decision-making by inducing a generalized overestimation of risks (Butler & Mathews, 1987; Stöber, 1997) and an attentional bias toward threatening information (Gasper & Clore, 1998)—potentially attenuating or even blocking the behavioral expression of genetic propensities related to cognition.

Whereas the ECF focuses on how adverse environments suppress genetic expression—thereby limiting variability in traits—Adaptive Calibration Models (ACMs; Del Giudice et al., 2011) emphasize how biological systems (e.g., stress reactivity) are adaptively calibrated by early-life cues to prepare the individual for anticipated future environments. In this framework, genetic expression is not blocked, but rather channelled in ways that are contextually adaptive. For instance, in advantaged environments, genetic resources may be calibrated toward long-term planning and opportunity recognition, while in disadvantaged environments, these same cognitive resources may be directed toward detecting environmental threats, uncertainty, and resource scarcity—leading to more conservative decision-making strategies that prioritize security (Belsky & Pluess, 2009). Indeed, evidence suggests that early adversity can result in enhanced cue detection and greater physiological sensitivity to environmental challenges—particularly among individuals with higher processing capacities (Obradović et al., 2010; Obradović, 2012). This is consistent with Frankenhuis and de Weerth (2013), and Frankenhuis, Panchanathan, and Nettle (2016), who review the growing body of evidence showing that early-life stress and harsh environments can lead to the specialization of certain cognitive functions. It also aligns with the analysis by Chowdhury, Sutter, and Zimmermann (2022), which finds, in a developing country, a negative correlation between IQ and patience, as well as a negative influence of mothers' education on risk tolerance. Using data from the Global Preferences Survey of Falk et al. (2018), the authors further show a positive relationship

between cognitive ability, risk-taking, and patience in high-income countries, and a negative relationship in low-income countries.

In this paper, we investigate these potential gene-by-environment mechanisms and address this notable gap in the literature—one likely driven by the demanding data requirements involved, including rich measures of childhood environment, incentivized experimental assessments of risk and time preferences, and genetic markers related to cognitive potential. We leverage a unique dataset of 624 individuals that includes these essential components, as well as a larger sample of 5,881 individuals in which time preferences are captured through a validated survey-based measure. Our data provide detailed indicators of early environments—encompassing human capital, material resources, and household (in)stability—as well as a polygenic score for educational attainment—which proxies cognitive and non-cognitive traits relevant to educational success in addition to the '*g'* factor of intelligence (Belsky et al., 2016; Lee et al., 2018; Okbay et al., 2016; Smith-Woolley, Selzam & Plomin, 2019).

By exploring how early environmental factors shape the expression of genetic propensity for educational success onto economic preferences, we extend the literature on gene-by-environment interactions beyond "pure" cognitive outcomes to foundational behavioral traits that influence economic decision-making. This work provides new insights into how nature and nurture jointly contribute to the development of preferences that matter for life outcomes. In doing so, we add to a growing body of research that seeks to uncover the mechanisms underlying intergenerational inequality and the developmental origins of economic behavior (Chetty et al., 2014; Heckman, 2006).

2. Materials and Method

2.1. Participants

This study is based on the analysis of anonymized secondary data from the English Longitudinal Study of Ageing (ELSA) available from the UK Data Service. Information on the ethical approval received for each wave of ELSA can be found at: https://www.elsa-project.ac.uk/ethical-approval. This study (application reference number: 10956-12470) received a favorable opinion through the University of Bath's Social Science Research Ethics Committee's review process.

We use the English Longitudinal Study of Ageing (ELSA) 2002 to 2023 (Waves 1-10). ELSA is a representative sample of older people (aged 50 and over) in England, originally drawn from participants in the Health Survey for England, an annual national survey. Starting in 2002 (Wave 1), ELSA involves re-interviewing participants approximately every two years, with younger groups replaced or refreshed (Waves 3, 4, 6, 7, 9) to retain the panel. In Wave 5, 1,063 respondents aged 50-75 completed an incentivized experimental module designed to measure risk and time preferences.[3] ELSA also provides genotyped data for 7,412 participants, and polygenic scores (PGSs) available for several behavioural phenotypes, including educational attainment. We combine the Wave 5 experimental data, the genotyped data, and the life-history interview module from Wave 3—which captures information about key life events, including childhood environment. This yields an analytic sample, which we refer to as the experimental sample, of 624 individuals who had valid polygenic scores, responses to the incentivized experimental module, and completed the life-history interviews. Additionally, we

[3] Participants in the experimental module were paid a participation fee of £10. The choice tasks in the experimental module involved real (but small) payoffs, and at the end of the module one of the tasks was randomly picked and the respondent won the amount of money corresponding to their choice for this task. Participants could not lose more than £5 from their initial £10 participation fee and the expected payment was approximately £35.

use a survey-based measure of time preferences from Waves 1 and 2, resulting in a separate analytic sample, the survey sample, comprising 11,521 observations from 5,881 individuals. The mean age of the experimental (survey) sample is approximately 64 (64) years and 46% (45%) are male. Table S1 in Section A of the Supplemental Material provides an overview of the sample characteristics for the key variables used in the subsequent analysis.

2.2. Polygenic Score (PGS) for Educational Attainment

Polygenic scores (PGSs) represent an individual's genetic propensity toward a given phenotype. They are calculated as the sum of genome-wide genotypes—i.e., the combination of alleles across many loci—weighted by effect sizes (e.g., beta coefficients) derived from genome-wide association study (GWAS) summary statistics. GWAS test hundreds of thousands of genetic variants, typically single nucleotide polymorphisms (SNPs), to identify those statistically associated with a specific phenotype; in our case, educational attainment (EA). Because most phenotypes are polygenic, involving many SNPs of small effect, an individual's polygenic score is calculated as a weighted sum of their SNP genotypes:

$$PGS_i^{EA} = \sum_{j=1}^{j} W_j^{EA} G_{ij} \qquad (1)$$

where PGS_i^{EA} is the educational attainment polygenic score for individual i, G_{ij} represents the genotype of individual i at SNP j, and W_j^{EA} is the GWAS-derived effect size (beta) for SNP j.

We used a single p-value threshold of 1 (i.e., including all SNPs), as prior research has shown that such PGSs either explain the most phenotypic variance or perform comparably to scores constructed using more restrictive thresholds (Iob, Ajnakina, & Steptoe, 2023). Our PGS for educational attainment is based on the GWAS summary statistics from Lee et al. (2018; for additional details on PGS construction in ELSA, see Ajnakina and Steptoe (2022)). The PGS

for educational attainment is associated with larger brains, a range of cognitive functions—such as learning, neural function, cognitive development, and regions of the brain associated with language, memory, visual recognition, and cognitive processing—in addition to the '*g*' factor of intelligence (Belsky et al., 2016; Elliott, 2019, Lee et al., 2018; Mitchell et al., 2020; Okbay et al., 2016). However, it should be noted, studies have also shown a genetic overlap between educational attainment and non-cognitive traits related to educational success, including personality domains such as Neuroticism and Openness to Experience (Smith-Woolley, Selzam & Plomin, 2019).

2.3. Risk Preferences

In Wave 5, as part of the incentivized experimental module designed to measure risk and time preferences, subjects responded to the incentive-compatible task by Binswanger (1980, 1981) and Eckel and Grossman (2008; B-EG). The B-EG procedure required respondents to make a choice between 6 lotteries. These lotteries are illustrated in Table 1. Assuming Expected Utility Theory (EUT) and Constant Relative Risk Aversion (CRRA; and assuming that the experimental prizes are integrated into a background income equal to zero), the coefficient of relative risk aversion associated to each choice is described by the intervals reported in the rightmost column in Table 1. Within this CRRA functional form, a score equal to zero implies risk-neutral choices, a score above zero denotes risk aversion, and scores below zero implies risk-seeking behavior. Therefore, responses to the BE-G task allow us to infer the upper and lower bounds of participants risk aversion coefficient, which we use directly in the formal empirical analysis.

Table 1: B-EG Lottery Probabilities, Expected Value, Standard Deviation and CRRA Range

	Payoff				**CRRA ranges**	
Lottery	Low	High	Expected value	Std. Dev.	Lower bound	Upper bound
A	28	28	28	0	3.46	∞
B	24	36	30	8.5	1.16	3.46
C	20	44	32	17	0.71	1.16
D	16	52	34	25.5	0.499	0.71
E	12	60	36	33.9	0	0.499
F	2	70	36	48.1	-∞	0

Respondents were also asked to complete an adapted version of the B-EG procedure that includes losses. Indeed, loss aversion (Kahneman, & Tversky, 1979)—the tendency to feel more pain when experiencing losses than pleasure from equal gains—is the primary driver of risk-taking behavior in the context of small-stakes gambles (Barberis, Huang & Thaler, 2006). These lotteries are illustrated in Table 2, alongside the expected values and standard deviations. We utilize the full distribution of responses from the adapted BE-G procedure in the formal empirical analysis, as well as a binary indicator which equals one if the respondent picked Lotteries A, B or C, and zero otherwise (i.e., Lotteries D, E or F, that contain losses).

Table 2: Loss Aversion Lottery Probabilities, Expected Value and Standard Deviation

	Payoff			
Lottery	Low	High	Expected value	Std. Dev.
A	10	10	10	0
B	6	18	12	8.5

C	2	22	12	14.1
D	-2	28	13	21.2
E	-4	35	15.5	27.6
F	-5	38	16.5	30.4

2.4. Time Preferences

As part of the experimental session in Wave 5, following Harrison, Lau and Williams (2002) and Andersen et al. (2008), subjects are asked a sequence of questions within a multiple price list (MPL) to elicit time preferences. Specifically, respondents are asked to choose between £25 in a "sooner" period or £25 + x in a "future" period of $time\ t + T$, where $x > 0$. The "sooner" period was two weeks' time, and the "future" periods of time corresponded to 1 month and 2 months. Therefore, each participant responded to a total of two MPLs. The MPLs are illustrated in Table 3. We are interested in the point at which the respondent switches from Option A to Option B, which would reveal the upper and lower bounds of the respondent's discount rate. For example, if an individual takes the sooner option, Option A, in Payoff Alternative 4 in Panel A of Table 3, then takes the future option, Option B, in Payoff Alternative 5, assuming exponential discounting we can infer that the respondents weekly discount rate lies between 10.69% and 14.85%. Therefore, responses to the MPL tasks allow us to infer the upper and lower bounds of participants discount rate over different time horizons, which we use directly in the formal empirical analysis. Respondents who have unusually high (low) discount rates may not switch, instead accepting Option A (B) in all Payoff Alternatives. For these respondents the bounds of the inferred weekly discount rate are less precise. Lastly, there are a number of inconsistent subjects in our sample—those who for instance, after having chosen Option B in Payoff Alternative 5, switched back to Option A in Payoff Alternative 6. In the MPL where the future period was 1 (2) month(s) there were a total of 60 (47) subjects

who made inconsistent choices. For this group we take their first switching point, and we include in the subsequent analysis a dichotomous control variable that equals one if the subject was inconsistent and zero otherwise.

Table 3. Payoff Table for Time Preference Tasks

Payoff Alternative	**Payment Option A** *(Two Weeks)*	**Payment Option B** *(One Month)*	**Payment Option B*** *(Two Months)*	**Switching Point –Weekly Discount Rate**	
				Lower bound	Upper bound
Panel A					
1	£25	£26		$-\infty$	0.0163
2	£25	£28		0.0163	0.0477
3	£25	£30		0.0477	0.0779
4	£25	£32		0.0779	0.1069
5	£25	£35		0.1069	0.1485
6	£25	£38		0.1485	0.1880
*				0.1880	∞
Panel B					
1	£25		£26	$-\infty$	0.0059
2	£25		£30	0.0059	0.0275
3	£25		£35	0.0275	0.0514
4	£25		£37	0.0514	0.0602
5	£25		£40	0.0602	0.0726
6	£25		£45	0.0726	0.0915

*		0.0915	∞

Note. These weekly discount rates assume exponential discounting, specifically, that the present value PV of a future reward FV is given by: $PV = FV/(1+d)^t$, where d is the discount rate and t is the time delay in weeks. * Indicates the discount rate bounds for those participants who never switched, that is, those that always chose Payment Option A.

We also supplemented our experimental measure of time preferences with a question on planning horizons from the main survey. Specifically, in Waves 1 and 2 respondents were asked: *"In deciding how much of your income to spend or save, people are likely to think about different financial planning periods. In planning your saving and spending, which of the following time periods is more important to you?"*. This question has been used in previous research as a marker of more general future-orientation, or time perspective in financial behaviour (Gladstone & Hundtofte, 2023; Picone, Sloan & Taylor, 2004) and is correlated with other established markers of time perspective, including smoking and body mass index (BMI; Adams & Nettle, 2009). Respondents can choose from seven possible answers, indicating planning timelines ranging from: '*does not plan/plans day to day'*; '*the next few weeks'*; '*the next few months'*; '*the next year'*; '*the next few years'*; '*the next 5-10 years'*; or '*longer than 10 years'*. In addition to analyzing the full distribution of responses to the spending horizon question, we construct a binary indicator equal to one if the respondent's reported horizon extends beyond the next year, and zero otherwise. This threshold aligns with standard distinctions in the strategic management and financial planning literature between short-term and long-term financial planning.

2.5. Childhood Disadvantage

We take the approach in Ronda et al. (2022) and consider childhood disadvantage over four dimensions. The first dimension is human capital disadvantage, which is measured as having neither parent with education above the compulsory level, which at the time (1918-1947) meant children had to stay in school until the age of 14 (49.84% of the experimental sample; 41.71% of the survey sample). The second dimension measured family resources and is measured using a question on the main carer's occupation when the respondent was aged 14. The classifications include armed forces; manager or senior official; running own business; professional or technical; administrative, clerical or secretarial; skilled trade; caring, leisure, travel or personal services; sales or customer service; plant, process or machine drivers; other jobs; something else; casual jobs; retired; unemployed; and lastly, sick / disabled. Family resource disadvantage was main carer's occupation falling into either of the following categories: plant, process or machine drivers; other jobs; something else; casual jobs; unemployed; and lastly, sick / disabled (27.56% of the experimental sample; 33.14% of the survey sample). Our third dimension is another measure of family resources disadvantaged, measured as growing up in a home with no central heating or hot water (23.72% of the experimental sample; 28.72% of the survey sample). The fourth dimension is a measure of family instability. We measured instability as parents being permanently separated or divorced before the respondent was aged 16, or having not lived with both natural parents for most of their childhood (15.22% of the experimental sample; 16.31% of the survey sample). In the experimental (survey) sample, 27.72% (28.45%) respondents experienced no dimension of disadvantage, 37.18% (35.88%) experienced one dimension of disadvantage, 27.08% (24.55) experienced two dimensions of disadvantage, 7.05% (9.59%) experienced three dimensions of disadvantage, and lastly, 0.96% (1.53%) experienced all four dimensions of disadvantage. Following Ronda et al. (2022) we categorize disadvantage as those respondents who experienced two or more dimensions of disadvantage, which corresponds to 35.10% (35.67%) of the experimental (survey) sample. It

should also be noted that for the experimental (survey) sample 29.01% (25.06) respondents had a missing response to one of the four disadvantage dimensions, with 1.76% (16.51%) of respondents having a missing response to two of the four disadvantage dimensions. To maintain statistical power, we treat missing values as zero.[4]

2.6. Analytic strategy

The first step of our analysis is to validate the polygenic score for educational attainment (PGS EA) by estimating its relationship with two cognitive phenotypes: educational attainment and cognitive ability. In our analysis we use the standard between-family model for gene-by-environment interactions (Papageorge & Thom, 2020; Ronda et al., 2022), described as:

$$P_{ig}^{k} = \alpha^{k}(g)PGS_{ig}^{EA} + X_{ig}b^{k}(g) + \epsilon_{ig}^{k} \qquad (2)$$

where P_{ig}^{k} is the kth phenotypic outcome (i.e., educational attainment or cognitive ability) for individual i in group g (i.e., 'no disadvantage' or 'disadvantage'), PGS_{ig}^{EA} is the polygenic score for educational attainment, and X_{ig} is a vector of exogenous control variables including year of birth, sex, and the first 10 principal components of the respondent's SNPs—which

[4] In Section A of the Supplemental Material, we show that the results are almost identical when restricting the sample to those respondents who gave valid responses to each of the four dimensions of disadvantage (Tables S2 and S3) and using the missing indicator method, whereby we include a dichotomous control variable for missingness (Tables S4 and S5). We also consider alternative specifications of childhood disadvantage, including each dimension individually (Tables S6 and S7) and the number of dimensions experienced during childhood (Tables S8 and S9).

allows us to control for any ancestry differences in genetic structures. For instance, if a particular SNP variant is more common in a specific ancestry group, an observed association between PGS^{EA} and phenotypic outcomes may reflect cultural norms shared by this ancestry group. Lastly, ϵ_{ig}^{k} is the usual random error component. This approach allows all parameters to differ across g, with our primary interest being the estimated genetic effects, α^{k} and the comparison of the estimated genetic effects by group, g.

After establishing validity, we focus on the impact of PGS EA on the formation of economic preferences, and the comparison of the estimated genetic effects between the groups. Again, we use the standard between-family model for gene-by-environment interactions, described as:

$$Y_{ig}^{h} = \alpha^{h}(g)PGS_{ig}^{EA} + X_{ig}b^{h}(g) + \epsilon_{ig}^{h} \qquad (3)$$

where Y_{ig}^{h} is the hth economic preferences (i.e., risk or time preference) for individual i in group g (i.e., 'no disadvantage' or 'disadvantage'), with the remaining variables defined in the same way as described for Equation (2). Here, our primary interest being the estimated genetic effects α^{k} and the comparison of the estimated genetic effects by group, g.

Our standard between-family approach, commonly used in gene-by-environment studies, faces an important limitation. While our polygenic score for educational attainment is plausibly exogenous—owing to Mendel's Law of Segregation, whereby individuals inherit genes randomly from their parents—a major concern is potential bias stemming from unmeasured environmental influences, particularly those arising from genetic nurture. Simply put, although genes are fixed at conception, they are determined by parental genes, which also shape the environment in which children are raised (Houmark, Ronda & Rosholm, 2020; Kong et al., 2018; Marks & O'Connell, 2023; Young et al., 2018). In our setting, if parents' genes

influence parenting styles—such as the time and resources invested in their children—which in turn affect the respondents' economic preferences, then the between-family model will ignore these effects of parental genes and incorrectly attribute them to the offspring's genetic endowment.

While data limitations prevent us from entirely eliminating environmental confounding—e.g., through within-family analysis (Fletcher et al., 2020; Ronda et al., 2022) or controlling for the polygenic scores of respondents' natal parents (Breinholt & Conley, 2023; Ghirardi et al., 2024)—we do have broad information on parenting styles, which we leverage as a robustness check. [5]

Open Practices Statement. The data that support the findings of this study are publicly available from the UK Data Archive. The complete STATA analysis script to replicate the results is openly available in Open Science Framework (OSF) at:

https://osf.io/f8teh/?view_only=72895eadfc3547e7ac6d15825981c0d2

This study was not preregistered.

3. Results

We first establish the validity of the polygenic score for educational attainment (PGS EA) by establishing its association with phenotypic educational attainment and cognitive ability, following Equation (2). Educational attainment is measured as a 7-point categorical variable, ranging from those with no formal qualifications to those respondents who reported a university or college degree. Cognitive ability is measured, following Dawson (2015), as a general IQ factor from a range of cognitive function tasks—designed to measure memory, executive

[5] Another limitation involves measurement error in the polygenic score. This bias will operate in the opposite direction to unmeasured environmental influences (Trejo & Domingue, 2018).

function, and basic cognitive skills/abilities—with acceptable-to-good internal consistency reliability across the items. For a full description of the cognitive tasks, see Section B of the Supplemental Material. In keeping with Equation (2), we allow the parameters to differ across group g and as a test of the Scarr–Rowe hypothesis, we compare the estimated genetic effects, $\alpha^k(g)$. The results are presented in Table 4 (Figure 1), where $\beta 1$ ($\beta 1 + \beta 2$) is an estimate of the effect of the PGS EA on the dependent variable for the 'no disadvantage' ('disadvantage') group and $\beta 2$ is the comparison of the estimated genetic effects, $\alpha^k(g)$, between the 'no disadvantage' and 'disadvantage' groups.

Table 4. Gene-Environment Interaction and Cognitive Phenotypes

Regression:	1	2	3	4
Dependent variable:	Educational attainment	Cognitive ability	Educational attainment	Cognitive ability
Sample:	Experimental		Survey	
Estimator:	Ordered Logit	OLS	Ordered Logit	OLS
$\beta 1$ PGS EA	0.392***	0.110***	0.519***	0.138***
	[4.022]	[4.215]	[23.616]	[17.273]
$\beta 2$ PGS EA × Disadv.	-0.132	0.020	-0.108***	-0.002
	[-0.882]	[0.398]	[-2.907]	[-0.145]
$\beta 1 + \beta 2$	0.260**	0.131***	0.412***	0.136***
	[2.279]	[2.953]	[13.769]	[11.608]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	11521	11521

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets (cluster-robust standard errors in Regressions 3 and 4). Additional controls include date of birth; sex; and the first 10 principal components of the respondent's SNPs. In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage'). Educational attainment is a categorical measure, indicating the highest level of attainment. These include university or college degree - either at undergraduate or postgraduate level and includes National Vocational Qualifications (NVQ's: practical work-based awards) at Level 4 (Higher Education Certificate) and Level 5 (Higher Education Diploma/Foundation Degree); Higher national certificates and diplomas below degree level - which includes diplomas in higher education, teaching qualifications, nursing or other medical qualifications; A-level - which are post-compulsory examinations taken at 18 to qualify for college or university entrance and includes NVQ's at Level 3; GCSE standard - which are a standard pass (Grades 9-4) in schooling attainment qualifications taken at 16 and includes NVQ's at Level 2; GCSE - which are a low pass (Grades 3-1) in schooling attainment qualifications taken at 16 and includes NVQ's at Level 1; other qualification - which includes school leaving exam certificate or matriculation; and lastly, no formal qualifications. Cognitive ability is a general cognitive factor from a barrage of cognitive tests designed to measure memory, executive function, and basic cognitive skills/abilities.

* $p<0.10$, ** $p<0.05$, *** $p<0.01$.

The results illustrate that PGS EA is a strong predictor of educational attainment and, in line with the Scarr-Rowe hypothesis, is less predictive for those who experienced childhood disadvantage. However, the gene-by-environment interaction is only statistically significant for the survey sample. For cognitive ability, the predictive power of PGS EA is independent of the childhood environment. This supports previous research that the influence of the childhood

environment, as a moderator for the heritability of cognitive factors, changes as people get older—with results showing the effect diminishes or disappears entirely at older ages (Gottschling et al., 2019; Tucker-Drob & Bates, 2016). This is consistent with the observation that the importance of early environmental factors for explaining phenotypic variance decreases over time for most traits (Briley & Tucker-Drob, 2013; Plomin & Daniels, 1987; Plomin & von Stumm, 2018; Segal & Pratt-Thompson, 2024; Turkheimer, 2000). Whilst the slope estimates are independent of childhood disadvantage, at every level of PGS EA those from backgrounds with childhood disadvantage have lower predicted cognitive ability. These relationships are illustrated in Figure 1, which plots the predict probability (score) for attainment of a university or college degree (cognitive ability), for each group g across the PGS EA distribution.

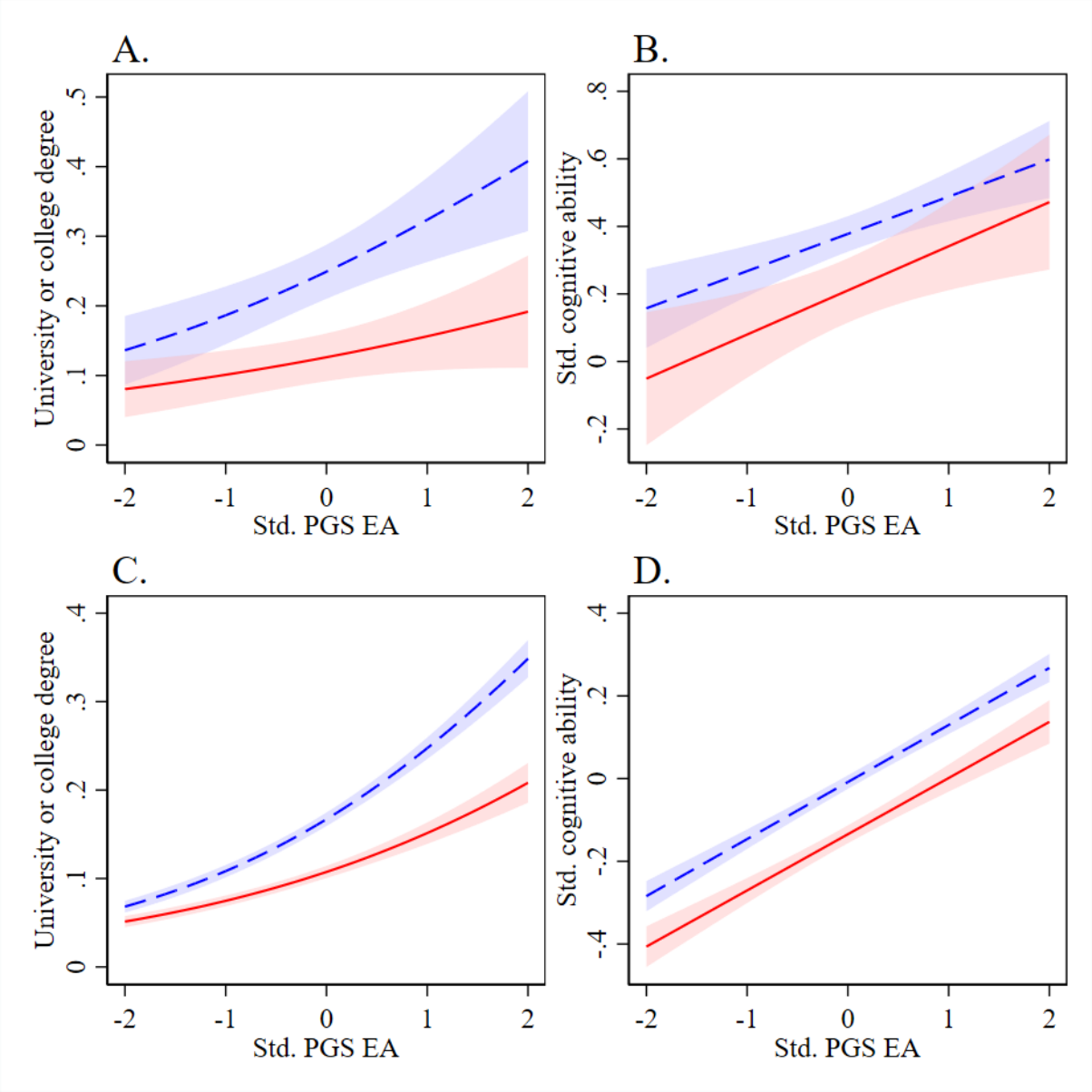

Fig. 1. Gene-Environment Interaction and Cognitive Phenotypes

Note. Dashed (solid) lines are for the 'no disadvantage' ('disadvantage') group. Shaded areas represent 95% confidence intervals. Panel A (Regression 1, Table 4). Panel B (Regression 2, Table 4). Panel C (Regression 3, Table 4). Panel D (Regression 4, Table 4).

We now turn our attention to Equation (3) and the influence of gene-by-environment interactions on the formation of economic preferences. In keeping with Equation (3), we allow

the parameters to differ across group g and as a direct test of the gene-by-environment interaction we compare the estimated genetic effects, $\alpha^k(g)$. The results are presented in Tables 5 and 6, where $\beta 1$ ($\beta 1 + \beta 2$) is an estimate of the effect of the PGS EA on the dependent variable for the 'no disadvantage' (disadvantage) group and $\beta 2$ is the comparison of the estimated genetic effects, $\alpha^k(g)$, between the 'no disadvantage' and 'disadvantage' groups. An alternative representation of the results contained in Tables 5 and 6 is provided in Figures 2 and 3, respectively, were we plot the predictions for each group for each level of standardized PGS EA.

Starting with Table 5, the estimated genetic effect operates in different direction for the 'no disadvantage' and 'disadvantage' groups. Specifically, for our 'no disadvantage' group the effect of PGS EA on risk aversion is negative and statistically significant—which is consistent with the literature on economic preferences and intelligence (Dohmen et al., 2010, 2018)—whilst for our childhood disadvantage group the effect is always positive, and in Regressions 1 and 2, statistically significant at the 5% level. These effects are not small. For instance, from Regression 1 of Table 5 (Panel A of Figure 2) and focusing on our 'no disadvantage' group, high-PGS EA respondents (+2 standard deviations from the mean) have a predicted risk aversion coefficient of 1.60 whilst the equivalent prediction for low-PGS EA respondents (-2 standard deviations from the mean) is 3.07. For our 'disadvantage' group, high-PGS EA respondents have a predicted risk aversion coefficient of 3.42 whilst the equivalent prediction for low-PGS EA respondents is 1.70. Furthermore, in all Columns, the comparison of the estimated genetic effects, $\alpha^k(g)$, between the 'no disadvantage' and 'disadvantage' groups is statistically significant. The pattern observed in our 'disadvantage' group is consistent with the 'fast' life strategy from life history theory (Roff, 1992)—prioritizing risk-taking in response to environmental unpredictability—but only under cognitive genetic disadvantage. Conversely, it aligns with the uncertainty management perspective (Amir & Jordan, 2017; Amir, Jordan &

Rand, 2018)—which emphasizes preferences that help manage uncertainty, such as risk aversion—but only under cognitive genetic advantage.

Table 5. Gene-Environment Interaction and Risk Preferences

Regression:	1	2	3	4
Dependent variable:	Risk aversion (B-EG)	Risk aversion (LA)	Risk aversion (LA-binary)	Risk aversion (LA-binary)
Estimator:	Interval	Ordered Logit	Logit	Logit
$\beta 1$ PGS EA	-0.368***	-0.350***	-0.543***	-0.483***
	[-3.000]	[-4.072]	[-4.677]	[-3.960]
$\beta 2$ PGS EA × Disadv.	0.799***	0.715***	0.706***	0.575**
	[3.609]	[4.135]	[3.446]	[2.524]
$\beta 1 + \beta 2$	0.431**	0.365**	0.164	0.092
	[2.334]	[2.442]	[0.968]	[0.478]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	624	624

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets. Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regression 4, we also include a series of dummy variables representing respondents' choice in the risk aversion lottery (B-EG). In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

The results reveal a similar pattern when turning to Regression 2 of Table 5 where our dependent variable is the adapted version of the B-EG procedure that includes losses. We order the lottery choices so that they reflect increasing levels of risk aversion. Here, focusing on our 'no disadvantage' group, high-PGS EA respondents (+2 standard deviations from the mean) have a predicted probability of choosing Lottery A in Table 2 (Panel B of Figure 2)—CRRA range = $[3.46, \infty]$—of 29.15% whilst the equivalent predicted probability for low-PGS EA respondents (-2 standard deviations from the mean) is 61.49%. Focusing on our 'disadvantage' group, high-PGS EA respondents have a predicted probability of choosing Lottery A of 66.22%, and 35.07% for low-PGS EA respondents. Equivalently, focusing on our 'no disadvantage' group, the predicted probabilities of choosing Lottery F in Table 2, for high-PGS EA respondents is 17.90%, and 4.83% for low-PGS EA respondents. For our 'disadvantage' group, the corresponding estimates are 5.18% and 16.80%, respectively. These results are confirmed in Regression 3 (Panel C of Figure 2), where we use a binary indicator which equals one if the respondent picked Lotteries A, B or C in Table 2, and zero if the respondent picked a lottery with a loss, i.e., Lotteries D, E or F. As an extension, in Regression 4 (Panel D of Figure 2) we estimate the effects on our binary measure of the adapted B-EG lottery that includes losses, but controlling for the respondent's choice in the standard B-EG lottery. This allows us to test whether the effects in Regression 3 remain statistically significant after controlling for risk preferences, which are known to be conceptually related to, but distinct from, loss aversion (Barberis et al., 2001; Rabin & Thaler, 2001). In short, the same pattern of results is revealed, suggesting that the gene-by-environment interaction exerts an independent effect on loss aversion, above and beyond the correlation with general risk tolerance.[6]

[6] The results are quantitively and qualitatively analogous when estimating Regression 2 of Table 5 whilst controlling for choices in the standard B-EG lottery.

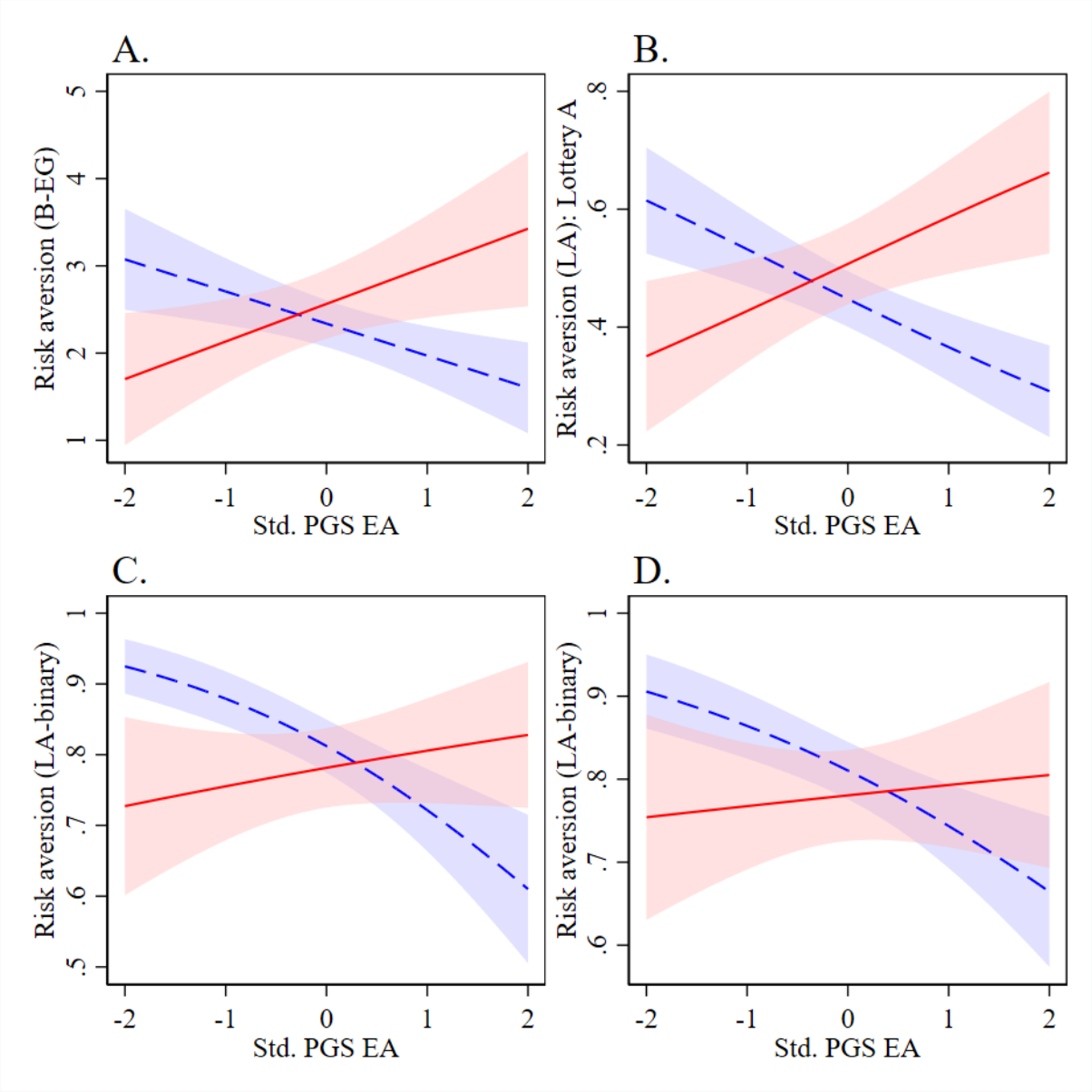

Fig. 2. Gene-Environment Interaction and Risk Preferences

Note. Dashed (solid) lines are for the 'no disadvantage' ('disadvantage') group, Shaded areas represent 95% confidence intervals. Panel A (Regression 1, Table 5). Panel B (Regression 2, Table 5). Panel C (Regression 3, Table 5). Panel D (Regression 4, Table 5).

Turning our attention to time preferences, the results in Table 6 suggest for our 'no disadvantage' group the effect of PGS EA on impatience is negative and statistically significant—which is consistent with the literature on time preferences and intelligence

(Dohmen et al., 2010; Shamosh & Gray, 2008). Again, these effects are not small. For instance, from Regression 1 of Table 6 (Panel A of Figure 3)—where our dependent variable reflects the weekly discount rate from the MPL where the larger-later rewards correspond to a time horizon of one month—and focusing on our 'no disadvantage' group, high-PGS EA respondents (+2 standard deviations from the mean) have a predicted weekly discount rate of -2.40% whilst the equivalent prediction for low-PGS EA respondents (-2 standard deviations from the mean) is 5.87%. The effects are comparable for the 'disadvantage' group, with the small difference between the groups not being statistically significant. Turning to Regression 2 of Table 6 (Panel B of Figure 3)—where our dependent variable reflects the weekly discount rate from the MPL where the larger-later rewards correspond to a time horizon of two months—the effect of PGS EA on the discount rate for our 'no disadvantage' group is again negative and statistically significant. However, the predictive effect is smaller for the 'disadvantage' group, although the difference is marginally statistically insignificant. Here, the longer delay in the future period (Regression 2 of Table 6) may have improved our ability to measure respondents' impatience. This is consistent with established evidence from the intertemporal choice literature, whereby longer delays tend to amplify impulsivity and present bias, making individuals more likely to choose the smaller-sooner reward (Frederick, Loewenstein, & O'Donoghue, 2002). This pattern aligns with the principles of hyperbolic discounting, which suggest that people disproportionately devalue rewards as the delay increases (Laibson, 1997).[7]

[7] We also estimate Regression 2 of Table 6, controlling for the respondents' discount rate over a 1-month delay. This estimation strategy allows us to estimate the degree to which respondents are disproportionately impacted by longer delays. The effect for the 'no disadvantage' group suggests that a higher PGS EA lowers present-bias ($b = -0.006, t = -2.41, p = 0.016$) whilst for the 'disadvantage' group the effect was positive ($b = 0.004, t =$

Table 6. Gene-Environment Interaction and Time Preferences

Regression:	1	2	3	4
Dependent variable:	Discount rate (1 month)	Discount rate (2 months)	Planning horizon	Planning horizon (binary)
Estimator:	Interval	Interval	Ordered Logit	Logit
$\beta 1$ PGS EA	-0.021**	-0.011***	0.199***	0.196***
	[-2.479]	[-3.690]	[7.913]	[7.183]
$\beta 2$ PGS EA × Disadv.	-0.007	0.007	-0.131***	-0.146***
	[-0.477]	[1.351]	[-3.263]	[-3.249]
$\beta 1 + \beta 2$	-.027**	-.004	.068**	.049
	[-2.419]	[-1.039]	[2.173]	[1.368]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	11521	11521

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets (cluster-robust standard errors in Regressions 3 and 4). Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regressions 1 and 2 we also include a dichotomous control variable that equals one if the subject made an inconsistent choice in the MPL (Multiple Price List) and zero otherwise. In all regressions, all

$1.29, p = 0.196$) with the difference being statistically significant ($b = 0.009, t = 2.48, p = 0.013$). See Figure S1 in Section A of the Supplemental Material for a diagrammatic representation of the results.

parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').
* p<0.10, ** p<0.05, *** p<0.01.

The results are more definitive when we turn our attention to Regressions 3 and 4 of Table 6, which utilize the survey-based question on planning horizons. For instance, when turning to Regression 3 (Panel C of Figure 3)—where our dependent variable is the ordinal variable of planning horizon—focusing on the 'no disadvantage' group, high-PGS EA respondents (+2 standard deviations from the mean) have a predicted probability of reporting a planning horizon of '*longer than 10 years'* of 10.48% whilst the equivalent predicted probability for low-PGS EA respondents (-2 standard deviations from the mean) is 5.05%. For our 'disadvantage' group, high-PGS EA respondents have a predicted probability of reporting a planning horizon of '*longer than 10 years'* of 6.70% whilst low-PGS EA respondents have a predicted probability of 5.19%. Equivalently, focusing on our 'no disadvantage' group, the predicted probabilities of reporting a planning horizon of '*does not plan/plans day to day'* for high-PGS EA respondents is 6.3%, and 9.43% for low-PGS EA respondents. For our 'disadvantage' group, the corresponding estimates are 12.82% and 11.99%, respectively.

Lastly, and consistently from Regression 4 of Table 6 (Panel D of Figure 3), focusing on our 'no disadvantage' group, the predicted probability of reporting a long-term planning horizon (i.e., that their planning horizon extends beyond the next year) is 60.11% for high-PGS EA respondents and 41.42% for low-PGS EA respondents. For our 'disadvantage' group the effects operate in the same direction, but the effect is smaller than for the 'no disadvantage' group. Furthermore, in both Regressions 3 and 4 of Table 6, the comparison of the estimated genetic effects, $\alpha^k(g)$, between the 'no disadvantage' and 'disadvantage' groups is negative and statistically significant. In summary, childhood disadvantage tends to attenuate the relationship between genetic potential and time preferences, meaning that no matter the genetic

cognitive potential, childhood disadvantage leads to behaviour that is relatively myopic. This is consistent with both the life history theory and the uncertainty management perspective, that is, prioritizing immediate rewards in response to environmental unpredictability and to manage uncertainty.

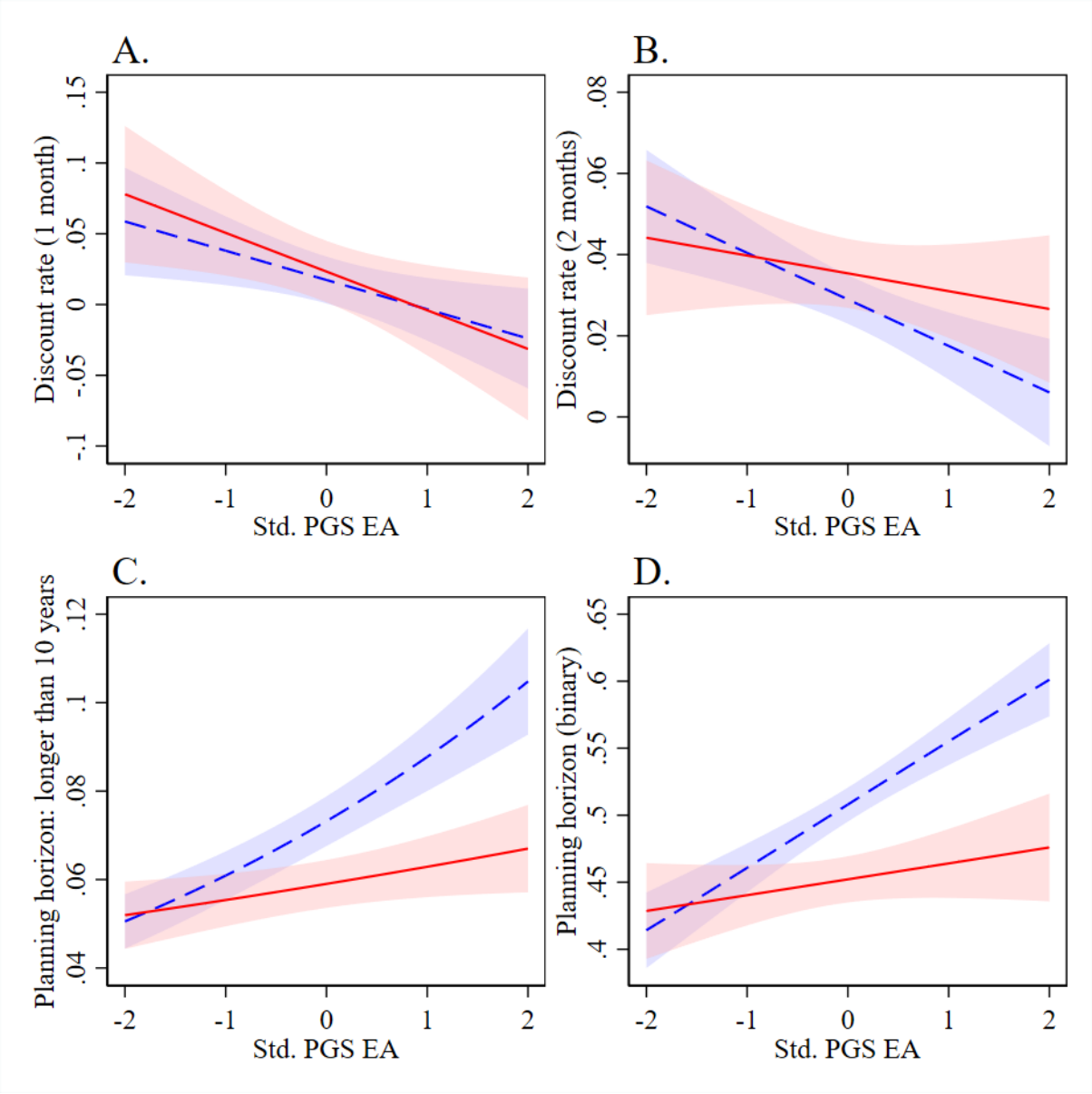

Fig. 3. Gene-Environment Interaction and Time Preferences

Note. Dashed (solid) lines are for the 'no disadvantage' ('disadvantage') group, Shaded areas represent 95% confidence intervals. Panel A (Regression 1, Table 6). Panel B (Regression 2, Table 6). Panel C (Regression 3, Table 6). Panel D (Regression 4, Table 6).

3.1. Robustness

Two main issues potentially arise from our main analysis, that is, (a) between-family gene-by-environment designs are potentially biased from unobserved environmental factors, such as genetic nurture (Houmark, Ronda & Rosholm, 2020; Kong et al., 2018; Marks & O'Connell, 2023; Young et al., 2018), and (b) PGS EA as well as capturing a broad array of cognitive functions (Belsky et al., 2016; Lee et al., 2018; Mitchell et al., 2020; Okbay et al., 2016) also exhibits genetic overlap with personality domains such as Neuroticism and Openness to Experience (Smith-Woolley, Selzam & Plomin, 2019).

Regarding (a), our results could be explained by dynastic effects—whereby the expression of parental genotype in the parent's phenotype directly affects the outcome variable. Specifically, genes, although fixed at conception, are inherited from parental genes, which also shape the childhood environment (Kong et al., 2018; Young et al., 2018). For instance, as illustrated in Figure 4—which shows the distribution of the PGS EA scores separately for each group—individuals from disadvantaged backgrounds have PGS EA scores that are 0.097 standard deviations lower than the overall sample mean in the experimental sample, and 0.101 standard deviations lower in the survey sample. This observed gap is expected, as individuals inherit their genetic makeup from their parents, whose own genetic traits influence their educational attainment, occupational status, and acquisition of resources.

In our setting, if there are other childhood environmental factors correlated with respondents' PGS EA—such as parenting practices—that are also associated with respondents'

economic preferences, then our between-family model will fail to separate the effects of parental genes on offspring economic preferences from the offspring's own genetic endowment. To address this possibility, we re-estimate Equation (3) and replicate Tables 5 and 6 including the Parental Bonding Instrument (PBI; Roe & Siegelman, 1963; Schaefer, 1965), which measures respondents' retrospective experiences of their parents' parenting style before age 16. The PBI focuses on parental care and overprotection, consisting of 14-items (7 for each parent) including "She/he let me do the things I liked doing"; "She/he made me feel I was not wanted" [reverse-coded]; and "She/he liked me to make my own decisions"; all on 1 ("strongly disagree") to 4 ("strongly agree") scales. We use the average score across items, where internal consistency reliability across the 14-items was good ($\alpha = 0.85$; average interitem correlation of 0.15). For non-responders (227 individuals in the experimental sample and 2,025 individuals in the survey sample) we code missing values as zero and including a separate dichotomous control indicator for missingness (i.e., the missing indicator method). Although the relationship between PGS EA and PBI is positive, the correlation is small and statistically insignificant for our experimental sample ($r = 0.023, p = 0.568$) and small and statistically significant for our survey sample ($r = 0.054, p < 0.001$). More importantly, the inclusion of PBI in the estimation of Equation (3) leads to results that are quantitively and qualitatively similar to those presented in the main text (see Tables S10 and S11 in Section A of the Supplemental Material).

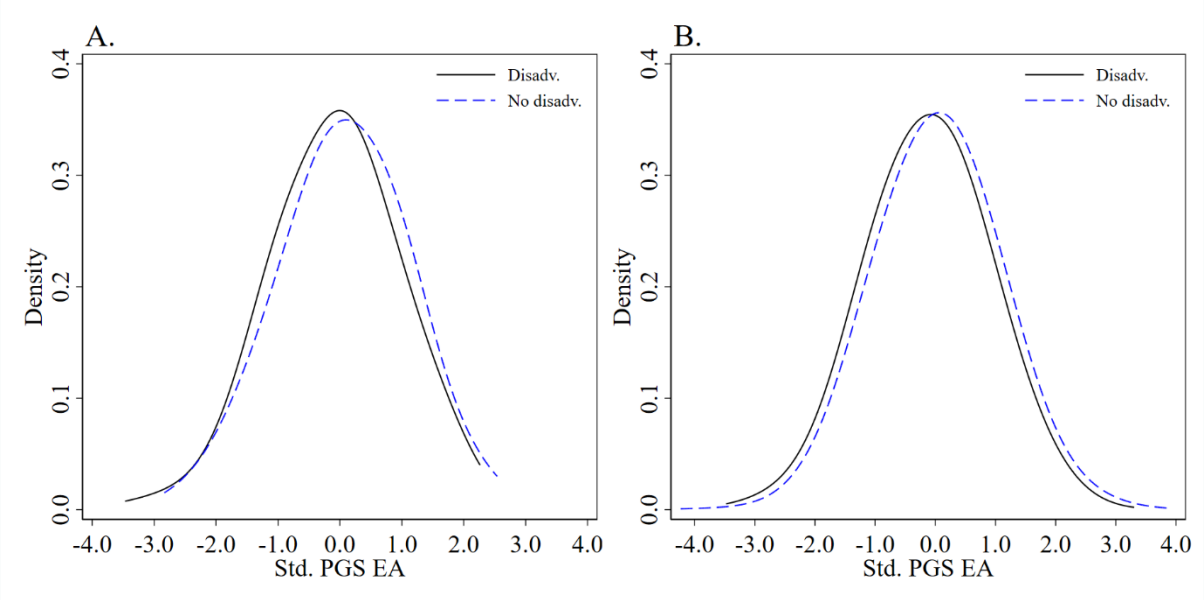

Fig. 4. Kernel density plots of standardized PGS EA by childhood disadvantage.

Note. Panel A: experimental sample of 624 individuals (219 'disadvantage' and 405 'no disadvantage' individuals), $p = 0.078$ (*t*-test), $p = 0.132$ (Kolmogorov-Smirnov test). Panel B: survey sample of 11,521 observations from 5881 individuals (4,119 'disadvantage' and 7,402 'no disadvantage' observations), $p < .001$ (*t*-test), $p < .001$ (Kolmogorov-Smirnov test).

Regarding (b), we aim to more precisely estimate the effect of genetic variants linked to cognitive function on economic preferences by accounting for the influence of non-cognitive genetic factors. To start, we focus on genetic variants associated with personality, given that polygenic scores for personality traits are correlated with PGS EA (Smith-Woolley, Selzam & Plomin, 2019). Importantly, personality traits have also been shown to influence risk-taking (Lauriola & Levin, 2001) and time preferences (Keidel et al., 2024; Manning, 2014). To this end, we re-estimate Equation (3) and replicate Tables 5 and 6 with the inclusion of polygenic

scores for the Big Five personality traits.[8] The results, presented in Tables S12 and S13 in Section A of the Supplemental Material, are nearly identical to those reported in the main text. Additionally, Tables S14 and S15 report the correlations between polygenic scores for personality traits and the PGS for educational attainment, for both the experimental and survey samples. Next, we re-estimate Equation (3) and replicate Tables 5 and 6, but replace the polygenic score for educational attainment (PGS EA) with the polygenic score for IQ (PGS IQ). Our PGS IQ is derived from the large-scale GWAS meta-analysis conducted by Savage et al. (2018), which focuses specifically on fluid cognitive ability—defined as an individual's capacity to process and integrate information, act, and solve novel problems. In contrast, PGS EA captures a broader set of cognitive traits associated with educational achievement (Lee et al., 2018). Importantly, PGS EA benefits from substantially larger and more statistically powerful GWAS datasets compared to those available for IQ, which translates into higher predictive accuracy in independent samples (Dudbridge, 2013). The smaller discovery sample for PGS IQ results in lower SNP-heritability capture and reduced statistical power, making it a less precise proxy for genetic influences on cognitive functioning. Despite these limitations, our re-analysis using PGS IQ (see Tables S16 and S17 in Section A of the Supplemental Material) yields results that are qualitatively similar to those reported in the main text, reinforcing the robustness of our findings. Similarly, our main findings are robust when using

[8] The GWAS meta-analyses for Extraversion was conducted by the Genetics of Personality Consortium (GPC; van den Berg et al., 2018); the polygenic scores for Agreeableness, Openness to Experience and Conscientiousness were based on the GWAS meta-analysis from Moor et al (2012); and the polygenic score for Neuroticism was calculated based in the GWAS summary statistics that collated results from the Genetics of Personality Consortium (GPC) and results from a new analysis of UKB data cohort (Okbay et al., 2016).

phenotypic IQ—for a full description of the cognitive tasks, see Section B of the Supplemental Material—instead of PGS EA as the predictor of economic preferences (see Tables S18 and S19 in Section A of the Supplemental Material). This approach, despite offering clearer attribution to cognitive ability, is however more susceptible to socio-economic and lifestyle confounders. Although, in our ELSA sample of individuals aged 50 and above, phenotypic IQ may reflect genetic influences more strongly than early environmental factors (Briley & Tucker-Drob, 2013; Plomin & Daniels, 1987; Plomin & von Stumm, 2018; Segal & Pratt-Thompson, 2024; Turkheimer, 2000).

Lastly, following Dohmen et al. (2010), we investigate whether the estimated genetic effects are indirect—operating through the channels of income, wealth, and education to influence economic preferences. Higher wealth and income may affect preferences by lowering the tangible and psychological costs of negative income shocks from risk-taking, or by reducing the need for immediate payoffs. Similarly, education may increase financial knowledge, which could help individuals better understand and evaluate probabilities and outcomes. To examine this, we re-estimate Equation (3) and replicate Tables 5 and 6, now including controls for the logarithm of net total household wealth (the sum of savings, investments, physical wealth, and housing wealth after financial debt and mortgage debt has been subtracted); the logarithm of annual total household income; and a set of dummy variables capturing the highest reported level of educational attainment.

Tables S20 and S21 in Section A of the Supplemental Material, report that both risk aversion and impatience are significantly correlated with the PGS for educational attainment, controlling for these additional characteristics. In Table S20, which focuses on risk preferences, we see that the coefficients are almost identical to those results presented in the main text. In Table S21, which focuses on time preferences, we see there is a large reduction in the (absolute) value of the coefficients relating impatience and the PGS for educational attainment, although

for our survey-based planning horizon measure, the coefficients of interest remain statistically significant at the 10% level or better. Taken together, the contrasting influences of genetic effects related to educational success on economic preferences in our 'no disadvantage' and 'disadvantage' groups do not operate solely through these indirect channels.

4. Discussion

Research has shown a robust link between cognitive ability and economic preferences (Dohmen et al., 2010, 2018; Shamosh & Gray, 2008) and how childhood disadvantage and poverty, impact our preferences towards risk and time (Bertrand, Mullainathan & Shafir, 2004; Caner & Okten, 2010; Carvalho, Meier & Wang, 2016; Eckel et al., 2012; Falk et al., 2021; Shah, Mullainathan & Shafir, 2012; Sheehy-Skeffington, Sidanius & Price, 2016; Sheehy-Skeffington, 2020). We illustrate, for the first time, the interplay of genetic endowments associated with educational success and childhood environmental factors in shaping economic preferences, which strongly influence important socioeconomic outcomes (Åkerlund et al., 2016; Anderson & Mellor, 2008; Barsky et al., 1997; Bonin et al., 2007; Cadena & Keys, 2015; DellaVigna & Paserman, 2005; Dohmen & Falk, 2011; Golsteyn, Grönqvist & Lindahl, 2014; Ventura, 2003).

Our results reveal an important yet disconcerting pattern. No matter what the level of genetic potential, childhood disadvantage produces a sub-optimal combination of traits and preferences in adulthood that favors social immobility (Falk et al., 2021). For instance, respondents with a low polygenic score for educational attainment who experienced childhood disadvantage have lower levels of education, lower cognitive ability, are less risk-averse, and less patient than their genetically equivalent counterparts from more advantaged childhood environments. This combination of mental process and internal preferences are important for addictive behaviors such as smoking and gambling, which combine intertemporal elements with risk considerations (Ida and Goto 2009; Sutter, Kocher, Glätzle-Rützler & Trautmann,

2013). Of course, this joint consideration of endowments is important, as no economic decision involves only one preference or cognitive aspect (Dohmen et al., 2018; Falk, Kosse, Pinger, Schildberg-Hörisch & Deckers, 2021). When childhood disadvantage is combined with high genetic potential, the story is equally discouraging. Here, higher levels of educational attainment and phenotypic cognitive skills, are coupled with more risk-aversion and less patience than their genetically equivalent counterparts from more advantaged childhood environments. Whilst it may be advantageous to couple low cognitive skills with cautious tendencies (Dohmen et al., 2010), optimal investment decisions likely require a combination of a willingness to take calculated risks and high cognition (Grinblatt, Keloharju & Linnainmaa, 2011, 2012). Taken together, disadvantaged environments appear to limit the ability of individuals to capitalize on genetic potential, suggesting that environmental inequality can reinforce patterns of social immobility. These findings also underscore the limitations of genetic determinism, illustrating how genetic predispositions are not fixed but interact dynamically with environmental factors, reinforcing the importance of nurturing environments for maximizing potential and policies to reduce childhood poverty. Indeed, findings from childhood intervention programs, illustrate that enriching early environments raises noncognitive skills that promote success in social and economic life (Borghans et al., 2008; Cunha et al., 2006; Heckman et al., 2010).

The results also provide an opening for new perspectives on the mechanisms through which gene-by-environment interactions shape economic preferences. Specifically, in disadvantaged childhood environments, cognitive resources may be directed toward detecting environmental threats, uncertainty, and resource scarcity—leading to greater risk aversion (Belsky & Pluess, 2009; Obradović et al., 2010; Obradović, 2012), as suggested by Adaptive Calibration Models (Del Giudice et al., 2011). In such contexts, risk-avoidant behavior may therefore reflect an adaptive response to environmental unpredictability and perceived threat

(Ellis et al., 2009; Frankenhuis & de Weerth, 2013; Haushofer & Fehr, 2014; Kidd, Palmeri & Aslin, 2013). Additionally, the finding that individuals from disadvantaged backgrounds exhibit similarly short-term (impatient) planning horizons across all PGS EA levels suggests that early adversity may impose pervasive constraints—such as scarcity-driven cognitive load or learned present-biased behaviors—that limit the expression of cognitive potential in decision-making (Mani et al., 2013; Rutter, 2006). This pattern is consistent with the Experiential Canalization Framework (Blair & Raver, 2012; Gottlieb, 1991), which posits that environmental adversity can interfere with the developmental processes and pathways that typically enable genetic predispositions to influence behavior—effectively muting trait differences in disadvantaged contexts. In contrast, in more advantaged childhood environments, the absence of such scarcity constraints appears to allow individuals with higher PGS EA to express economic preferences that are more aligned with findings from the cognitive ability literature (Dohmen et al., 2010). Specifically, these individuals may be better able to override the impulsive, affect-driven tendencies of System 1 (Benjamin, Brown & Shapiro, 2013; McClure et al., 2004; Shiv & Fedorikhin, 1999; Shiv et al., 2005; Stanovich & West, 2008), and have reduced tendencies toward narrow bracketing—approaches that otherwise fosters risk aversion and myopic preferences (Tversky & Kahneman, 1981; Read, Loewenstein & Rabin, 1999).

Importantly, our findings may also help explain why the literature on early-life stress and economic preferences literature has been unable to provide a consensus perspective. The first perspective comes from the life history approach from evolutionary biology (Roff, 1992) whereby early-life experience, such as exposure to stress or resource scarcity, can shape and individual life history strategy and their preferences for risk and time. Here, individuals who perceive their environment as more uncertain and unpredictable may be likely to adopt a ‘fast’ life strategy, which prioritizes rapid reproduction and survival in unpredictable environment—

associated with higher risk-taking and focus on immediate rewards—whilst a 'slow' strategy focuses on long-term survival and reproduction in stable environments—associated with a higher value placed on future rewards and more risk aversion. A contrasting view comes from the uncertainty management perspective (Amir & Jordan, 2017; Amir, Jordan & Rand, 2018) where early-life stress predicts a dangerous future environment, shaping economic preferences in a way that facilitates the management of uncertainty. These strategies include a focus on immediate outcomes—as future outcomes are more uncertain—and risk-aversion—to avoid potential losses. Empirical evidence has found support for both the life histories (Birn, Roeber & Pollak, 2017; Hill, Jenkins & Farmer, 2008; Simpson et al., 2012; Wu et al., 2020; i.e., early-life stress predicting impatience and risk-taking behaviour) and uncertainty perspectives (Amir, Jordan & Rand, 2018; i.e., early-life stress predicting impatience and risk-averse behaviour). Our results provide a unifying boundary condition (i.e., the specific situations or context where a theories prediction is accurate). Simply, our results are consistent with the life history perspective (uncertainty management perspective), but only when childhood disadvantage is accompanied by a lower (higher) genetic propensity toward educational success.

Our results also speak to the endogeneity debate within the literature on cognitive ability and economic preferences (Dohmen et al., 2010, 2018). For instance, economic preferences like impatience may shape cognitive investments (e.g., through investments in education). Alternatively, cognitive ability may affect economic preferences, perhaps because cognition governs the degree to which the emotional system, System 1—risk averse and myopic—can be overridden by the deliberative system, System 2—risk neutral and patient (Stanovich & West, 2008). Moreover, the level of cognitive skills and economic preferences could conceivably be set simultaneously, perhaps because evolutionary pressures might have created a tendency for low cognitive skills to be coupled with cautious tendencies (Dohmen et al., 2010). Whilst our results cannot be interpreted as causal, given that genotypes are randomly

assigned from parents to an offspring at conception, the estimated effect of the PGS for educational attainment should be unsusceptible to the problem of reverse causality (Madole & Harden, 2023) and less susceptible to socio-economic and life-style confounders (Smith et al., 2007). To address causality, future work can extend our analysis by leveraging within-sibling variation in polygenic scores or by controlling for the genes of the respondent's natal parents.

A further limitation of our study is that the polygenic score for educational attainment (PGS EA) captures not only genetic influences related to cognitive ability, but also overlaps with non-cognitive traits, such as personality. This conflation raises concerns about whether observed associations attributed to cognitive factors may, in fact, reflect other psychological or behavioral dispositions. In our analysis, we do control for genetic variants associated with the Big Five personality traits. We also replicate our main findings using both the polygenic score for IQ and phenotypic IQ. This however does not eliminate the possibility that other non-cognitive traits suspected to be important for both educational success and performance on cognitive tests—such as grit, motivation, curiosity, self-control, or perseverance (Demange et al., 2021; Heckman, 2006; Heckman & Kautz, 2012; Moffitt et al., 2011; Richardson, Abraham & Bond, 2012; Rimfeld et al., 2016)—are driving the observed effects, highlighting the need for further research that can more precisely disentangle cognitive from non-cognitive genetic influences.

Lastly, whilst not within the scope of this paper, our findings make way for a wider perspective of whether these data patterns of cognitive ability on economic preferences might translate to developing and developed countries. Indeed, Chowdhury, Sutter, and Zimmermann (2022) provide evidence from a developing country of a negative correlation between IQ and patience, as well as a negative influence of mothers' education on risk tolerance. Their interpretation is that patience and risk taking are strategies that payoff in resource-rich and stable environments (Bonin et al., 2007; Cadena & Keys, 2015; DellaVigna & Paserman, 2005;

Dohmen & Falk, 2011; Golsteyn, Grönqvist & Lindahl, 2014; Ventura, 2003), whereas in resource-scarce and threatening environments, impatience and risk aversion may be more adaptive for survival (Amir & Jordan, 2017; Amir, Jordan & Rand, 2018). In both contexts, cognitive abilities are deployed to identify the strategies most likely to yield success.

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Supplemental Material

Section A

Table S1. Summary statistics

	Min.	Max.	Mean/Frequency	
			No disadv.	Disadv.
Panel A – Experimental sample				
Age	50	75	63.41	65.6
Male	0	1	0.45	0.48
PGS EA	4206.28	4279.9	4249.58	4247.75
Risk aversion (B-EG):				
Lottery A	0	1	0.35	0.40
Lottery B	0	1	0.20	0.18
Lottery C	0	1	0.13	0.11
Lottery D	0	1	0.09	0.09
Lottery E	0	1	0.12	0.08
Lottery F	0	1	0.11	0.13
Risk aversion (LA):				
Lottery A	0	1	0.43	0.49
Lottery B	0	1	0.27	0.19
Lottery C	0	1	0.09	0.07
Lottery D	0	1	0.06	0.05
Lottery E	0	1	0.05	0.07
Lottery F	0	1	0.10	0.12
Discount rate (1 month):				

Payoff Alternative 1	0	1	0.5	0.44
Payoff Alternative 2	0	1	0.13	0.16
Payoff Alternative 3	0	1	0.12	0.12
Payoff Alternative 4	0	1	0.03	0.05
Payoff Alternative 5	0	1	0.05	0.05
Payoff Alternative 6	0	1	0.02	0.01
Never switched	0	1	0.15	0.16
Discount rate (2 months):				
Payoff Alternative 1	0	1	0.34	0.31
Payoff Alternative 2	0	1	0.19	0.16
Payoff Alternative 3	0	1	0.14	0.17
Payoff Alternative 4	0	1	0.08	0.06
Payoff Alternative 5	0	1	0.07	0.09
Payoff Alternative 6	0	1	0.01	0.02
Never switched	0	1	0.17	0.18
Observations			405	219
Panel B – Survey-based sample				
Age	31	90	63.87	65.5
Male	0	1	0.46	0.45
PGS EA	4196.97	4296.68	4249.29	4247.37
Planning horizon:				
does not plan/plans day to day	0	1	0.09	0.10
the next few weeks	0	1	0.10	0.14
the next few months	0	1	0.13	0.15
the next year	0	1	0.16	0.17

the next few years	0	1	0.24	0.22
the next 5-10 years	0	1	0.20	0.17
longer than 10 years	0	1	0.08	0.06
Observations			7402	4119

Table S2. Gene-Environment Interaction and Risk Preferences – Valid Responses to all four Childhood Disadvantage Dimensions

Regression:	1	2	3	4
Dependent variable:	Risk aversion (B-EG)	Risk aversion (LA)	Risk aversion (LA-binary)	Risk aversion (LA-binary)
Estimator:	Interval	Ordered Logit	Logit	Logit
$\beta 1$ PGS EA	-0.430***	-0.407***	-0.605***	-0.551***
	[-3.012]	[-3.669]	[-4.163]	[-3.390]
$\beta 2$ PGS EA × Disadv.	0.912***	0.771***	0.689***	0.531*
	[3.717]	[3.648]	[2.740]	[1.841]
$\beta 1 + \beta 2$	0.482**	0.364**	0.084	-0.021
	[2.400]	[2.043]	[0.410]	[-0.086]
Controls	Yes	Yes	Yes	Yes
Observations	432	432	432	432

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets. Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regression 4, we also include a series of dummy variables representing respondents' choice in the risk aversion lottery (B-EG). In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S3. Gene-Environment Interaction and Time Preferences – Valid Responses to all four Childhood Disadvantage Dimensions

Regression:	1	2	3	4
Dependent variable:	Discount rate (1 month)	Discount rate (2 months)	Planning horizon	Planning horizon (binary)
Estimator:	Interval	Interval	Ordered Logit	Logit
$\beta 1$ PGS EA	-0.017*	-0.012***	0.241***	0.248***
	[-1.737]	[-3.283]	[7.065]	[6.472]
$\beta 2$ PGS EA × Disadv.	-0.006	0.010*	-0.174***	-0.189***
	[-0.370]	[1.705]	[-3.390]	[-3.283]
$\beta 1 + \beta 2$	-0.023*	-0.002	0.068*	0.059
	[-1.718]	[-0.324]	[1.763]	[1.390]
Controls	Yes	Yes	Yes	Yes
Observations	432	432	6770	6770

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets (cluster-robust standard errors in Regressions 3 and 4). Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regressions 1 and 2 we also include a dichotomous control variable that equals one if the subject made an inconsistent choice in the MPL (Multiple Price List) and zero otherwise. In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S4. Gene-Environment Interaction and Risk Preferences – Missingness

Regression:	1	2	3	4
Dependent variable:	Risk aversion (B-EG)	Risk aversion (LA)	Risk aversion (LA-binary)	Risk aversion (LA-binary)
Estimator:	Interval	Ordered Logit	Logit	Logit
$\beta 1$ PGS EA	-0.373***	-0.352***	-0.540***	-0.471***
	[-3.040]	[-4.072]	[-4.648]	[-3.847]
$\beta 2$ PGS EA × Disadv.	0.800***	0.717***	0.704***	0.565**
	[3.620]	[4.098]	[3.443]	[2.475]
$\beta 1 + \beta 2$	0.428**	0.365**	0.164	0.094
	[2.320]	[2.408]	[0.975]	[0.487]
Controls	Yes	Yes	Yes	Yes
Missingness indicator	Yes	Yes	Yes	Yes
Observations	624	624	624	624

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets. Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. Missingness indicator is a dichotomous variable that takes on the value of one if the respondent did not have valid responses to all four disadvantage dimensions and zero otherwise. In Regression 4, we also include a series of dummy variables representing respondents' choice in the risk aversion lottery (B-EG). In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S5. Gene-Environment Interaction and Time Preferences – Missingness

Regression:	1	2	3	4
Dependent variable:	Discount rate	Discount rate	Planning horizon	Planning horizon
	(1 month)	(2 months)		(binary)
Estimator:	Interval	Interval	Ordered Logit	Logit
$\beta 1$ PGS EA	-0.021**	-0.011***	0.184***	0.179***
	[-2.505]	[-3.672]	[7.275]	[6.531]
$\beta 2$ PGS EA × Disadv.	-0.006	0.007	-0.122***	-0.135***
	[-0.431]	[1.376]	[-3.037]	[-2.982]
$\beta 1 + \beta 2$	-0.027**	-0.004	0.061*	0.044
	[-2.362]	[-0.987]	[1.942]	[1.221]
Controls	Yes	Yes	Yes	Yes
Missingness indicator	Yes	Yes	Yes	Yes
Observations	624	624	11521	11521

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets (cluster-robust standard errors in Regressions 3 and 4). Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regressions 1 and 2 we also include a dichotomous control variable that equals one if the subject made an inconsistent choice in the MPL (Multiple Price List) and zero otherwise. Missingness indicator is a dichotomous variable that takes on the value of one if the respondent did not have valid responses to all four disadvantage dimensions and zero otherwise. In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage'). *

p<0.10, ** p<0.05, *** p<0.01.

Table S6. Gene-Environment Interaction and Risk Preferences – Separate Dimensions of Childhood Disadvantage

Regression:	1	2	3	4
Dependent variable:	Risk aversion (B-EG)	Risk aversion (LA)	Risk aversion (LA-binary)	Risk aversion (LA-binary)
Estimator:	Interval	Ordered Logit	Logit	Logit
Panel A. Human capital disadvantage - neither parent with education above the compulsory level				
$\beta 1$ PGS EA	-0.284*	-0.260**	-0.436***	-0.441***
	[-1.809]	[-2.362]	[-3.019]	[-2.862]
$\beta 2$ PGS EA × Disadv.	0.292	0.229	0.260	0.266
	[1.363]	[1.500]	[1.321]	[1.227]
$\beta 1 + \beta 2$	0.008	-0.031	-0.175	-0.175
	[0.055]	[-0.297]	[-1.304]	[-1.146]
Panel B. Resource disadvantage - main caregiver low status occupation				
$\beta 1$ PGS EA	-0.213*	-0.248***	-0.354***	-0.335***
	[-1.786]	[-3.080]	[-3.318]	[-2.940]
$\beta 2$ PGS EA × Disadv.	0.334	0.515***	0.334	0.382
	[1.294]	[2.805]	[1.470]	[1.449]
$\beta 1 + \beta 2$	0.121	0.267	-0.020	0.048
	[0.528]	[1.621]	[-0.101]	[0.201]

Panel C. Instability disadvantage - parents being permanently separated or divorced				
$\beta 1$ PGS EA	-0.231**	-0.209**	-0.373***	-0.318***
	[-1.993]	[-2.523]	[-3.454]	[-2.785]
$\beta 2$ PGS EA × Disadv.	0.735**	0.415*	0.657**	0.442
	[2.391]	[1.750]	[2.146]	[1.168]
$\beta 1 + \beta 2$	0.504*	0.206	0.284	0.124
	[1.771]	[0.929]	[0.992]	[0.345]
Panel D. Resource disadvantage - childhood home no central heating or hot water				
$\beta 1$ PGS EA	-0.222*	-0.228***	-0.448***	-0.415***
	[-1.863]	[-2.713]	[-4.090]	[-3.634]
$\beta 2$ PGS EA × Disadv.	0.465*	0.345*	0.517**	0.467*
	[1.703]	[1.796]	[2.193]	[1.903]
$\beta 1 + \beta 2$	0.243***	0.117***	0.069***	0.052***
	[0.989]	[0.674]	[0.331]	[0.238]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	624	624

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets. Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regression 4, we also include a series of dummy variables representing respondents' choice in the risk aversion lottery (B-EG). In all regressions, all parameters are

allowed to differ across groups (i.e., ‘no disadvantage’ and ‘disadvantage’).

* p<0.10, ** p<0.05, *** p<0.01.

Table S7. Gene-Environment Interaction and Time Preferences – Separate Dimensions of Childhood Disadvantage

Regression:	1	2	3	4
Dependent variable:	Discount rate (1 month)	Discount rate (2 months)	Planning horizon	Planning horizon (binary)
Estimator:	Interval	Interval	Ordered Logit	Logit
Panel A. Human capital disadvantage - neither parent with education above the compulsory level				
$\beta 1$ PGS EA	-0.026**	-0.012***	0.188***	0.180***
	[-2.569]	[-3.047]	[7.095]	[6.250]
$\beta 2$ PGS EA × Disadv.	0.004	0.004	-0.086**	-0.084*
	[0.320]	[0.745]	[-2.182]	[-1.913]
$\beta 1 + \beta 2$	-0.022**	-0.008**	0.102***	0.096***
	[-2.445]	[-2.313]	[3.443]	[2.885]
Panel B. Resource disadvantage - main caregiver low status occupation				
$\beta 1$ PGS EA	-0.025***	-0.011***	0.174***	0.184***
	[-3.287]	[-3.773]	[7.082]	[6.932]
$\beta 2$ PGS EA × Disadv.	0.006	0.008	-0.086**	-0.140***
	[0.431]	[1.406]	[-2.118]	[-3.031]
$\beta 1 + \beta 2$	-0.019	-0.003	0.088***	0.045

	[-1.507]	[-0.700]	[2.707]	[1.182]
Panel C. Instability disadvantage - parents being permanently separated or divorced				
$\beta 1$ PGS EA	-0.020***	-0.009***	0.164***	0.146***
	[-2.887]	[-3.500]	[7.630]	[6.210]
$\beta 2$ PGS EA × Disadv.	-0.029	0.001	-0.064	0.009
	[-1.388]	[0.161]	[-1.164]	[0.142]
$\beta 1 + \beta 2$	-0.049**	-0.008	0.101**	0.155***
	[-2.508]	[-1.054]	[1.993]	[2.740]
Panel D. Resource disadvantage - childhood home no central heating or hot water				
$\beta 1$ PGS EA	-0.021***	-0.008***	0.185***	0.177***
	[-2.832]	[-2.619]	[7.810]	[6.878]
$\beta 2$ PGS EA × Disadv.	-0.017	-0.008	-0.100**	-0.102**
	[-1.049]	[-1.361]	[-2.353]	[-2.149]
$\beta 1 + \beta 2$	-0.038***	-0.015***	0.085**	0.075*
	[-2.630]	[-3.095]	[2.389]	[1.885]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	624	624

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets (cluster-robust standard errors in Regressions 3 and 4). Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regressions 1

and 2 we also include a dichotomous control variable that equals one if the subject made an inconsistent choice in the MPL (Multiple Price List) and zero otherwise. In all regressions, all parameters are allowed to differ across groups (i.e., ‘no disadvantage’ and ‘disadvantage’).

* $p<0.10$, ** $p<0.05$, *** $p<0.01$.

Table S8. Gene-Environment Interaction and Risk Preferences – Continuous Childhood Disadvantage Measure

Regression:	1	2	3	4
Dependent variable:	Risk aversion (B-EG)	Risk aversion (LA)	Risk aversion (LA-binary)	Risk aversion (LA-binary)
Estimator:	Interval	Ordered Logit	Logit	Logit
$\beta 1$ PGS EA	-0.644***	-0.562***	-0.730***	-0.632***
	[-3.815]	[-4.245]	[-4.499]	[-3.493]
$\beta 2$ PGS EA × Disadv.	0.429***	0.360***	0.337***	0.268**
	[3.613]	[3.828]	[3.175]	[2.165]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	624	624

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets. Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regression 4, we also include a series of dummy variables representing respondents' choice in the risk aversion lottery (B-EG). In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S9. Gene-Environment Interaction and Time Preferences – Continuous Childhood Disadvantage Measure

Regression:	1	2	3	4
Dependent variable:	Discount rate (1 month)	Discount rate (2 months)	Planning horizon	Planning horizon (binary)
Estimator:	Interval	Interval	Ordered Logit	Logit
$\beta 1$ PGS EA	-0.022*	-0.013***	0.214***	0.211***
	[-1.947]	[-3.031]	[6.919]	[6.248]
$\beta 2$ PGS EA × Disadv.	-0.001	0.003	-0.057***	-0.061***
	[-0.187]	[1.075]	[-3.019]	[-2.889]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	11521	11521

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets (cluster-robust standard errors in Regressions 3 and 4). Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regressions 1 and 2 we also include a dichotomous control variable that equals one if the subject made an inconsistent choice in the MPL (Multiple Price List) and zero otherwise. In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S10. Gene-Environment Interaction and Risk Preferences – Parental Bonding Instrument

Regression:	1	2	3	4
Dependent variable:	Risk aversion (B-EG)	Risk aversion (LA)	Risk aversion (LA-binary)	Risk aversion (LA-binary)
Estimator:	Interval	Ordered Logit	Logit	Logit
$\beta 1$ PGS EA	-0.373***	-0.354***	-0.541***	-0.477***
	[-3.070]	[-4.116]	[-4.684]	[-3.914]
$\beta 2$ PGS EA × Disadv.	0.796***	0.725***	0.716***	0.586**
	[3.625]	[4.215]	[3.517]	[2.557]
$\beta 1 + \beta 2$	0.423**	0.370**	0.175	0.109
	[2.310]	[2.501]	[1.044]	[0.563]
Controls	Yes	Yes	Yes	Yes
Parental Bonding Instrument	Yes	Yes	Yes	Yes
Observations	624	624	624	624

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets. Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regression 4, we also include a series of dummy variables representing respondents' choice in the risk aversion lottery (B-EG). Parental Bonding Instrument is a continuous measure of respondents' retrospective experiences of their parents' parenting style before age 16. In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S11. Gene-Environment Interaction and Time Preferences – Parental Bonding Instrument

Regression:	1	2	3	4
Dependent variable:	Discount rate (1 month)	Discount rate (2 months)	Planning horizon	Planning horizon (binary)
Estimator:	Interval	Interval	Ordered Logit	Logit
$\beta 1$ PGS EA	-0.021**	-0.011***	0.185***	0.181***
	[-2.473]	[-3.618]	[7.340]	[6.581]
$\beta 2$ PGS EA × Disadv.	-0.007	0.007	-0.121***	-0.136***
	[-0.504]	[1.348]	[-2.996]	[-2.997]
$\beta 1 + \beta 2$	-0.028**	-0.004	0.064**	0.045
	[-2.427]	[-1.000]	[2.040]	[1.241]
Controls	Yes	Yes	Yes	Yes
Parental Bonding Instrument	Yes	Yes	Yes	Yes
Observations	624	624	11521	11521

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets (cluster-robust standard errors in Regressions 3 and 4). Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regressions 1 and 2 we also include a dichotomous control variable that equals one if the subject made an inconsistent choice in the MPL (Multiple Price List) and zero otherwise. Parental Bonding Instrument is a continuous measure of respondents' retrospective experiences of their parents'

parenting style before age 16. In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage'). * $p<0.10$, ** $p<0.05$, *** $p<0.01$.

Table S12. Gene-Environment Interaction and Risk Preferences – PGS Big 5

Regression:	1	2	3	4
Dependent variable:	Risk aversion (B-EG)	Risk aversion (LA)	Risk aversion (LA-binary)	Risk aversion (LA-binary)
Estimator:	Interval	Ordered Logit	Logit	Logit
$\beta 1$ PGS EA	-0.394***	-0.354***	-0.573***	-0.532***
	[-3.215]	[-4.064]	[-4.735]	[-4.047]
$\beta 2$ PGS EA × Disadv.	0.797***	0.740***	0.657***	0.512**
	[3.473]	[4.057]	[3.064]	[2.036]
$\beta 1 + \beta 2$	0.404**	0.385**	0.084	-0.020
	[2.075]	[2.423]	[0.476]	[-0.093]
Controls	Yes	Yes	Yes	Yes
PGS Big 5	Yes	Yes	Yes	Yes
Observations	624	624	624	624

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets. Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regression 4, we also include a series of dummy variables representing respondents' choice in the risk aversion lottery (B-EG). PGS Big 5 includes the polygenic scores for the Big Five personality traits. In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S13. Gene-Environment Interaction and Time Preferences – PGS Big 5

Regression:	1	2	3	4
Dependent variable:	Discount rate (1 month)	Discount rate (2 months)	Planning horizon	Planning horizon (binary)
Estimator:	Interval	Interval	Ordered Logit	Logit
$\beta 1$ PGS EA	-0.022***	-0.012***	0.199***	0.198***
	[-2.711]	[-3.824]	[7.857]	[7.226]
$\beta 2$ PGS EA × Disadv.	-0.004	0.008	-0.129***	-0.146***
	[-0.253]	[1.478]	[-3.178]	[-3.195]
$\beta 1 + \beta 2$	-0.026**	-0.004	0.070**	0.052
	[-2.309]	[-0.953]	[2.207]	[1.434]
Controls	Yes	Yes	Yes	Yes
PGS Big 5	Yes	Yes	Yes	Yes
Observations	624	624	11521	11521

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets (cluster-robust standard errors in Regressions 3 and 4). Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regressions 1 and 2 we also include a dichotomous control variable that equals one if the subject made an inconsistent choice in the MPL (Multiple Price List) and zero otherwise. PGS Big 5 includes the polygenic scores for the Big Five personality traits. In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage'). * p<0.10, ** p<0.05, *** p<0.01.

Table S14. Correlation of Polygenic Scores – Experimental Sample

Variables	(1)	(2)	(3)	(4)	(5)
(1) PGS EA					
(2) PGS Agreeableness	0.135***				
(3) PGS Conscientiousness	0.010	-0.030			
(4) PGS Extraversion	0.010	0.115***	0.040		
(5) PGS Neuroticism	-0.088**	-0.181***	0.000	-0.304***	
(6) PGS Openness	0.050	0.020	-0.150***	0.040	0.030

Note. Sample of 624 individuals.

Table S15. Correlation of Polygenic Scores – Survey Sample

Variables	(1)	(2)	(3)	(4)	(5)
(1) PGS EA					
(2) PGS Agreeableness	0.116***				
(3) PGS Conscientiousness	-0.010	-0.034***			
(4) PGS Extraversion	-0.010	0.138***	-0.000		
(5) PGS Neuroticism	-0.102***	-0.177***	0.000	-0.265***	
(6) PGS Openness	0.010	0.020**	-0.165***	0.010	0.010

Note. Sample of 11521 observations from 5881 individuals.

Table S16. Gene-Environment Interaction and Risk Preferences – PGS IQ

Regression:	1	2	3	4
Dependent variable:	Risk aversion (B-EG)	Risk aversion (LA)	Risk aversion (LA-binary)	Risk aversion (LA-binary)
Estimator:	Interval	Ordered Logit	Logit	Logit
$\beta 1$ PGS IQ	-0.290**	-0.231***	-0.203*	-0.133
	[-2.341]	[-2.778]	[-1.807]	[-1.044]
$\beta 2$ PGS IQ × Disadv.	0.533**	0.379**	0.370*	0.245
	[2.385]	[2.145]	[1.811]	[1.155]
$\beta 1 + \beta 2$	0.243	0.148	0.168	0.112
	[1.308]	[0.952]	[0.981]	[0.661]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	624	624

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets. Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regression 4, we also include a series of dummy variables representing respondents' choice in the risk aversion lottery (B-EG). In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S17. Gene-Environment Interaction and Time Preferences – PGS IQ

Regression:	1	2	3	4
Dependent variable:	Discount rate (1 month)	Discount rate (2 months)	Planning horizon	Planning horizon (binary)
Estimator:	Interval	Interval	Ordered Logit	Logit
$\beta 1$ PGS IQ	-0.020**	-0.005*	0.137***	0.150***
	[-2.421]	[-1.778]	[5.677]	[5.570]
$\beta 2$ PGS IQ × Disadv.	0.018	0.005	-0.082**	-0.092**
	[1.326]	[1.121]	[-2.078]	[-2.107]
$\beta 1 + \beta 2$	-0.002	0.000	0.055*	0.058*
	[-0.218]	[0.075]	[1.746]	[1.693]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	11521	11521

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets (cluster-robust standard errors in Regressions 3 and 4). Controls include age (in linear and quadratic form); sex; and the first 10 principal components of the respondent's SNPs. In Regressions 1 and 2 we also include a dichotomous control variable that equals one if the subject made an inconsistent choice in the MPL (Multiple Price List) and zero otherwise. In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S18. Phenotypic IQ-Environment Interaction and Risk Preferences

Regression:	1	2	3	4
Dependent variable:	Risk aversion (B-EG)	Risk aversion (LA)	Risk aversion (LA-binary)	Risk aversion (LA-binary)
Estimator:	Interval	Ordered Logit	Logit	Logit
$\beta 1$ Phenotypic IQ	-0.757***	-0.428***	-0.131	0.079
	[-3.064]	[-2.703]	[-0.636]	[0.339]
$\beta 2$ Phenotypic IQ × Disadv.	1.063***	0.801***	0.687**	0.397
	[2.873]	[2.962]	[2.199]	[1.157]
$\beta 1 + \beta 2$	0.305	0.373*	0.556**	0.476*
	[1.108]	[1.704]	[2.364]	[1.886]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	624	624

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets. Controls include age (in linear and quadratic form) and sex. In Regression 4, we also include a series of dummy variables representing respondents' choice in the risk aversion lottery (B-EG). In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S19. Phenotypic IQ-Environment Interaction and Time Preferences

Regression:	1	2	3	4
Dependent variable:	Discount rate (1 month)	Discount rate (2 months)	Planning horizon	Planning horizon (binary)
Estimator:	Interval	Interval	Ordered Logit	Logit
$\beta 1$ Phenotypic IQ	-0.059***	-0.021***	0.509***	0.527***
	[-3.809]	[-3.664]	[14.771]	[13.147]
$\beta 2$ Phenotypic IQ × Disadv.	-0.006	-0.002	-0.207***	-0.275***
	[-0.256]	[-0.266]	[-3.476]	[-4.227]
$\beta 1 + \beta 2$	-0.065***	-0.023***	0.302***	0.251***
	[-3.901]	[-3.453]	[6.168]	[4.885]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	11521	11521

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets (cluster-robust standard errors in Regressions 3 and 4). Controls include age (in linear and quadratic form) and sex. In Regressions 1 and 2 we also include a dichotomous control variable that equals one if the subject made an inconsistent choice in the MPL (Multiple Price List) and zero otherwise. In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S20. Gene-Environment Interaction and Risk Preferences – Including Wealth, Income, and Educational Attainment

Regression:	1	2	3	4
Dependent variable:	Risk aversion (B-EG)	Risk aversion (LA)	Risk aversion (LA-binary)	Risk aversion (LA-binary)
Estimator:	Interval	Ordered Logit	Logit	Logit
$\beta 1$ PGS EA	-0.358***	-0.340***	-0.599***	-0.541***
	[-2.737]	[-3.678]	[-4.783]	[-4.087]
$\beta 2$ PGS EA × Disadv.	0.826***	0.713***	0.755***	0.635**
	[3.619]	[3.952]	[3.372]	[2.570]
$\beta 1 + \beta 2$	0.468**	0.373**	0.156	0.093
	[2.497]	[2.417]	[0.841]	[0.448]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	624	624

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets. Controls include age (in linear and quadratic form); sex; the first 10 principal components of the respondent's SNPs; logarithm of net total household wealth (the sum of savings, investments, physical wealth, and housing wealth after financial debt and mortgage debt has been subtracted); logarithm of annual household total income; and the highest reported level of educational attainment. In Regression 4, we also include a series of dummy variables representing respondents' choice in the risk aversion lottery (B-EG). In all regressions, all parameters are allowed to differ across groups (i.e., 'no disadvantage' and 'disadvantage').

* p<0.10, ** p<0.05, *** p<0.01.

Table S21. Gene-Environment Interaction and Time Preferences – Including Wealth, Income, and Educational Attainment

Regression:	1	2	3	4
Dependent variable:	Discount rate (1 month)	Discount rate (2 months)	Planning horizon	Planning horizon (binary)
Estimator:	Interval	Interval	Ordered Logit	Logit
$\beta 1$ PGS EA	-0.012	-0.009***	0.059**	0.058**
	[-1.429]	[-2.895]	[2.288]	[2.005]
$\beta 2$ PGS EA × Disadv.	-0.009	0.008	-0.077*	-0.100**
	[-0.671]	[1.490]	[-1.860]	[-2.123]
$\beta 1 + \beta 2$	-.022**	-.002	-0.018	-0.042
	[-1.991]	[-0.372]	[-0.546]	[-1.132]
Controls	Yes	Yes	Yes	Yes
Observations	624	624	11472	11472

Note. Unadjusted coefficients, t-statistics using robust standard errors in brackets (cluster-robust standard errors in Regressions 3 and 4). Controls include age (in linear and quadratic form); sex; the first 10 principal components of the respondent's SNPs; logarithm of net total household wealth (the sum of savings, investments, physical wealth, and housing wealth after financial debt and mortgage debt has been subtracted); logarithm of annual household total income; and the highest reported level of educational attainment. In Regressions 1 and 2 we also include a dichotomous control variable that equals one if the subject made an inconsistent choice in the MPL (Multiple Price List) and zero otherwise. In all regressions, all parameters

are allowed to differ across groups (i.e., ‘no disadvantage’ and ‘disadvantage’).

* p<0.10, ** p<0.05, *** p<0.01.

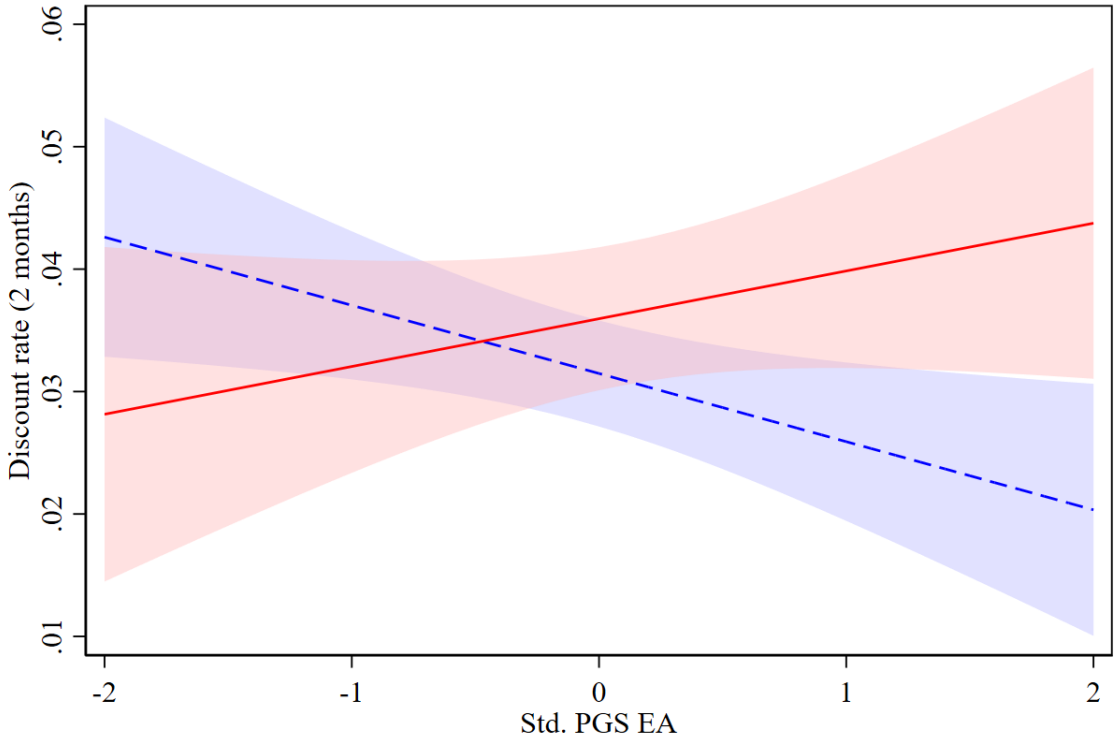

Fig. S1. Gene-Environment Interaction and Time Preferences

Note. Dashed (solid) lines are for the 'no disadvantage' ('disadvantage') group, Shaded areas represent 95% confidence intervals.

Section B

The English Longitudinal Study of Ageing (ELSA) includes a wide array of tasks aimed at evaluating various aspects of cognitive functioning, such as memory, executive processing, and fundamental cognitive capabilities.

The first task focused on temporal orientation—conducted in all waves (i.e., Waves 1 to 9)—where respondents were asked to report the current day, month, year, and day of the week. The final score is based on a simple count of number of correct items.

The second task involved a word recall assessment designed to measure verbal memory. Participants in all Waves were read a list of 10 words and immediately afterwards were asked to recall as many words as possible, in any order. They were then prompted again, later in the interview, to recall as many words as possible, in any order. The total number of correctly recalled words across both the immediate and delayed implementation were combined into a single score.

A third task assessed prospective memory and was conducted during Waves 1 to 5 and again in Wave 8. Participants were given two tasks to remember: one involved—when handed a clipboard and a pencil by the interviewer—writing their initials on the top left-hand corner of piece of paper attached to the clipboard, and another required them to remind the interviewer to record the time at the end of the session. Performance was evaluated based on task completion and the need for prompts.

Numeracy skills were assessed through a fourth task, administered in Waves 1, 4, and to refreshment samples in Waves 6 to 9. This task involved solving up to five arithmetic problems of increasing difficulty, depending on performance on initial questions. Specifically, based on performance on the first three items, participants can get two additional (more difficult) questions or one additional (simpler) question. Sample items included estimating the

original cost of a car sold at two-thirds of its price and calculating an equal lottery payout. Scoring reflected the number of accurate responses.

Executive function was examined through a counting backwards task (Wave 7 to 9), which asked participants to count backwards quickly from 20. Scoring was binary: either participants counted correctly from 19 to 10 or from 20 to 11, or they did not.

Executive function was also assessed through an object-naming assessment, conducted in Waves 7 to 9, where participants were required to name five common objects or people in response to prompts. The type of questions asked included: "*What do you call the kind of prickly plant that grows in the desert?*" and "*Who is the reigning monarch now?*". Scores reflected the number of correct identifications.

A Serial 7s subtraction task (also in Waves 7 to 9) tested numerical working memory and attention by asking respondents to sequentially subtract 7 from 100, up to five times. The final score captured the number of correct subtractions.

Semantic verbal fluency was tested using an animal-naming task (all waves except Wave 6), where participants had one minute to list as many animals as possible. The score represented the count of unique, valid responses.

A further task measuring processing speed and accuracy was a letter cancellation task (Waves 1 to 5), in which participants scanned lines of letters and marked all instances of the letter's "P" and "W." Participants were asked to start at the top left-hand corner of the list and work along each line of the list sequentially, as if they were reading a page. Participants were asked to work as quickly and as accurately as they could, until the computer says stop. At this point, participants were asked to underline the last letter on which they finished. Two scores were generated: one for the number of P's and W's correctly marked and another for the number of missed letters up to the marked stopping point.

A fluid intelligence task was added in Waves 6, 8, and 9 using a number series exercise. Respondents were presented with number sequences containing a missing element and asked to identify the correct missing number. Based on their performance in an initial set of three sequences, they received a second set of problems, with difficulty tailored to their earlier performance. Scoring incorporated both accuracy and problem difficulty.

Finally, literacy was assessed through a reading comprehension task included in Waves 2 and 5, and with refreshment samples in Waves 6 to 9. Participants were asked to read a medication label and answer four comprehension questions, such as identifying usage restrictions or appropriate conditions for consulting a doctor. The final score counted the number of correct answers.

A composite measure representing general cognitive ability was constructed from twelve individual cognitive test scores. This was calculated by standardizing and combining respondents' average scores—as individuals may take each cognitive task more than once—across the tasks. The general cognitive ability score includes all available non-missing items for each individual. As a result, observations were retained as long as at least one component variable was observed, rather than being dropped due to any single missing value. The resulting composite demonstrated acceptable internal reliability, with a Cronbach's alpha of 0.78 and an average inter-item correlation of 0.23.