

The “Depression Trap”: Genetic Risk, Labor Supply, and the Case for Precision Psychiatry*

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Abstract

We analyze how genetic risk for depression influences the joint dynamics of labor supply, health, disability, and treatment decisions using a life-cycle model and longitudinal data. We find that genetic risk for depression substantially reduces earnings and employment, worsens health, and increases long-term sickness and disability. We identify the “depression trap”, a self-reinforcing cycle of depression and non-employment, as a key mechanism linking genetic risk to persistent labor market exit. We evaluate a precision psychiatry framework that reduces treatment barriers for high risk individuals, and find that it improves outcomes at zero net fiscal cost, primarily by reducing disability-related non-employment.

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1 Introduction

While it has long been known that genetics plays a role in mental health disorders, it has only recently become possible to use genome sequencing data to predict *who* will suffer from depression (Murray et al., 2021). Given the role of mental health in productivity and labor supply, can genetic risk predict who will be employed and how much they will earn? As the UK National Health Services moves towards universal newborn genotyping (Davies, 2025), will a precision psychiatry framework, integrating genomics data in the provision of mental health care, be cost-effective in improving mental health and employment outcomes?

This paper studies how genetic risk for depression affects the joint dynamics of labor supply, health and treatment decisions of working age men in the UK. It documents new facts about the effects of genetic risk for depression on earnings, employment and health. It then estimates a structural life-cycle model of labor supply and mental health investment that matches these empirical patterns. Finally, it uses the model to evaluate the efficiency of improving access to mental health treatment for high-risk individuals.

We exploit within and between family variation in genetics to estimate the effects of a Polygenic Index (PGI) for depressive symptoms on health and labor market outcomes. We show that men with above-median risk for depression are 12 p.p. more likely to suffer from depression and earn 0.17 s.d. less labor income. This “genetic penalty” in earnings is partially as a result of lower educational and occupational attainment, but disability-related non-employment is a key driver. It explains one-third of the income penalty, and is in turn driven by worse contemporaneous physical and mental health. Drawing on evidence from the literature, we explain this pattern through the mechanism of a “depression trap”: a self-reinforcing cycle where poor mental health reduces labor supply, which in turn worsens mental health. We show that high-risk men are twice as likely to enter the trap and half as likely to leave it.

Our model incorporates this mechanism, allowing for a two-way interaction between employment and health. We embed labor supply and mental health treatment choices in a consumption savings model with physical and mental health dynamics, with observed (genetic) and unobserved heterogeneity in productivity and health. Mental and physical health transitions depend on employment, and health in turns affects productivity and the cost of working. Poor health also increases the benefit receipt associated with unemployment¹, further reducing the incentive to work.

We estimate the model using a multi-step estimation procedure with Understanding Society data. First, we identify the discrete distribution of latent individual heterogeneity by applying the clustering approach of Bonhomme et al. (2022) to outcomes that have been residualized on genetic risk for depression. This ensures that our estimated types capture unobserved productivity and health traits that are distinct from the biological predisposition measured by the PGI. Second, physical and mental health dynamics are estimated by maximizing the likelihood of health transitions conditional on individual characteristics.

¹We do not include a distinct “disabled” employment state in our model. Instead, benefits for individuals out of the labor force depend directly on their health status. Because disabled individuals predominantly fall into severe mental or physical health states, the benefit levels for these groups implicitly capture disability payments.

Third, we estimate parameters relating to preferences and wage offers using the Method of Simulated Moments (MSM).

Our model delivers several new findings. First, we find that the dynamic effect of genetic risk for depression is important in explaining life-cycle outcomes, and that these effects are not captured by other initial characteristics. We isolate the dynamic effects of genetics by simulating the same individuals with varying genetic risk for depression for a 10-year period. This simulation generates substantial differences in labor market and health outcomes, largely consistent with reduced-form evidence for the lifetime effect of genetic risk. This highlights genetics as an important source of dynamic heterogeneity, that captures not only current health or labor market status, but also future trajectories.

Second, we find that genetic risk amplifies adverse shocks, driving the persistence of the “depression trap”, the joint state of severe mental illness and non-employment. Following a severe mental health shock, high-risk individuals take longer to recover and are twice as likely to remain in the trap after four years. Crucially, while health shocks eventually narrow, extreme negative wage shocks induce highly persistent divergence. For high-risk individuals, economic setbacks trigger a self-reinforcing feedback loop where non-employment exacerbates mental illness, transforming transient productivity losses into long-term scarring.

Third, we find that improving access to mental health via subsidies can improve mental health and employment outcomes at zero net cost to the government. While universal subsidies generate higher aggregate uptake and clinical gains for any given level of spending, targeted interventions produce stronger employment responses, especially in the post-COVID environment. We demonstrate that offering modest financial incentives (£100) to encourage therapy attendance among genetically at-risk individuals — analogous to existing clinical engagement tools used in cancer screening — generates net-positive fiscal returns, while yielding larger mental health improvements than cost-neutral universal alternatives. These results suggest that precision psychiatry integrating genomics data can cost-effectively improve mental health and employment outcomes.

Our paper makes three primary contributions, bridging the gap between the health, labor and genetic psychiatry literatures. First, we establish that genetic predisposition for depression is a fundamental driver of life-cycle inequality in health and employment outcomes, providing a biologically grounded measure of heterogeneity that captures latent vulnerabilities not captured by traditional observables. Second, we formalize and quantify the “depression trap”, demonstrating how genetic risk amplifies the persistence of adverse shocks through a self-reinforcing feedback loop between non-employment and mental illness. Finally, we provide the first structural evaluation of precision medicine incorporating genomics data. We show that targeted psychotherapy subsidies can generate a substantial “fiscal dividend” by disrupting long-term disability more effectively than universal alternatives, particularly in the post-COVID landscape.

The paper is organized as follows. Section 2 discusses how this paper relates to the existing literature. Section 3 presents the data and our reduced-form evidence. Section 4 presents our model and Section 5 explains our multi-step identification and estimation procedure. Section 6 presents various counterfactual

analyses and Section 7 concludes. Various robustness checks and additional results are presented in Appendices A-F.

2 Related Literature

This paper bridges the gap between the genetic psychiatry and structural labor and health literatures by incorporating genetics, health, labor supply and endogenous treatment in a rich life-cycle labor supply model.

This paper is most closely related to the burgeoning literature incorporating mental health in structural models of labor supply. However, our model is unique in its focus on the two-way relationship between the employment and mental health and in incorporating genetics as a source of ex-ante heterogeneity. Jolivet and Postel-Vinay (2025) feature a two-way relationship between work and mental health in model of occupation choice. Stressful occupations are more costly for workers with poor mental health and feature worse health transition probabilities. Cronin et al. (2024) use a rich model of mental health treatment and labor supply to explore why psychotherapy is under-utilized relative to antidepressants despite having larger treatment effects. Abramson et al. (2024) incorporate mental health in a consumption-savings model with endogenous treatment. They focus on how the pessimism associated with poor mental health can induce individuals to under-invest in risky assets and pursue less challenging occupations, thus negatively impacting asset accumulation. These three papers allow mental health to affect labor supply, but only Jolivet and Postel-Vinay (2025) models the effect of employment on mental health. Given that their focus is on occupation choice, they do not model the persistently non-employed or mental health treatment. To model persistent non-employment in our model, we jointly model physical and mental health dynamics, and incorporate disability benefits which vary with health status.

This paper also relates to the larger literature on physical health and labor supply. A branch of the literature focuses on the effect of physical health and retirement decisions (French, 2005; French and Jones, 2011) or on long-term earnings and welfare (Hosseini et al., 2021; De Nardi et al., 2024). This literature generally does not consider the feedback effects of employment on health. Exceptions include Salvati (2025), who incorporates a bidirectional relationship between work and health for women near retirement. Models of physical health and labor supply typically do not consider mental health dynamics. Sauer et al. (2025) is an exception, modelling joint physical and mental health dynamics, as well as labor supply for women. Interestingly, they find negative effects of employment on health, in contrast with evidence from RCTs, exogenous plant closures and longitudinal analyses (Picchio and Ubaldi, 2024; Hussam et al., 2022; Sterud et al., 2025). However, the focus of these studies is on women, and findings do suggest that the protective mental and physical health effects of employment are larger for men (Picchio and Ubaldi, 2024).

We also contribute to the ‘Genoeconomics’ literature, which links molecular genetic data to economic surveys to study the interaction between genetic and environmental effects. We show how genetic risk for depression predicts economic outcomes and demonstrate how it reinforces health and economic shocks.

Biroli et al. (2022) provides a useful introduction to this literature. The literature relies on Genome-Wide Association Studies (GWAS) to estimate the association between genetic variants and a trait of interest in a large population (Uffelmann et al., 2021). These estimated associations can then be used to generate individual-level predictors of a trait of interest, called Polygenic Scores (PGS) or Indices (PGI). Thus far, this literature has almost entirely focussed on educational attainment (EA) as a trait of interest (Okbay et al., 2022), which has been shown to be a strong predictor of asset accumulation (Barth et al., 2020), labor market outcomes (Papageorge and Thom, 2020), as well as parental investment and skill formation (Bolt et al., 2025). There have been limited attempts to incorporate genetics in structural models, and PGIs other than EA have thus far received very little attention in economics. Barth et al. (2022) incorporate the EA PGI in a consumption-savings model of asset accumulation. Houmark et al. (2024), Bolt et al. (2025) and Agostinelli and Weingarten (2025) model how genetics affect parental investment and child skill formation, with the latter incorporating genetic predictors of ADHD. Jeong et al. (2025) use reduced-form approaches to explore a genetic predictor of Alzheimer's, while De Nardi et al. (2024) briefly discusses how PGIs for educational attainment, BMI, smoking and longevity predict physical health. While Amin et al. (2019) explore gene-environment associations for a depressive symptoms PGI with education as the outcome, ours is the first study to link it to labor market outcomes, mental health treatment and dynamics, and to explore the role of targeted interventions.

This paper also bridges the gap between labor economics and precision psychiatry literatures. We demonstrate that integrating genomics data in the provision of mental health care is clinically effective, and that the resulting improvements in employment and health make such interventions fiscally self-sustaining. The fiscal returns generated by an increase in labor supply and reduced benefit expenditure finance the costs of subsidising treatment. While there is a large, long-established body of evidence for the effectiveness of psychotherapy (Baranov et al., 2020; Cuijpers et al., 2014), recent research has highlighted how PGIs can be used to identify high-risk patients and eventually develop personalized interventions (Torkamani et al., 2018). To date, there remains little evidence of how treatments could be *adapted* depending on an individual's genetics, but a recent RCT suggests that the treatment effect of an Cognitive Behavioural Therapy intervention for depressed patients does not appear to vary depending on their depression PGI (Bäckman et al., 2025). This suggests that high-risk individuals are not 'predetermined' to be depressed, but that treatments can be effective. The take-up of psychotherapy, however remains low. Cronin et al. (2024) consider various mechanisms and conclude that the under-utilization of psychotherapy is not primarily due to financial costs or time constraints, but likely relates to stigma or difficulties opening up to a stranger. To overcome these constraints Breza et al. (2026) conduct an RCT in which they offer financial incentives to attend therapy, which increases take-up only among symptomatic patients, consistent with the broader evidence that financial incentives can increase mental health treatment engagement (Khazanov et al., 2022). This finding is consistent with our model results, and we even find that paying high-risk individuals £100 to attend publicly-funded psychotherapy generates net positive tax revenue.

3 Empirical Insights

Our empirical analysis proceeds in two stages. First, we present the data and establish the reduced-form relationship between a genetic predisposition for depression and labor market earnings, which we term the “Genetic Penalty”. We explore the key drivers through a nested mediation framework, sequentially including potential mediators to gain insight into the mechanisms at play, such as education, occupation and labor supply decisions. Second, we draw on evidence from the existing literature to provide evidence for the “depression trap” mechanism: a self-reinforcing cycle whereby poor mental health reduces labor supply, which in turn worsens mental health. We show that these dynamics are present in our data and reinforced by genetic risk for depression.

3.1 Data and Sample Construction

The primary data source for this study is the UK Household Longitudinal Study (UKHLS), a large-scale nationally representative panel from 1991 to 2023. The UKHLS is uniquely suited for this analysis as it provides molecular genetic data linked to an annual longitudinal panel of health and labor market trajectories. Our genomic sample consists of 9,220 genotyped individuals, which we restrict to a primary estimation sample of working-age men (aged 25-55). We use this sample of $\approx 2,000$ individuals for our reduced-form analysis, but for our model estimation, we extend the sample to include individuals aged 65 to 85. This yields a longitudinal panel of about 2,000 – 3,000 individuals and 20 – 40,000 individual-wave observations. For our mental health measure, we use the “caseness” scoring of the GHQ-12², which counts the presence of relevant symptoms. This is a well-validated measure, commonly used to screen for depression (Wojujutari et al., 2024). Following the psychiatric literature, we use 2 and 4 as cut-offs to define mild depressive symptoms and major depressive disorder (Anjara et al., 2020).

3.2 Genetics Data

To measure genetic predisposition to depressive symptoms, we use a polygenic index (PGI). A PGI is an individual-level predictor constructed from genotype data. It can be interpreted as a weighted sum of genetic variants. Let $SNP_{i,j} \in \{0, 1, 2\}$ denote the number of effect alleles carried by individual i at variant j , where the allele may increase or decrease depression risk. The PGI aggregates many such variants with small individual effects into a single summary measure:

$$PGI_i = \sum_{j=1}^J \beta_j^{GWAS} SNP_{i,j}. \quad (1)$$

²This scoring method converts responses into a binary indicator. It determines whether an individual’s symptoms meet the clinical threshold for a probable mental health disorder.

The weights β_j^{GWAS} are estimated in an external GWAS discovery sample that is separate from our UKHLS target sample. In the GWAS, the association between each variant j and the trait of interest is estimated using regressions of the form

$$Y_k = \beta_j^{GWAS} SNP_{k,j} + \gamma \mathbf{X}_k + u_{k,j}, \quad (2)$$

where k indexes individuals in the discovery sample, Y_k is depressive symptoms, and $\mathbf{X}_k$ includes standard GWAS controls (sex, age polynomials, and genetic principal components) to adjust for population structure.

In our main analysis, we use weights from [Baselmans et al. \(2019\)](#), which studies depressive symptoms. The GWAS discovery sample does not overlap with the UKHLS target sample. As additional controls in some specifications, we also use PGIs constructed from GWAS of educational attainment ([Okbay et al., 2022](#)) and self-reported health ([Harris et al., 2017](#)). Both the discovery and target samples are restricted to individuals of European-like White ancestry. We impose this restriction because PGIs do not transport well across ancestry groups and because non-European GWAS sample sizes remain more limited. See [Price et al. \(2006\)](#) for discussion of population structure and related concerns. Details on the genetic data and PGI measurement are provided in [Appendix A](#).

3.3 Descriptive Statistics

Table 1 provides descriptive statistics for our primary sample of working-age men, stratified by whether their mental health polygenic index (MH PGI) is below or above the sample median. We find stark disparities in health, labor market, and socioeconomic outcomes between these genetic cohorts. Men with a low MH PGI, representing those with a high genetic liability for depression, are 11.1 percentage points more likely to suffer from clinical depression and 5 percentage points more likely to be in poor physical health. These health deficits correspond to significantly higher rates of labor market exit; the high-risk group is 3.4 percentage points more likely to report being unable to work due to disability and 5.2 percentage points less likely to be employed.

Table 1: Descriptive Statistics and Tests of Differences by Mental Health PGI

Variable	Low MH PGI		High MH PGI		Diff. (<i>p</i> -value)
	Mean	(Std. Dev.)	Mean	(Std. Dev.)	
<i>Health Outcomes</i>					
Depression (Prob.)	0.23	(0.42)	0.12	(0.32)	0.00
Poor Physical Health (Prob.)	0.18	(0.38)	0.13	(0.33)	0.00
Disability (Prob.)	0.05	(0.22)	0.02	(0.13)	0.00
Therapy (Prob.)	0.02	(0.15)	0.01	(0.11)	0.00
<i>Economic Outcomes</i>					
Annual Pay (£)	33,888	(18,942)	36,493	(21,051)	0.00
High Occ. (Prob.)	0.19	(0.39)	0.24	(0.43)	0.00
Total Assets (£)	18,825	(51,119)	24,342	(101,924)	0.10
Employed (Prob.)	0.89	(0.32)	0.94	(0.24)	0.00
Benefit Receipt (Prob.)	0.28	(0.45)	0.22	(0.42)	0.00
Annual Benefits (£)	3,197	(3,455)	2,765	(3,041)	0.00
<i>Demographics & Human Capital</i>					
College (Prob.)	0.27	(0.45)	0.32	(0.47)	0.00
Father High Occ. (Prob.)	0.18	(0.38)	0.20	(0.40)	0.31
Mother's Education (Yrs)	10.90	(1.92)	11.00	(2.01)	0.26
Age	42.81	(8.33)	42.74	(8.19)	0.55
Observations	9,770		10,574		

Notes: Standard deviations are reported in parentheses. The final column reports *p*-values from a two-sided *t*-test for the difference in means between individuals with an MH PGI below (Low) and above (High) the median. Parental education is mapped to years based on highest qualification. High occupation indicators are binary for managerial roles (SOC90 < 20). Sample sizes for parental variables are smaller ($N \approx 1,500$ –2,500) due to retrospective reporting. All variables are for UKHLS men aged 25–55.

These genetic predispositions translate into substantial economic penalties throughout the life cycle. Conditional on employment, men at high genetic risk for depression earn £2,605 less annually and are 5.1 percentage points less likely to hold a high-status managerial or professional occupation. The cumulative effect of these earnings and employment gaps is reflected in a large, though noisily estimated, wealth disparity, with the high-risk group holding £5,517 fewer assets on average ($p = 0.104$). Furthermore, high-risk individuals rely more heavily on the social safety net; they are 5.9 percentage points more likely to receive government benefits and, conditional on receipt, receive an additional £432 per year.

Importantly, these disparities appear to emerge early in working-age life cycle and are not driven by simple demographic confounding. While high-risk men are 4.6 percentage points less likely to hold a college degree, we find no significant differences in age across the two groups ($p = 0.545$). Furthermore, we observe a high degree of balance in parental socioeconomic background; neither the probability of having a father in a high-status occupation ($p = 0.309$) nor maternal years of education ($p = 0.256$) differ significantly by genetic risk. This suggests that the MH PGI is not merely proxying for dynastic socioeconomic advantage, but rather appears to have a direct genetic effect on health and economic trajectories (while this effect may operate through environmental channels). We provide more direct support for this view in Appendix C, where we include parental socioeconomic background as controls and perform within-family analyses for our key outcomes of interest and find consistent estimates.

3.4 The “Genetic Penalty”

To identify the relationship between genetic risk for depression and subsequent labor market outcomes, we estimate the following specification for individual i at time t :

$$Y_{i,t} = \alpha + \beta \text{HighMH}_i + \mathbf{X}'_{i,t} \gamma + \mathbf{M}'_{i,t} \theta + \epsilon_{i,t} \quad (3)$$

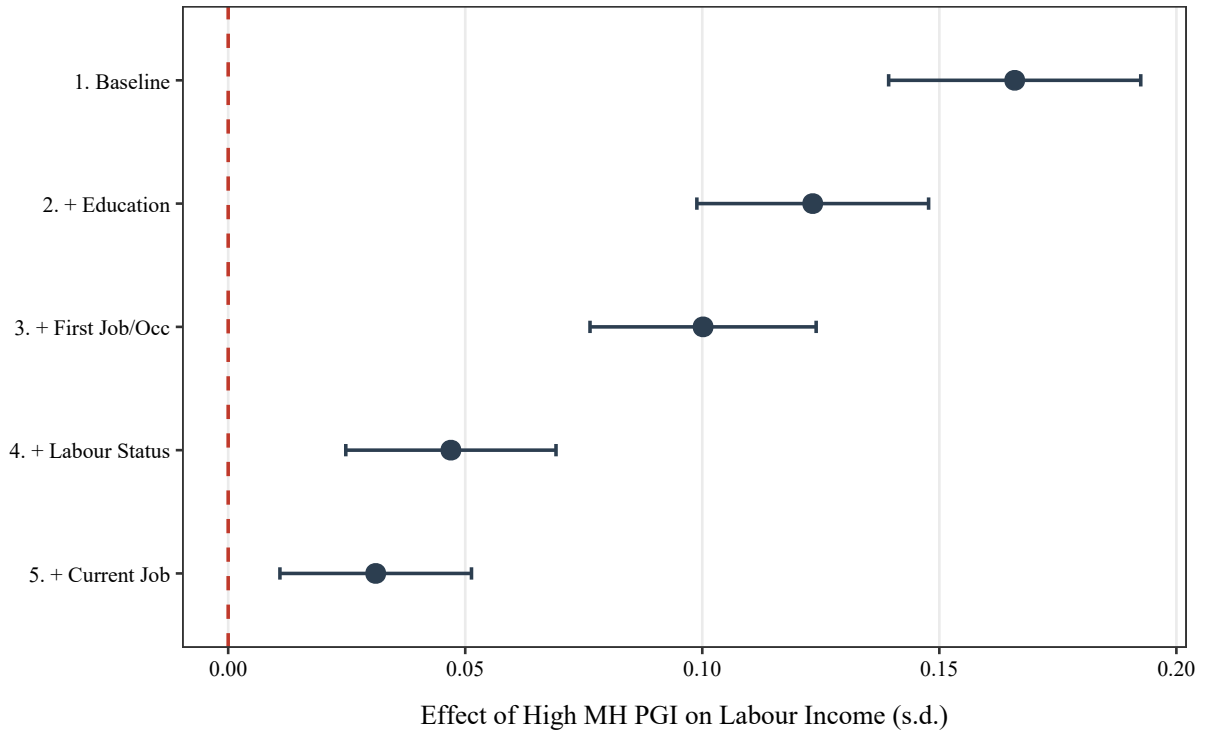
where $Y_{i,t}$ denotes standardized gross labor income. The primary parameter of interest, β , captures the earnings penalty associated with a *High Mental Health PGI*, defined as an index above the sample median. The vector $\mathbf{X}_{i,t}$ includes baseline exogenous controls: the first 10 principal components of the genomic data to account for population stratification, alongside fixed effects for age, wave, and interview year.

To investigate the structural mechanisms through which this genetic risk translates into lower earnings, we sequentially introduce a vector of life-cycle mediators, $\mathbf{M}_{i,t}$. These are incorporated in four chronological blocks: (1) educational attainment; (2) initial occupational choice; (3) contemporaneous labor force status; and (4) current occupation fixed effects. The attenuation of β across these specifications, presented in Figure 1, allows us to decompose the total genetic effect into components mediated by human capital accumulation, occupational sorting, and labor supply margins.

Our identification strategy assumes that the PGI reflects a biological vulnerability orthogonal to the household environment. In Appendix C, we subject this assumption to several rigorous tests. Specifically, we include controls for parental socioeconomic background (education and occupation), as well as the polygenic indices for educational attainment (EA) and physical health. Furthermore, we estimate family fixed-effects models on a subsample and obtain broadly consistent results. This is line with results from Okbay et al. (2022), who show that, in a much larger sample, within-family estimates of the effect of depression and wellbeing-related PGIs are generally consistent with between-family estimates. This is in stark contrast to the widely used EA PGI, which appears to largely capture socioeconomic background, and attenuates by about one-half when used to predict within-family differences.

We report the effect of a high MH PGI on earnings, and how this effect attenuates when sequentially incorporating potential channels, in Figure 1. Overall, men with a high MH PGI earn about 0.17 s.d. higher gross labor income. Education and first occupation appear to play a substantial role, together explaining about one-third of the association. This suggests that genetic risk for depression is associated with reduced human capital accumulation early in life. In our genetics sample, we have very few observations in early adulthood, so we cannot be sure that poor mental health drives this association. It does, however, seem plausible. Adolescence and early adulthood, a crucial period in determining educational and occupational attainment, is a high-risk period for the development of depression: about 50 % of all lifetime cases of Major Depressive Disorder start by age 25. (Kessler et al., 2007). There is also ample evidence to suggest that depression in these periods reduces the probability of high school or college completion and leads to assortment into lower status careers (Fletcher, 2010; Esch et al., 2014).

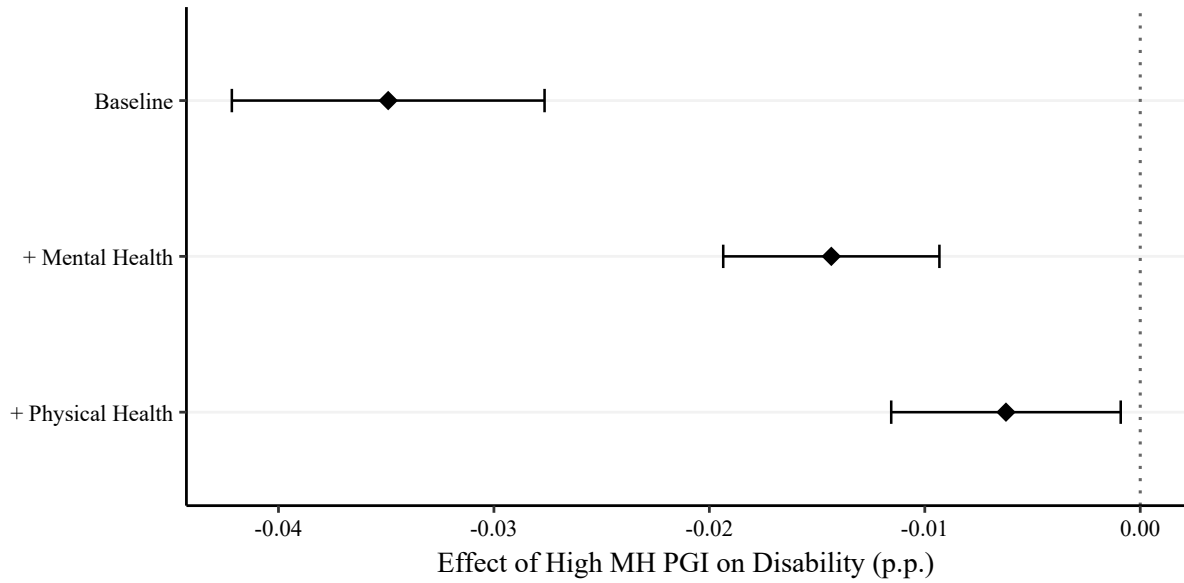
Figure 1: Depression Risk and Earnings: Key Channels



Notes: This figure plots the estimated coefficient on an indicator for being above-median in the mental health polygenic index (High MH PGI) across five regression specifications that sequentially add potential mediating channels. Specification (1) includes age, survey wave, and year fixed effects, as well as the first 10 genetic principal components. Specifications (2)–(5) add, in turn, education, first occupation, labor force status, and current occupation. Attenuation in the High MH PGI coefficient across specifications indicates how much of the baseline earnings difference is accounted for by human capital and by extensive and intensive labor supply margins. Standard errors are clustered at the household level.

The primary driver of the “genetic penalty” in earnings is current labor force status, which explains approximately one-third of the total association. Decomposing this effect (Figure 15), we find it is driven almost exclusively by high-risk men reporting long-term sickness or disability rather than shorter term unemployment. Using ‘Disability’ status as an outcome (Figure 2), we find that men with high MH PGI are 3.5 percentage points more likely to be disabled. This association is largely mediated by current health: the effect attenuates to 1.5 p.p. when controlling for mental health, and further drops by another percentage point once physical health is included. The fact that current health explains nearly the entire association suggests a mechanistic pathway: genetic predisposition for depression manifests in poor contemporaneous health, which subsequently triggers a withdrawal from the labor force. Drawing on evidence from the literature, we argue that this reinforces the “depression trap” - a self-reinforcing cycle of persistent poor mental health and non-employment.

Figure 2: Genetic Risk and Disability: Health Mechanisms



Notes: This figure plots the estimated coefficient on an indicator for being above-median in the mental health polygenic index (High MH PGI) across specifications that sequentially add observed health measures. Each point is from an OLS regression with a binary disability indicator as the dependent variable. The baseline specification includes age, survey wave, and interview-year fixed effects, as well as the first 10 genetic principal components. Subsequent specifications add contemporaneous mental health (GHQ-12) and physical health (SF-12 Physical Component Summary, PCS). Attenuation of the High MH PGI coefficient across specifications indicates how much of the baseline association with disability is captured by observed mental and physical health. Standard errors are clustered at the household level. The sample includes UKHLS men aged 25–55.

3.5 The “Depression Trap”

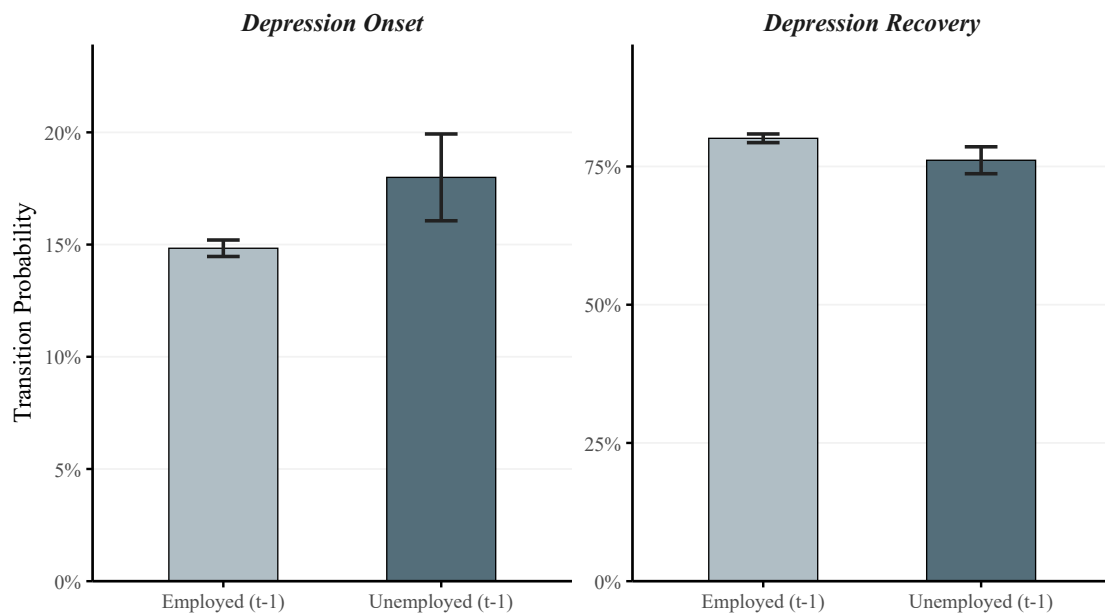
First, we provide an overview of evidence from the literature demonstrating a bidirectional relationship between mental health and labor supply: depression induces a reduction in labor supply, which in turn worsens the prospects of recovery. We present descriptive results in our data consistent with this evidence, which creates a potentially persistent “depression trap”, characterized by poor mental health and non-employment. We demonstrate that men with a low mental health PGI are more likely to enter this trap and less likely to leave it, suggesting that genetic risk for depression reinforces this mechanism.

How does employment affect mental health? There is strong evidence to suggest employment has a positive impact on mental health. A meta-analysis of longitudinal studies reports an effect size of 0.19 SMD, even after controlling for income (so employment improves mental health by 0.19 s.d.). Recent RCT evidence suggests that the non-pecuniary benefits of employment improve mental health by 0.23 SMD (Hussam et al., 2022), while a meta-analysis of studies exploiting exogenous variation in employment, such as plant closures, find estimates closer to 0.1 SMD for men (Picchio and Ubaldi, 2024). Historical estimates, widely cited but more likely to be confounded by the effects of mental health on employment, suggests a larger effect size of 0.51 SMD (Paul and Moser, 2009). To put these numbers in context, meta-analyses of mental health treatments suggest an effect size of 0.3 SMD for antidepressants (Cipriani et al., 2018) and 0.6 SMD for psychotherapy (Hofmann et al., 2012).

Modini et al. (2016) provide an overview of the key mechanisms by which employment fosters better mental health. Work meets important psychosocial needs in societies where employment is considered

to be a social norm (Waddell and Burton, 2006). discuss how work can provide social and emotional support, status and acceptance within society, a sense of purpose, opportunities for personal development and greater optimism (Fossey and Harvey, 2010). Unemployment also has a negative impact on physical health (Picchio and Ubaldi, 2024), which is in turn a risk factor for depression (Marx et al., 2023).

Figure 3: Mental Health Transition Probabilities by Employment Status



Notes: This figure reports predicted mental health transition probabilities from a Mundlak-style correlated random effects (CRE) logit model. Probabilities are average marginal effects evaluated over the sample covariate distribution. The left panel shows depression onset (healthy at $t - 1$ to depressed at t). The right panel shows depression recovery (depressed at $t - 1$ to healthy at t). The model controls for unobserved individual heterogeneity using within-person time averages of all time-varying regressors, and includes lagged depression, lagged employment, lagged earnings, household composition, and a quadratic in age. Survey-wave and year fixed effects are included. Error bars denote 95% confidence intervals computed using the delta method, with standard errors clustered at the individual level.

In Figure 3, we present depression transition dynamics predicted using a random effects model estimated in our data. Controlling for time-invariant characteristics and income, the unemployed³ face a significantly higher probability of experiencing depression and are less likely to recover. They are 3 p.p. more likely to develop depression and 4 p.p. less likely to recover. The effect size estimated in this model corresponds to 0.17 SMD, broadly in line with estimates from the existing literature. The model estimates are displayed in Table 6 in Appendix B.

There is robust evidence from the existing literature that depression contributes to a significant reduction in labor supply. Ettner et al. (1997) provided early estimates suggesting that a psychiatric diagnosis reduces male employment by 13 percentage points. More recently, Frijters et al. (2014) utilized the death of a close friend as an instrument for mental health shocks, estimating that a one standard deviation (s.d.) worsening of mental health reduces employment by 30 p.p. Longitudinal panel estimates

³Unemployed here includes 'Long-term sick'.

further suggest that the onset of a mental health disorder leads to a 7–18 p.p. increase in the probability of transitioning out of employment, while recovery is associated with a 6–21 p.p. increase in the probability of returning to work (Mitra and Jones, 2017).

Experimental evidence regarding the effect of psychotherapy on employment corroborates these observational findings. Lund et al. (2024) meta-analyzed RCTs from low- and middle-income countries, finding that treating depression improves employment and job search intensity by 0.16 s.d. Similarly, Smith et al. (2025) conducted an RCT in Norway comparing psychotherapy to “treatment as usual” for patients with anxiety and depression. They estimated a 0.2 s.d. improvement in employment (a 5 p.p. increase). Rzepnicka et al. (2025) estimate an event study specification in the UK and find a small, but very persistent increase in employment of 1.5 p.p., even seven years after receiving therapy. This likely represents lower bounds on the total impact of depression *per se*, as psychotherapy interventions typically correspond to a 0.6 s.d. improvement in symptoms rather than a complete remission.

Table 2: Clinical Criteria for Major Depressive Disorder (D-SIG E CAPS)

Letter	Symptom	Clinical Description
D	Depressed Mood	Feeling sad, empty, hopeless, or tearful; core gateway symptom.
S	Sleep	Insomnia or hypersomnia nearly every day.
I	Interest	Anhedonia: markedly diminished interest or pleasure in all activities.
G	Guilt	Feelings of worthlessness or excessive/inappropriate guilt.
E	Energy	Fatigue or loss of energy nearly every day.
C	Concentration	Diminished ability to think, concentrate, or indecisiveness.
A	Appetite	Significant weight change or increase/decrease in appetite.
P	Psychomotor	Observable agitation or retardation (slowing down).
S	Suicidality	Recurrent thoughts of death or suicidal ideation.

Notes: Diagnosis of Major Depressive Disorder (MDD) requires five or more symptoms to be present during the same 2-week period, with at least one symptom being either **Depressed Mood (D)** or **Loss of Interest (I)**. This mnemonic summarizes the symptom domains used in the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2022).

To understand why depression acts as such a potent barrier to labor supply, it is necessary to examine its clinical manifestations. The diagnostic criteria for Major Depressive Disorder (MDD) center on a persistently depressed mood or anhedonia. A widely used clinical mnemonic for the associated symptoms is *D-SIG E CAPS* (American Psychiatric Association, 2022), detailed in Table 2.

Mechanisms of Labor Supply Reduction

The symptoms of depression are fundamentally in-conducive to productive labor, acting through three primary channels: reduced marginal productivity, increased dis utility of effort, and the erosion of physical health capital.

Productivity and the Wage Channel: Depression impairs productivity directly through cognitive dysfunction. Clinical symptoms such as diminished concentration and excessive guilt capture attention and facilitate rumination, distorting an individual’s beliefs regarding their own abilities (Ridley et al., 2020; Gotlib and Joormann, 2010). These impairments manifest as “presentee-ism”, where individuals remain physically present at work but exhibit diminished output per hour (Mall et al., 2015). In a

structural framework, this cognitive tax acts as a negative shock to human capital, reducing the wage offer. Furthermore, external barriers such as employer stigma and discrimination exacerbate this channel, as others may underestimate the productivity of individuals with mental health conditions, hindering job retention and wage growth (Ridley et al., 2020).

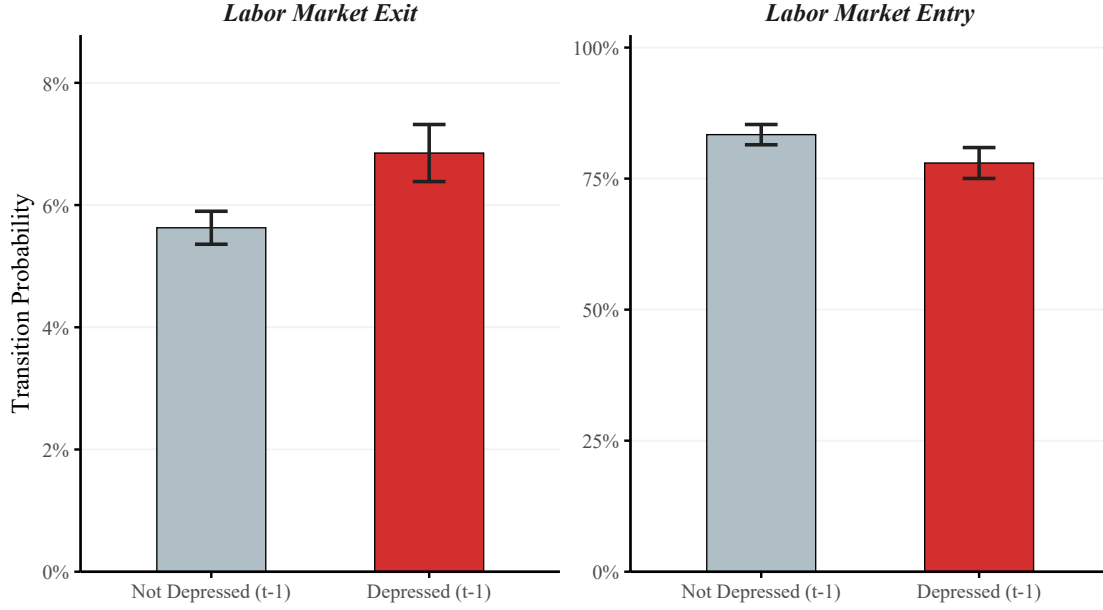
Disutility and the Participation Channel: Beyond hourly productivity, depression reduces the overall desire and ability to engage in the labor market. Symptoms like fatigue and anhedonia significantly increase the non-pecuniary cost of effort. This is compounded by a “pessimism about the returns to effort” (De Quidt and Haushofer, 2016), where depressed individuals struggle with delayed gratification and reduced motivation. This psychological state makes job search activities—which require high upfront effort for uncertain future rewards—particularly taxing. Functional impairments like sleep disturbances and psychomotor retardation further restrict the ability to adhere to rigid work schedules, often precipitating the transitions into long-term sickness or disability observed in our descriptive analysis.

Physical Health Spillovers: Finally, depression reduces labor supply through its negative impact on physical health. Clinical evidence suggests that chronic mental distress triggers systemic inflammatory responses (Dantzer et al., 2008), which accelerate physiological ageing and cardiovascular decay (Prince et al., 2007). This “weathering” effect leads to higher rates of chronic physical conditions (Katon, 2011), which in turn impairs productivity and labor supply (Currie and Madrian, 1999).

We incorporate these pathways in our structural model, allowing mental health health to affect not only contemporaneous wage offers and the cost of working but also an individual’s physical health trajectory. This multi-channel approach is essential for capturing the “depression trap,” where initial psychological shocks lead to a self-reinforcing cycle of non-employment and physical-mental co-morbidity.

In Figure 4, we present the estimated labor market transition probabilities conditional on mental health status. The descriptive evidence reveals a substantial employment penalty: individuals suffering from depression are 1 percentage point more likely to transition out of employment and 5 percentage points less likely to return to work compared to those who are not depressed. These estimates correspond to a 0.27 standard deviation (SMD) effect size for the impact of depression on employment participation. While these transition probabilities provide a stark illustration of the labor market barriers associated with mental distress, they do not capture the contemporaneous effects of depression on employment, which we will estimate in our structural model.

Figure 4: Labor Market Transition Probabilities by Mental Health Status

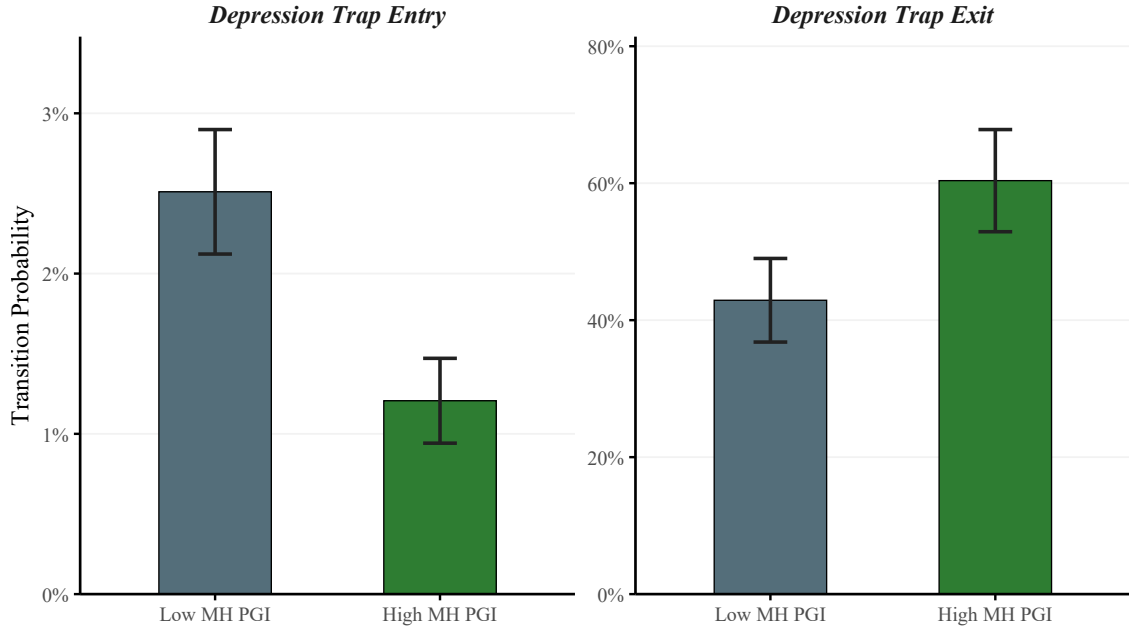


Notes: This figure reports predicted labor market transition probabilities from a Mundlak-style correlated random effects (CRE) logit model. Probabilities are average marginal effects evaluated over the sample covariate distribution. The left panel shows labor market exit (employed at $t - 1$ to unemployed at t). The right panel shows labor market entry (unemployed at $t - 1$ to employed at t). The model controls for unobserved individual heterogeneity using within-person time averages of time-varying regressors, and includes lagged employment, current mental health status, lagged income, household composition, and a quadratic in age. Survey-wave and year fixed effects are included. Error bars denote 95% confidence intervals computed using the delta method, with standard errors clustered at the individual level.

The bidirectional relationship between employment and mental health suggests the potential for a “depression trap”: a self-reinforcing state of non-employment and psychological distress. We characterize this trap as the joint state of being out of work and having mild or severe depression. A central question is whether genetic predisposition for depression exacerbates the persistence of this state. In Figure 5, we present the transition probabilities into and out of this state for men above and below the median MH PGI.

These results indicate that genetic risk significantly influences the duration of depressive episodes. Men with high genetic risk (low MH PGI) are 2 percentage points more likely to enter the trap in any given year. More strikingly, they are approximately 20 percentage points less likely to exit this state once they have entered it. This suggests that the “depression trap” is remarkably more persistent for high-risk individuals, a phenomenon driven by the dual disadvantage of slower mental health recovery and diminished labor market re-entry rates. To untangle the mechanisms underlying this trap and evaluate the efficacy of targeted policy interventions, we turn to a structural life-cycle model. Our framework explicitly allows genetic risk to govern the stochastic transitions of both mental and physical health, while also impacting wage offers to capture the cumulative effects of genetic predisposition on early-life human capital accumulation.

Figure 5: “Depression Trap” Dynamics by Mental Health PGI



Notes: This figure displays the annual transition probabilities into and out of the “depression trap,” defined as the joint state of non-employment and poor mental health ($\text{GHQ-12} \geq 2$). The left panel (Entry) shows the probability of transitioning from a healthy/employed state into the trap; the right panel (Exit) shows the probability of leaving the trap for any other state. Transitions are stratified by genetic risk for depression, comparing men above and below the median MH PGI. High-risk men (low PGI) exhibit a significant deficit in exit rates, illustrating the increased persistence of the trap for genetically vulnerable individuals. Standard errors are clustered at the household level.

4 Model

We develop a partial equilibrium life-cycle model of consumption, labor supply, and health dynamics, incorporating endogenous decisions regarding psychotherapy. The life cycle commences at age $t = 25$ and terminates at $T = 80$. Agents remain active in the labor market until the exogenous retirement age $t_{ret} = 65$, after which they withdraw from the labor force and rely on pension income and accumulated savings.

State Space and Timing

Time is discrete and indexed by $t \in \{25, \dots, T\}$. An agent is characterized by the state vector $S_t = \{x, g, t, mh_t, ph_t, a_t, \epsilon_t\}$. The vector includes endogenous and predetermined variables: x captures unobserved permanent heterogeneity (type); g indicates genetic predisposition for depressive symptoms; t denotes age; mh_t and ph_t represent mental and physical health status, respectively; a_t denotes assets; and ϵ_t is an idiosyncratic stochastic shock to wages. The timing of events within each period t is as follows:

Shock Realization: At the beginning of the period, stochastic shocks are realized. These include shocks to wages (during the working life), health transitions, and, for retired agents, survival. These realizations fully resolve the state vector S_t .

Decisions: Observing the fully realized state S_t , the agent makes choices conditional on their life-cycle stage:

- *Working Life* ($25 \leq t < 65$): The agent simultaneously chooses consumption c_t , labor supply $n_t \in \{0, 1\}$ (where $n_t = 1$ denotes employment), and mental health therapy $\tau_t \in \{0, 1\}$.
- *Retirement* ($65 \leq t \leq 80$): labor supply is fixed at zero ($n_t = 0$) and therapy is unavailable ($\tau_t = 0$). The agent chooses only consumption c_t (and implicitly savings a_{t+1}).

Dynamics: Choices made in the current period endogenously influence the state transition for period $t + 1$:

- *Health Evolution:* During the working life, health dynamics are endogenous; the transition probability to future mental health state mh_{t+1} depends on current health, genetics, and the agent's choices of labor supply n_t and therapy τ_t . In retirement, health transitions become exogenous, depending only on the previous health state and age.
- *Survival:* Survival is assumed to be certain during the working life. Upon entering retirement ($t \geq 65$), agents face an age- and health-dependent survival probability $\zeta_t(mh_t, ph_t)$. The model terminates deterministically at age $T = 80$.

Agents choose decision rules to maximize the expected present discounted value of lifetime utility, subject to the period budget constraint and survival risk in retirement.

Heterogeneity

We characterize agent heterogeneity along two distinct dimensions: observable genetic predisposition and unobserved permanent heterogeneity.

First, a key feature of our framework is the inclusion of an observable genetic predisposition for depressive symptoms, denoted by g . By treating this genetic endowment as observable, we directly map biological risk factors into the economic model.

Second, beyond genetic factors, we incorporate time-invariant unobserved heterogeneity to capture persistent ex-ante differences in endowments (e.g., innate ability or non-genetic health robustness). Accounting for these persistent traits is crucial for identification; neglecting them risks confounding permanent individual characteristics with state dependence in the stochastic processes. For instance, without this correction, the model might misattribute a sub-population's persistently poor health to a high probability of adverse shocks, rather than to their underlying type.

To implement this computationally, we discretize unobserved heterogeneity following the classification method of [Bonhomme et al. \(2022\)](#). We postulate that agents belong to a finite set of latent types, denoted by x , which govern their productivity and health trajectories.

Health Dynamics

We model the joint evolution of physical (ph) and mental (mh) health as a first-order Markov process. Each dimension is discretized into three ordered states, $h \in \{G, M, S\}$, corresponding to *Good*, *Moderate*, and *Severe* health. In our empirical application, physical health states map to Excellent/Good (G), Fair

(M), and Poor/Very Poor (S), while mental health states represent the clinical progression of depression: asymptomatic (G), mild-to-moderate symptoms (M), and Major Depressive Disorder (S).

Health transitions are governed by an ordered logit framework, where the observed state h_{t+1} is determined by an underlying latent continuous variable h_{t+1}^* falling within estimated thresholds $\{\kappa_j\}$. Assuming the idiosyncratic shocks $(\iota_{t+1}, \upsilon_{t+1})$ follow a standard logistic distribution, the latent process for physical health evolves as:

$$ph_{t+1}^* = ph_t\pi_1 + mh_t\pi_2 + n_t\pi_3 + S_t^{exo}\pi_4 + \iota_{t+1}, \quad (4)$$

where ph_t and mh_t are vectors of indicators for current health status, $n_t \in \{0, 1\}$ is the labor supply choice, and S_t^{exo} collects exogenous covariates including age, unobserved type (x), and genetic predisposition (g). The latent mental health process is analogous, except that it also incorporates the endogenous therapy decision, $\tau_t \in \{0, 1\}$:

$$mh_{t+1}^* = mh_t\mu_1 + \tau_t\mu_2 + n_t\mu_3 + ph_t\mu_4 + S_t^{exo}\mu_5 + \upsilon_{t+1}, \quad (5)$$

where μ_2 captures the mental health returns to psychotherapy. While the standard ordered logit model assumes proportional odds, implying constant coefficients across health thresholds, this restriction is rejected for several key predictors in our data. Consequently, we generalize the specification to a *Partial Proportional Odds* (PPO) model. This approach allows coefficient vectors to vary across thresholds, capturing critical non-linearities in how employment and therapy influence transitions out of severe versus moderate states. The resulting probability of transitioning to state j or better is:

$$P(h_{t+1} \leq j \mid \mathbf{X}_t) = \Lambda(\kappa_j - \mathbf{X}_t\beta_j), \quad (6)$$

where $\Lambda(\cdot)$ denotes the logistic cumulative distribution function and β_j is the threshold-specific parameter vector.

Flow Utility

In each period t , agents derive utility from a bundle of consumption c_t and effective leisure L_t , while incurring a distinct disutility if they engage in therapy ($\tau_t = 1$). The flow utility function takes a Constant Relative Risk Aversion (CRRA) form over the consumption-leisure composite:

$$U(c_t, n_t, \tau_t) = \frac{(c_t^\gamma L_t^{1-\gamma})^{1-\omega}}{1-\omega} - \psi(n_t)\tau_t, \quad (7)$$

where ω represents the coefficient of relative risk aversion, and γ governs the relative weight placed on consumption. Effective leisure, L_t , is defined as the total time endowment (normalized to 1) less the time costs associated with employment:

$$L_t = 1 - (0.292 + \delta(mh_t, ph_t)) \cdot n_t. \quad (8)$$

Here, $n_t \in \{0, 1\}$ denotes labor supply. The constant 0.292 represents the fixed time cost of employment, corresponding to a standard work week of approximately 37.5 hours (roughly 29.2% of total available time).⁴

The term $\delta(mh_t, ph_t)$ acts as a health-dependent “time tax” on labor. It captures the additional effective time lost due to poor health when employed—reflecting factors such as absenteeism, presentee-ism, or increased recovery time. We specify this penalty as linear in the health state vectors:

$$\delta(mh_t, ph_t) = \delta'_1 mh_t + \delta'_2 ph_t. \quad (9)$$

This specification generates a complementarity between health and consumption: poor health reduces effective leisure L_t , which in turn affects the marginal utility of consumption (governed by $\omega > 1$), thereby altering the incentive to work. This mechanism is crucial for explaining the high labor force exit rates among agents with deteriorating mental health (Abramson et al., 2024; Cronin et al., 2024).

Agents who engage in therapy incur a disutility cost $\psi(n_t)$. Despite its clinical effectiveness, therapy is often under-utilized (Cronin et al., 2024). We interpret $\psi(n_t)$ as capturing both non-pecuniary stigma and the time cost of attending sessions. We allow $\psi(n_t)$ to vary by employment status, since the opportunity cost of time is plausibly higher when the agent is working.

Finally, we allow for mortality risk after retirement. At the end of each period, a retired agent survives with probability $\zeta(mh_t, ph_t, t)$. If the agent dies, they derive utility from leaving bequests (a_{t+1}). Following French and Jones (2011) and De Nardi (2004), the bequest motive is:

$$\varphi(a_{t+1}) = \theta \frac{(a_{t+1} + \kappa)^{(1-\omega)\gamma}}{1 - \omega}, \quad (10)$$

where θ governs the strength of the bequest motive and κ controls the luxury-good nature of bequests.

Budget Constraint and Earnings

Agents face a period-by-period budget constraint that limits consumption and savings based on current asset holdings and disposable income. The constraint is given by:

$$c_t + a_{t+1} = (1 + r)a_t + y_t - \mathbb{I}_{t < 65} \cdot P_\tau \tau_t, \quad a_{t+1} \geq 0, \quad (11)$$

where r denotes the risk-free interest rate, and P_τ represents the out-of-pocket pecuniary cost of psychotherapy. We impose a strict borrowing constraint ($a_{t+1} \geq 0$).

⁴Based on average weekly working hours among employed working-age men in our sample.

Disposable income y_t depends on the agent's life-cycle stage, labor supply choice, and health status:

$$y_t = \begin{cases} n_t w_t (1 - \tau(w_t)) + (1 - n_t) b(mh_t, ph_t) & \text{if } t < 65 \text{ (Working Age)} \\ p(x) & \text{if } t \geq 65 \text{ (Retirement).} \end{cases} \quad (12)$$

During working life, employed agents ($n_t = 1$) earn the after-tax wage $w_t(1 - \tau(w_t))$, where $\tau(w_t)$ denotes the progressive tax rate. If they do not work ($n_t = 0$), they receive health-contingent government transfers $b(mh_t, ph_t)$, which capture disability benefits or social support. After retirement ($t \geq 65$), agents receive a fixed pension income $p(x)$, which varies by the agent's permanent unobserved type x .

The market wage offer w_t evolves according to a Mincerian process augmented with health and genetic traits:

$$\ln w_t = \beta_0 + \beta_1 g + \beta_2 x + \beta_3 mh_t + \beta_4 ph_t + \beta_5 t + \beta_6 t^2 + \epsilon_t. \quad (13)$$

Here, wages are determined by the agent's observable genetic predisposition g , unobserved permanent type x , and current health status mh_t and ph_t , alongside a quadratic age profile. The idiosyncratic productivity shock ϵ_t follows an AR(1) process:

$$\epsilon_{t+1} = \rho \epsilon_t + \eta_{t+1}, \quad \eta_{t+1} \sim \mathcal{N}(0, \sigma_\eta^2), \quad (14)$$

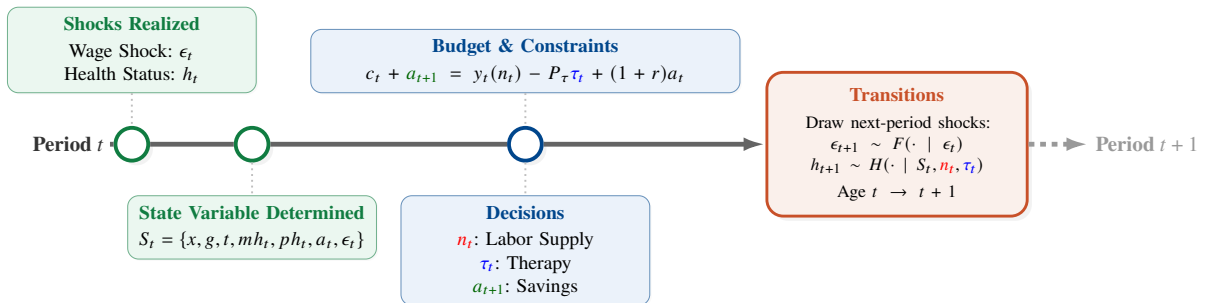
where η_{t+1} represents a transient shock to labor productivity.

Recursive Formulation

The agent enters the model at age $t = 25$ and lives up to a maximum age of $T = 80$. The life cycle is partitioned into a working stage ($t \in [25, 64]$) and a retirement stage ($t \in [65, 80]$).

Working Age ($25 \leq t < 65$): In each working-age period, the agent's state is summarized by $S_t = \{x, g, t, mh_t, ph_t, a_t, \epsilon_t\}$. Given S_t , the agent chooses labor supply n_t , therapy τ_t , consumption c_t , and next-period assets a_{t+1} to maximize the sum of flow utility and the discounted expected continuation value. The within-period dynamics is illustrated in Figure 6.

Figure 6: Working Age Within-Period Dynamics



The Bellman equation is

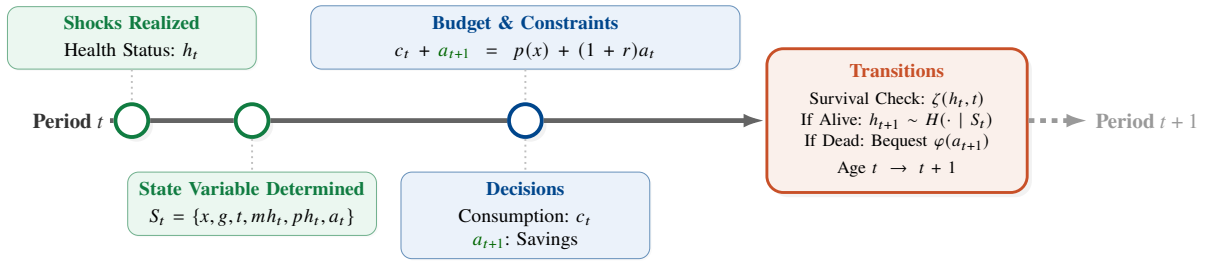
$$V_t(S_t) = \max_{n_t, a_{t+1}, \tau_t} \left\{ U(c_t, n_t, \tau_t) + \beta \iint V_{t+1}(S_{t+1}) dH(h_{t+1} | S_t, n_t, \tau_t) dF(\epsilon_{t+1} | \epsilon_t) \right\}, \quad (15)$$

subject to the period budget constraint and the non-negativity of assets. The expectation is taken over next-period wage shocks ϵ_{t+1} and next-period health $h_{t+1} \equiv (mh_{t+1}, ph_{t+1})$, which evolves endogenously with current choices (n_t, τ_t) .

Retirement ($t \geq 65$): Upon reaching age 65, the agent retires exogenously. The agent's state is summarized by $S_t = \{x, g, t, mh_t, ph_t, a_t\}$. labor supply is fixed at $n_t = 0$, and therapy is no longer available ($\tau_t = 0$). Income consists of a fixed pension $p(x)$ and returns on assets.

Retirement utility is shaped by survival risk. At the end of each period, the agent survives with probability $\zeta(h_t)$, where $h_t \equiv (mh_t, ph_t)$. If the agent dies, they derive utility from bequeathing remaining assets. The within-period dynamics for the retirement phase are depicted in Figure 7.

Figure 7: Retirement Within-Period Dynamics



The Bellman equation incorporates survival risk:

$$V_t(S_t) = \max_{a_{t+1}} \left\{ U(c_t, 0, 0) + \beta \left[\underbrace{\zeta(h_t, t) \int V_{t+1}(S_{t+1}) dH(h_{t+1} | S_t)}_{\text{Survival}} + \underbrace{(1 - \zeta(h_t, t)) \varphi(a_{t+1})}_{\text{Death}} \right] \right\}, \quad (16)$$

subject to the retirement budget constraint and the non-negativity of assets. At the terminal age $T = 80$, the continuation value is zero, and the agent consumes their remaining wealth or leaves a bequest with probability 1.

5 Estimation and Identification

We estimate the model parameters using a three-step procedure designed to ensure transparent identification. In the first step, we calibrate a subset of standard parameters to established values from the literature. In the second step, we estimate the exogenous processes governing health transitions and institutional constraints directly from the data, outside of the structural model. In the final step, we jointly estimate the remaining behavioural parameters within the model using the simulated method of moments (SMM). This strategy allows us to anchor the model's driving forces in empirical evidence while exploiting its full structure to identify the deep parameters shaping life-cycle decisions. Table 3 summarizes the parameters and their sources of identification.

5.1 Calibrated Parameters

We calibrate three parameters using external evidence. We set the annual risk-free interest rate to $r = 1.5\%$, following the long-run estimates in [Holston et al. \(2017\)](#). We set the coefficient of relative risk aversion to $\omega = 3.0$, consistent with the life-cycle estimates used in [De Nardi et al. \(2010\)](#). Finally, identifying the causal effect of psychotherapy (μ_2) is challenging because treatment is endogenous: individuals typically seek therapy following negative mental health shocks. We therefore calibrate μ_2 using external evidence from randomized controlled trials. We take the standardized mean difference ($SMD \approx 0.6$) reported in meta-analyses and convert it into a log-odds ratio suitable for our ordered-logit health transition, using the approximation in [Borenstein et al. \(2009\)](#):

$$\mu_2 \approx 0.6 \times SMD \times \frac{\pi}{\sqrt{3}}. \quad (17)$$

This calibration follows the approach in recent structural work on mental health treatment ([Abramson et al., 2024](#); [Cronin et al., 2024](#)).

5.2 Estimation Outside the Model

I. Individual Heterogeneity

Individuals have types x that take values on a discrete set $x \in \{1, \dots, K\}$. We estimate these types by building upon the approach of [Bonhomme et al. \(2022\)](#) (BLM) (see also [Bonhomme et al. \(2019\)](#)). BLM estimate individual heterogeneity classes by k -means clustering on relevant moments of outcome variables, with moments calculated at an individual level. Worker have types $x \in \{1, \dots, K\}$, there are individuals $i \in \{1, \dots, n\}$ and $\mathbf{m}_i$ is a vector of M moments of individual i 's outcomes. Workers are partitioned into K classes by finding the partition that solves

$$\min_{\{x_i\} \in \{1, \dots, K\}^N} \sum_{i=1}^N \|\mathbf{m}_i - \tilde{\mathbf{m}}(x_i)\|^2, \quad (18)$$

Table 3: Model Parameters, Identification Strategy, and Data Sources

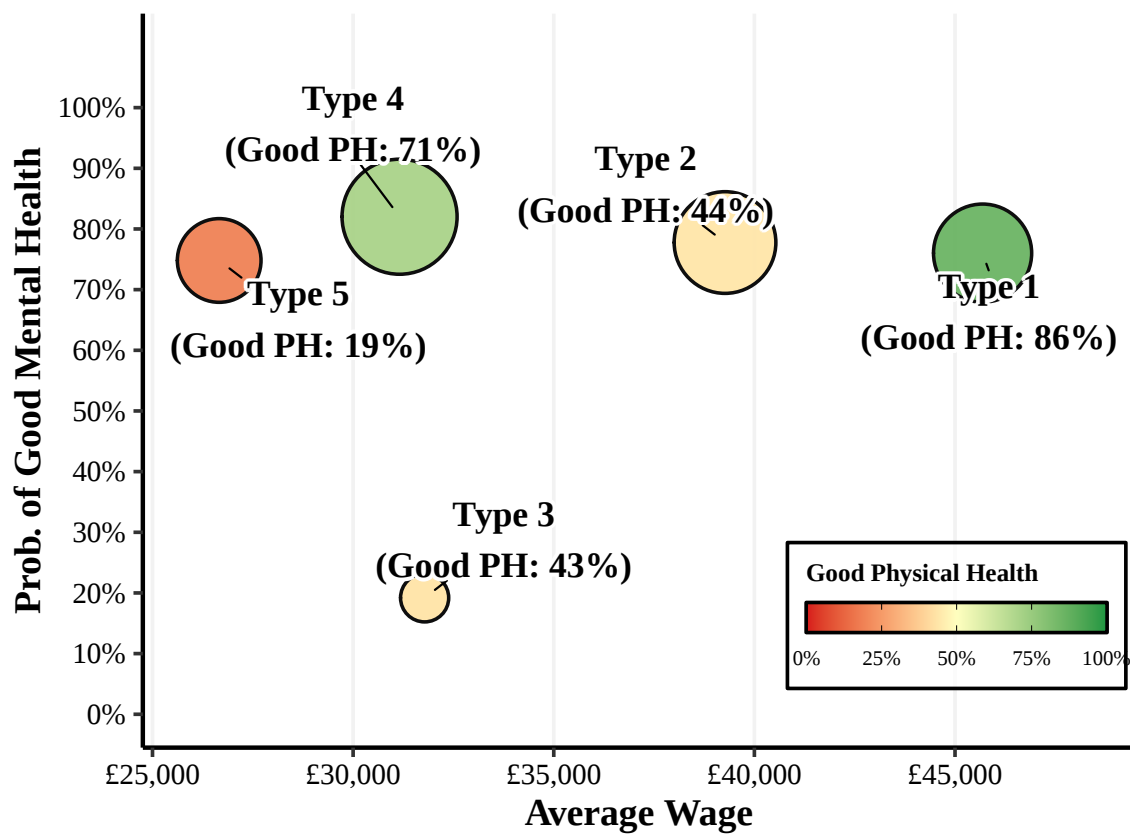
Parameter	Equation	Description	Targeted Empirical Moments	Source
Panel A: Calibrated Parameters				
r	Eq. 11	Annual risk-free interest rate	$r = 1.5\%$	Holston et al. (2017)
μ_2	Eq. 5	Effectiveness of therapy	RCT effect of therapy on MH outcomes	Cronin et al. (2024)
ω	Eq. 7	Coefficient of relative risk aversion	$\omega = 3.0$	De Nardi et al. (2010)
Panel B: Parameters Estimated Outside the Model				
$\mu_1, \mu_3 - \mu_5$	Eq. 5	Mental health process	MH transition process	UKHLS
$\pi_1 - \pi_4$	Eq. 4	Physical health process	PH transition process	UKHLS
$b(h_t)$	Eq. 12	Benefits schedule	Mean benefits by health and employment status	UKHLS
$\tau(w_t)$	Eq. 12	Progressive tax rate	Tax rate by labor income	UKHLS
P_τ	Eq. 11	Therapy pecuniary cost	Mean out-of-pocket therapy expenditure per treatment episode	UKHLS
$\zeta(h_t, t)$	Eq. 16	Survival probability	Survival rates by age and health	UKHLS & UK Life Tables
Panel C: Parameters Estimated via SMM				
$\psi(n_t)$	Eq. 7	Therapy disutility	Therapy take-up by employment status (2 moments)	UKHLS
$\delta(\cdot)$	Eq. 8	Health-dependent time tax on labor	Employment rates by MH/PH groups (6 moments)	UKHLS
ρ, σ_η	Eq. 14	Wage shock parameters	S.D. of wages by health, type, and genotype group (13 moments)	UKHLS
$\beta_0 - \beta_6$	Eq. 13	Wage process	Mean wages by age, health, type, and genotype group (21 moments)	UKHLS
β	Eq. 16	Discount factor	S.D. of wages by age group (8 moments)	UKHLS
γ	Eq. 7	Consumption share in utility	Aggregate saving ratio (1 moment)	UKHLS
θ, κ	Eq. 10	Bequest parameters	Mean and median of bequest (2 moments)	UKHLS

Notes: This table summarizes the model parameters and their corresponding moments. Panel A lists parameters calibrated to standard values in the literature. Panel B lists parameters estimated directly from the data outside the model in a first step. Panel C lists parameters estimated via Simulated Method of Moments (SMM) by minimizing the distance between model-generated moments and data moments. The column “Targeted Empirical Moments” reports the moments that primarily identify each parameter. Therapy cost is computed as the average session cost (£130; Steele, 2026) times the average number of sessions attended (8.6; Steele, 2026) times the share of patients attending private therapy (26%, UKHLS).

The consistency of BLM’s approach relies on an injective map between individual unobserved heterogeneity and the vector of moments m_i . In our application, productivity, mental health and physical health are the primary sources of individual heterogeneity. We therefore compute individual moments m using average wages, mental health status and physical health status.

Applying the clustering procedure described in Appendix D, we summarize persistent individual heterogeneity with a discrete type $x_i \in \{1, \dots, 5\}$. Figure 8 reports the five estimated types and their centroid characteristics in terms of average wages, the probability of good mental health, and physical health. Type 1 is a high-wage, high-health group. Type 2 has relatively high wages and good mental health but substantially worse physical health. Type 3 is characterized by very low mental health despite moderate wages and physical health. Type 4 combines high mental health with relatively good physical health at lower wages. Type 5 is a low-wage group with the poorest physical health. We treat x_i as a fixed state variable in the structural model.

Figure 8: Individual Heterogeneity: Estimated Types



Notes: This figure summarizes persistent individual heterogeneity using five discrete latent types, $x_i \in \{1, \dots, 5\}$, estimated via the clustering procedure described in Appendix D. Each type is represented by its centroid characteristics in average wages, the probability of good mental health, and the probability of good physical health. Type 1 is a high-wage, high-health group. Type 2 has relatively high wages and good mental health but worse physical health. Type 3 is characterized by low mental health despite moderate wages and physical health. Type 4 combines high mental health with relatively good physical health at lower wages. Type 5 is a low-wage group with the poorest physical health. Types are treated as a fixed state variable in the structural model.

II. Other Estimation

We estimate a set of parameters directly from the data outside the structural model. These objects discipline health dynamics, non-labor income, therapy costs, and survival risk. We treat them as inputs in the second-stage SMM estimation.

We estimate the mental and physical health transition processes using the ordered-logit specifications in Equation 5 and Equation 4. The parameters μ_1 and $\mu_3\text{--}\mu_5$ govern the evolution of mental health, while $\pi_1\text{--}\pi_4$ govern the evolution of physical health. We estimate these equations on the UKHLS panel and use the fitted probabilities to construct the health transition kernel in the model.

We also estimate the non-labor income components that enter the budget constraint. The benefits schedule $b(h_t)$ in Equation 12 is measured from UKHLS as mean benefit income by health and employment status. The pecuniary therapy cost P_τ in Equation 11 is constructed as the average out-of-pocket cost per treatment episode. We compute it as the product of the average session cost (£130), the average number of sessions attended (8.6), and the share of patients who attend private therapy (26%, UKHLS).

Finally, we model survival risk in retirement. We estimate the survival probability $\zeta(h_t, t)$ in Equation 16 using age- and health-specific survival rates. We combine UK life tables with the model's health classification to discipline mortality risk over the life cycle. These survival probabilities pin down effective retirement horizons and are central for matching asset de-cumulation and bequest behaviour.

Details on the first-stage estimates are reported in Table 8 to Table 10.

5.3 Estimation of Core Structural Parameters via SMM

We estimate the remaining structural parameters using the Simulated Method of Moments (SMM). Identification follows a simple economic logic: each parameter is disciplined by the empirical feature it most directly governs in the model. The moments we target and their mapping to parameters are summarized in Panel C of Table 3.

Treatment demand and labor–health interaction: The therapy disutility $\psi(n_t)$ governs the take-up decision conditional on health and prices. A higher $\psi(n_t)$ lowers treatment use, and allowing it to vary with employment shifts take-up differentially between workers and non-workers. We therefore discipline $\psi(n_t)$ using therapy participation rates by employment status. The health-dependent time tax on labor, $\delta(\cdot)$ in Equation 8, captures how poor mental and physical health reduce effective leisure when employed. A larger $\delta(\cdot)$ makes work less attractive in bad health and increases labor-force exit. We identify $\delta(\cdot)$ from employment rates across joint mental and physical health groups.

Earnings risk and the wage process: We model wages with a persistent shock component governed by (ρ, σ_η) and a deterministic component parameterized by $\beta_0\text{--}\beta_6$ in Equation 13. The persistence parameter ρ controls how strongly current wages predict future wages, while σ_η governs cross-sectional dispersion conditional on observables. We discipline (ρ, σ_η) using the standard deviation of wages across groups

defined by health, type, and genotype, and we identify the coefficients β_0 – β_6 using mean wages by age, health, type, and genotype group.

Inter-temporal choices and bequests: The discount factor β governs inter-temporal smoothing and precautionary saving. In our setting, it primarily affects the dispersion of wages over the life cycle through endogenous selection into work and the accumulation of health states. We therefore discipline β using age-group moments in wage dispersion. The consumption share parameter γ controls the relative weight on consumption in the consumption–leisure composite and shifts saving incentives. We identify γ using the aggregate saving ratio. Finally, the bequest parameters (θ, κ) govern the level and curvature of the bequest motive in Equation 10. They pin down wealth de-cumulation late in life and the mass of terminal assets. We discipline (θ, κ) using the mean and median of observed bequests.

Implementation proceeds as follows. For a given parameter vector, we solve the model and simulate life-cycle histories for a large synthetic panel. We compute the model-implied counterparts of the targeted moments and choose parameters to minimize a weighted quadratic distance between simulated and empirical moments. The identification strategy exploits the tight mapping from preferences, earnings risk, and health-dependent work costs into observed treatment, labor supply, wage, saving, and bequest patterns.

5.4 Model Fit

Overall, the model matches the targeted moments well. It reproduces the main life-cycle patterns in earnings and the key cross-sectional gradients by genetic endowment, health, and unobserved type. It also fits the central margins that discipline labor supply, treatment, saving, and bequests. We summarize the fit in Figure 9 and Figure 10.

Figure 9 compares earnings moments in the data to their simulated counterparts. The model matches the main life-cycle profile of mean earnings: earnings rise from the late 20s to the early 50s and then level off. The model also reproduces the cross-sectional gradients. High-*PGI* agents earn more than low-*PGI* agents. Earnings are higher for agents in better mental and physical health. Type means are well ordered, with Type 1 and Type 2 at the top and Type 5 at the bottom.

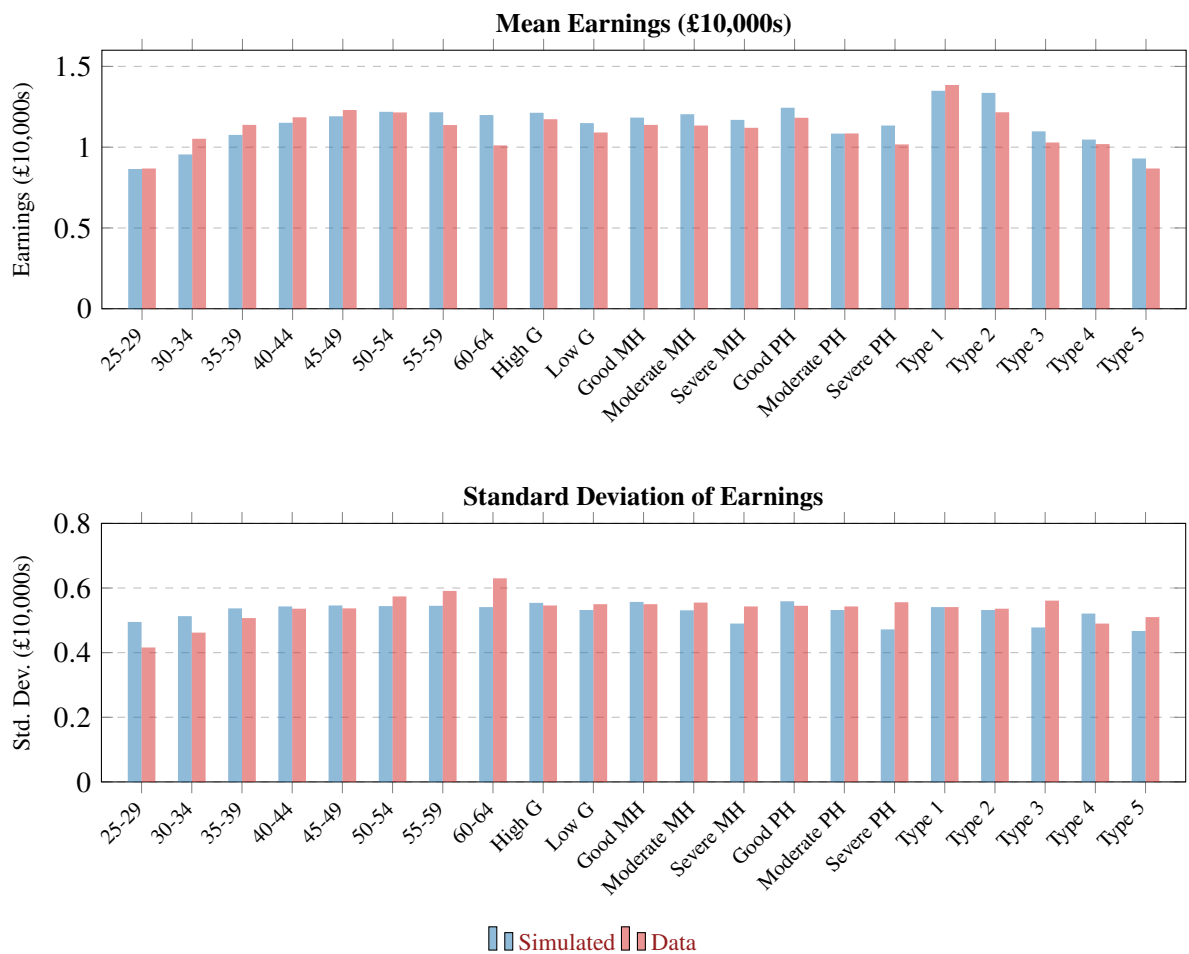
The remaining gaps are small and concentrated late in working life and in the dispersion profile. The model generates a slightly weaker decline in mean earnings after age 55. This likely reflects institutional features and selection mechanisms that become more salient near retirement, such as partial retirement and hours reductions within employment, which we abstract from. The model also produces a flatter age profile of earnings dispersion than in the data. In the data, dispersion rises strongly at older ages, while the model keeps dispersion closer to constant. This difference is consistent with the model’s parsimonious wage-risk structure, which does not allow the variance of shocks to increase with age or incorporate additional late-life heterogeneity in job loss and hours.

Figure 10 shows fit along other key margins. The model matches employment rates by mental and physical health states closely, indicating that the estimated health processes and the health-dependent

work cost capture the labor-supply gradients in the data. Treatment rates for employed and non-employed agents are also matched tightly by construction, validating the calibration of the monetary treatment cost and the estimation of therapy disutility. The model slightly over-predicts the aggregate saving ratio, but the level remains close, suggesting that inter-temporal incentives are broadly consistent with the data. Finally, the model matches the median bequest closely and modestly understates the mean bequest. This pattern indicates a good fit in the centre of the terminal wealth distribution, with a somewhat thinner right tail than in the data, which is expected in a model without rare, very large bequests.

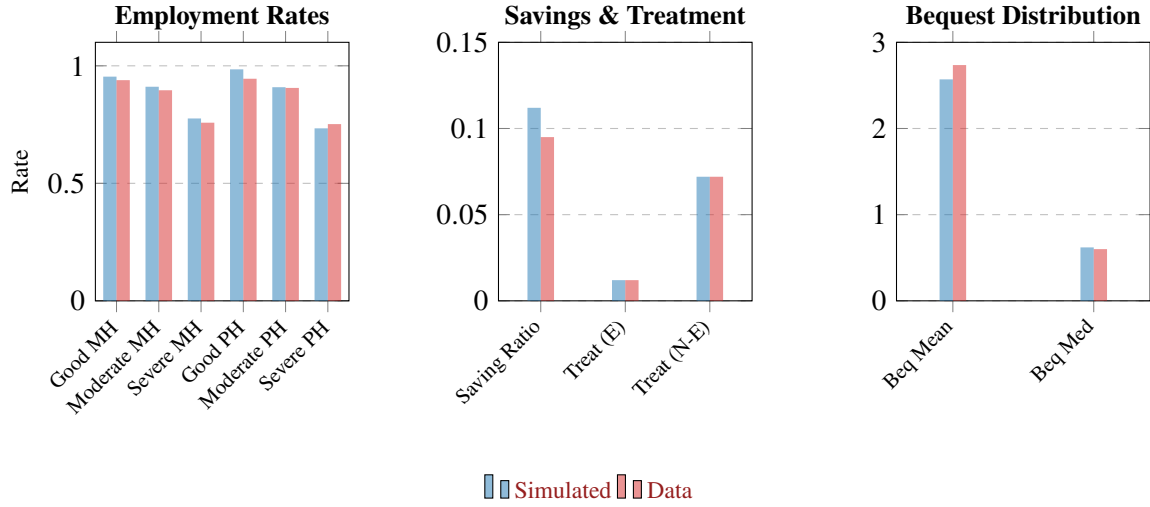
Details on the estimated structural parameters are reported in Appendix Table 11.

Figure 9: Model Fit: Simulated vs. Data Earnings Moments



Notes: The figure compares simulated (blue) and empirical (red) moments of earnings. The top panel shows the mean earnings (in £10,000s), and the bottom panel shows the standard deviation. High/Low G denote High/Low genetic endowment. Good/Moderate/Severe MH and Good/Moderate/Severe PH denote tertiles of mental and physical health. Type 1–5 denote unobserved worker types.

Figure 10: Model Fit: Other Key Moments



Notes: The figure compares simulated (blue) and empirical (red) moments across three categories. Left panel: employment rates by health tertiles. Middle panel: aggregate savings ratio and treatment rates by employment status (E: Employed, N-E: Non-Employed). Right panel: mean and median of the bequest distribution (£10,000s).

6 Results

This section presents the main findings from our structural model, focusing on the role of mental health polygenic index (MH PGI) in shaping individual life-cycle trajectories and determining the success of clinical policy interventions. Our framework allows us to disentangle latent genetic risk from observable initial conditions, providing a controlled environment to measure how genetic endowments interact with exogenous shocks and endogenous labor supply decisions.

The logic of the section flows from individual-level mechanisms to aggregate policy evaluation. We begin in Section 6.1 by establishing the baseline lifetime impact of mental health genetic risk, showing its compounding effects on employment, wealth, and health. Section 6.2 deepens this analysis by introducing exogenous health and wage shocks, revealing a critical feedback loop: high genetic risk not only delays recovery from health shocks but fundamentally transforms temporary wage losses into a persistent “depression trap”. Having established these mechanisms, Section 6.3 scales the analysis to the population level to evaluate the fiscal and clinical viability of psychotherapy subsidies. We test whether the provision of mental health care can pay for itself. By helping individuals escape the “depression trap”, the government can generate financial returns via higher tax revenues and lower benefit payouts. We specifically explore a novel approach to this problem by targeting subsidies based on ex-ante genetic risk.

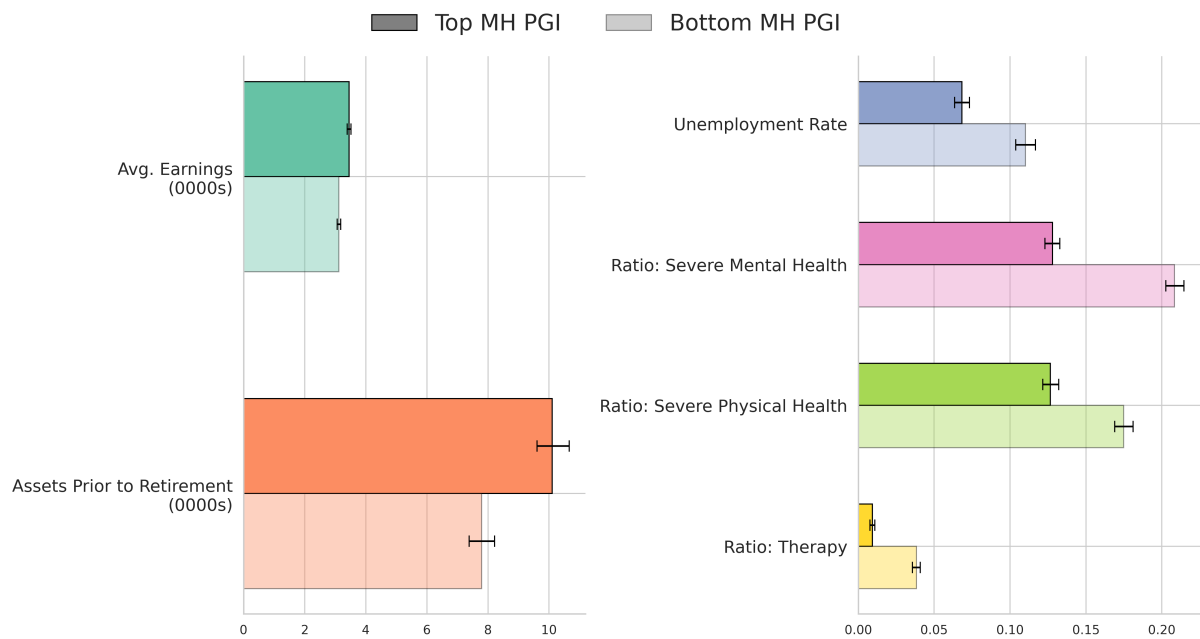
6.1 The Impact of Mental Health Genetic Endowment on Lifetime Outcomes

Mental health genetic endowment generates large and persistent differences in life-cycle trajectories, even when individuals start from the same non-genetic conditions. In our counterfactual, a high MH PGI lowers unemployment by about 4 p.p., raises average earnings, and increases pre-retirement wealth by roughly 25%. It also reduces severe mental health episodes and lowers the risk of severe physical health.

Figure 11 illustrates the dynamic effects of mental health genetic risk on life-cycle outcomes. To isolate the role of genetics, we simulate individuals over a 10-year period, holding all non-genetic initial conditions fixed. We then counterfactually swap each individual's MH genetic endowment (Top versus Bottom MH PGI) and aggregate the differences. By shutting down selection on initial health, earnings, and assets, any resulting gaps reflect the dynamic impact of genetic risk beyond baseline mental health.

The simulation generates substantial differences in labor market outcomes and wealth accumulation. Over the 10-year period, individuals with a favourable genetic endowment (Top MH PGI) earn noticeably more on average and spend less time out of work. The model implies an unemployment rate near 7 p.p. for the Top MH PGI group, compared to roughly 11 p.p. for the Bottom MH PGI group. These labor-market advantages compound over time. As a result, the top genetic group accumulates roughly 25% more in pre-retirement assets than the bottom group, despite starting the counterfactual with identical wealth.

Figure 11: Lifetime Outcomes by Mental Health PGI



Notes: This figure reports counterfactual life-cycle differences implied by swapping each individual's mental health genetic endowment (high $\leftrightarrow$ low) while holding all other initial characteristics fixed at their empirical values. For each individual and horizon, the model computes the implied high–low difference, which is then aggregated to obtain the outcomes shown. The sample includes individuals first observed between ages 25–45, and outcomes are simulated over the subsequent 10 years. All metrics are computed from this 10-year window, except pre-retirement assets, which are obtained by simulating each individual forward to age 65 and aggregating assets accumulated prior to retirement.

Health and behavioural outcomes follow a similarly stark pattern. The Bottom MH PGI group experiences a substantially higher incidence of severe mental health states. The simulation also generates worse physical health outcomes for this group, highlighting co-morbidity between mental and physical health in the model. Consequently, therapy utilization differs sharply: individuals with higher genetic risk are more likely to use therapy.

Taken together, these findings are consistent with reduced-form evidence on the lifetime impact of genetic risk. They show that MH genetic endowment is not merely a proxy for initial health status. Instead, it is a persistent driver of health, labor, and wealth dynamics over the life cycle.

6.2 Genetic Endowment as a Unique Predictor of the Mental Health Trap

Genetic risk predicts who gets stuck. Following an adverse shock, high-risk individuals are not only more likely to enter a mental health (MH) trap, but they also exit more slowly. While the gap in recovery is short-lived after a severe mental health shock, it becomes highly persistent following a extreme negative wage shock.

Figure 12 shows how individuals enter and exit the mental health trap after an adverse shock. We define the mental health trap as being non-employed and in a severe mental health state. We initialize the model at the empirical distribution and impose an exogenous shock in Year 2. We then track the gap between the counterfactual economy and the benchmark economy for the Top and Bottom MH PGI groups.

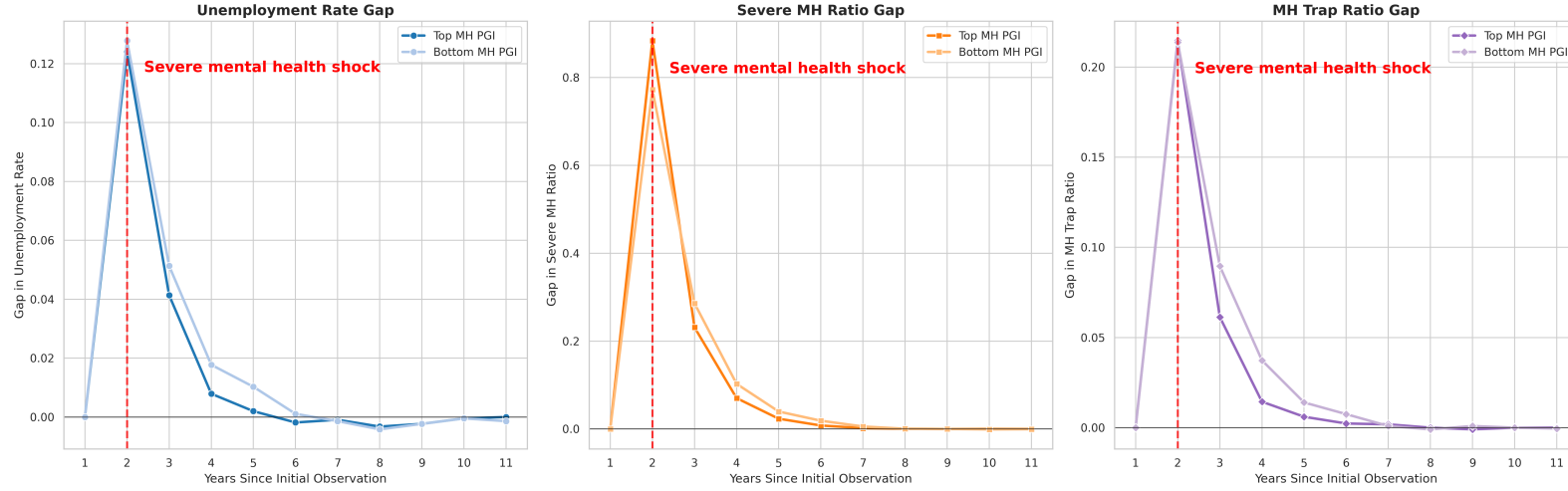
Panel A simulates a severe mental health shock by forcing all individuals into the severe MH state in Year 2. This triggers a sharp, immediate spike in both unemployment and the mental health trap. While individuals with high genetic risk experience larger initial disruptions and slower recoveries, the gap between the high- and low-risk groups eventually narrows as the shock dissipates. This suggests that following a severe mental health shock, genetic endowment primarily governs the *speed of recovery*.

Panel B, in contrast, simulates an economic shock by assigning individuals the extreme bad wage draw in Year 2. Unlike the health shock, the resulting gap does not close in the long run. The negative productivity shock persistently elevates the risk of falling into the mental health trap, particularly for high-risk individuals. This divergence highlights a powerful feedback loop: a wage loss increases the likelihood of non-employment, which deteriorates mental health, which in turn further depresses employment prospects. Genetic risk *amplifies* this reinforcing mechanism, turning a temporary economic setback into a persistent mental health trap.

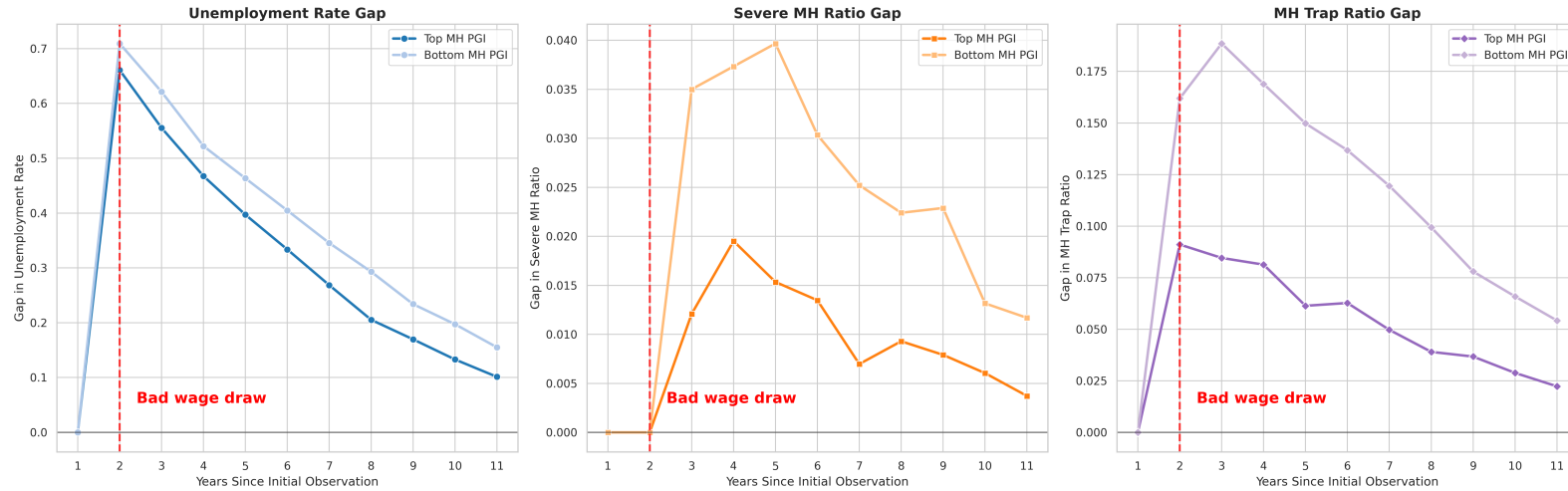
Crucially, these patterns hold even when conditioning on a strong pre-shock baseline (employed and in at least moderate mental health), as shown in Appendix Figures 17 and 18. This confirms that MH genetic endowment predicts vulnerability to the mental health trap even among observationally healthy and employed individuals. From a policy perspective, this underscores the value of genetic information for precision medicine and therapy (Collins and Varmus, 2015; Torkamani et al., 2018): it can help target early monitoring and preventative interventions toward individuals with high latent risk before an adverse shock cascades into a persistent trap.

Figure 12: Dynamic Responses to Exogenous Shocks by MH PGI Group

Panel A: Severe Mental Health Shock



Panel B: Bad Wage Draw



Notes: We start from the empirical initial distribution and select individuals first observed between ages 25–45. In Panel A, at the beginning of the year following an individual's first observation (Year 2), we exogenously force that individual to enter the severe mental health state, holding all other characteristics fixed. In Panel B, at the beginning of Year 2, we impose a bad wage draw by assigning the wage realization to the lowest of four discretized wage states. Under our discretization, this corresponds to a wage draw three standard deviations below the mean (approximately the bottom 0.14% of draws for individuals with the same characteristics). In both panels, we then simulate and track outcomes over the subsequent 10 years, separately for the Top and Bottom MH PGI groups. *mental health trap* is defined as the joint probability of being non-employed and in the severe mental health state. In each panel, the gap series are computed as the difference between the counterfactual economy with the exogenous shock and the benchmark economy.

6.3 Evaluation of a Therapy Subsidy

We evaluate the cost-effectiveness of a precision psychiatry framework that incorporates molecular genetics data to identify and support individuals with high underlying risk for depression. We simulate both universal psychotherapy subsidies and risk-informed alternatives directed at genetically vulnerable individuals, assessing their effects on treatment uptake, mental health, labor supply, and net fiscal balances.

Despite the clinical efficacy of treatment, psychotherapy is starkly underutilized: in our data, only 5% of individuals meeting the clinical criteria for Major Depressive Disorder report attending therapy. In our model framework, this low uptake is driven by both monetary costs (P_T) and non-pecuniary costs (ψ), which captures the effects of social stigma, the psychological burden of treatment-seeking and constraints in supply. We simulate a series of counterfactual experiments that vary the subsidy level from zero to over 100%, evaluating both “free at the point of use” care, and financial incentives for attendance.

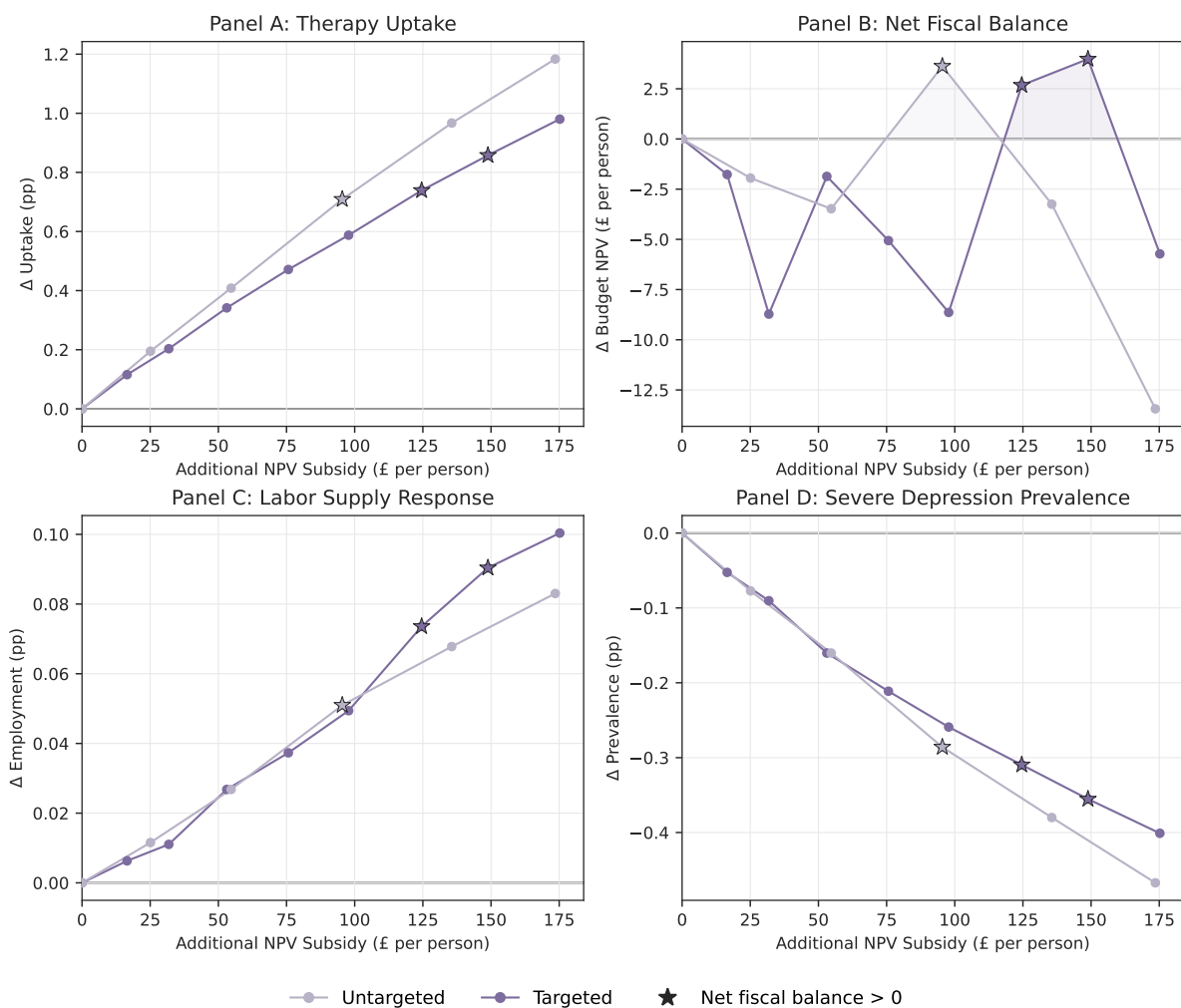
By incorporating endogenous labor supply alongside a realistic income tax and benefit system, we can evaluate the fiscal sustainability of expanded mental health care. This allows us to formally test the hypothesis that psychological interventions can be self-financing through increased tax revenue and diminished welfare dependency, a claim that famously provided the economic rationale for the UK’s Improving Access to Psychological Therapies (IAPT) initiative (Layard, 2006; Clark, 2011).

Furthermore, the unique advantage of our dataset and estimated model is that it features heterogeneity in genetic risk for depression. As the UK National Health Service moves towards offering whole-genome sequencing to all newborns (Davies, 2025), genetic risk for depression will increasingly be observable as part of a broader health assessment covering a variety of conditions — not as a standalone psychiatric label, but as one component of a holistic preventative framework. Within such a framework, elevated genetic risk could trigger more frequent clinical check-ins and mental health screening, facilitating timely treatment at the onset of symptoms. This mirrors established practice in other areas of preventive medicine, where biological risk markers (e.g., BRCA1/2 status for breast cancer; Antoniou et al., 2003) or familial hypercholesterolaemia screening in newborns (Wald et al., 2016) trigger enhanced monitoring and earlier clinical intervention.

Specifically, we compare universal subsidies to risk-informed alternatives that reduce treatment barriers for genetically at-risk individuals. It is not obvious *ex ante* whether a risk-informed intervention is preferable from a welfare perspective. While the effectiveness of psychotherapy does not appear to vary depending on genetic risk Bäckman et al. (2025), high-risk individuals are less productive and have worse physical health. Therapy interventions may therefore be less likely to elicit a positive labour supply response and may generate lower income tax revenue as a result. Moreover, high-risk individuals are more likely to attend psychotherapy in the status quo, so subsidies directed at this group may largely benefit infra-marginal users who would have sought treatment regardless. On the other hand, high-risk individuals are disproportionately likely to be in the “depression trap” and in receipt of large benefit payments; successfully facilitating their recovery may therefore generate a larger fiscal return.

Figure 13 illustrates the comparative impact of targeted versus untargeted therapy subsidies on population-level outcomes. An additional investment of £150 per person in NPV terms increases the uptake of psychotherapy by approximately 1 percentage point (Panel A). For context, this level of investment corresponds to either a universal near-zero price for therapy or, in the targeted scenario, providing high-risk individuals with a direct transfer of roughly £80 to attend. At every level of fiscal investment, the untargeted policy generates a higher aggregate uptake of treatment. This is driven by the baseline distribution of care: only 2.5% of high-risk and less than 1% of low-risk individuals attend therapy in the status quo. Consequently, targeted subsidies at low levels mostly go to infra-marginal users who would receive treatment anyway. Untargeted subsidies instead generate more new entry among low-risk individuals.

Figure 13: Policy Frontier under Alternative Subsidy Schedules: Pre-COVID Scenario



Notes: This figure plots policy frontiers for the pre-COVID calibration. Each point corresponds to a subsidy schedule indexed by its implied additional net present value (NPV) subsidy cost on the horizontal axis (in £ per person), measured relative to the baseline untargeted policy at a 74% subsidy rate. Lines connect points within each policy regime: an untargeted subsidy (uniform across individuals) and a targeted subsidy (varying by risk group). Panel A reports the change in therapy uptake (percentage points). Panel B reports the change in the government budget NPV (in £ per person); shaded areas indicate combinations with positive net fiscal balance (above zero). Panel C reports the change in employment (percentage points). Panel D reports the change in the prevalence of severe depression (percentage points). Star markers highlight policy points with positive net fiscal balance (Panel B > 0), and the same points are marked with stars in the other panels for comparison.

The increased treatment uptake translates directly into clinical gains. An investment of £150 per person generates an overall reduction in the prevalence of depression of 0.4 percentage points (Panel D). Because the untargeted intervention induces higher aggregate uptake, it generally results in a larger reduction in prevalence for a given level of expenditure. This effect is further bolstered by the fact that untargeted interventions reach individuals with fewer baseline risk factors, who are subsequently less prone to recurrence in the long run.

The labor supply and fiscal implications, however, reveal a more nuanced trade-off. For spending levels below £100 per person, the two policies generate broadly similar labor supply responses: while untargeted subsidies produce more total treatment, targeted subsidies are more effective at reaching the non-employed. Interestingly, at this £100 threshold, untargeted subsidies successfully “pay for themselves”, generating a fiscal surplus (Panel B). This fiscal dividend is driven by two factors: the higher tax revenue recovered from the more productive workers reached by universal subsidies, and the reduction in disability benefit expenditure among workers with poor physical health who lack a high genetic risk for depression.

As investment increases beyond £100, the targeted policy begins to dominate. At £150 per person, targeted subsidies generate both a stronger labor supply response and a larger fiscal surplus. This inflection point corresponds to the level where the subsidy becomes high enough to act as an explicit payment for attendance. These “incentive payments” successfully induce entry among high-risk men with mild depression who were previously reluctant to seek care. These findings suggest that while universal subsidies are more efficient at low levels, offering financial engagement incentives to genetically at-risk individuals is more effective at disrupting the ‘depression trap’, with the added benefit of generating positive fiscal returns

Therapy Subsidies in the Post-COVID Era

To examine the robustness of these policy trade-offs, we apply the same counterfactual experiments to the post-COVID (2021/2022) initial distribution⁵. As illustrated in Figure 14, the structural shifts in the economic and mental health landscape following the pandemic significantly alter the fiscal returns to therapy subsidies.

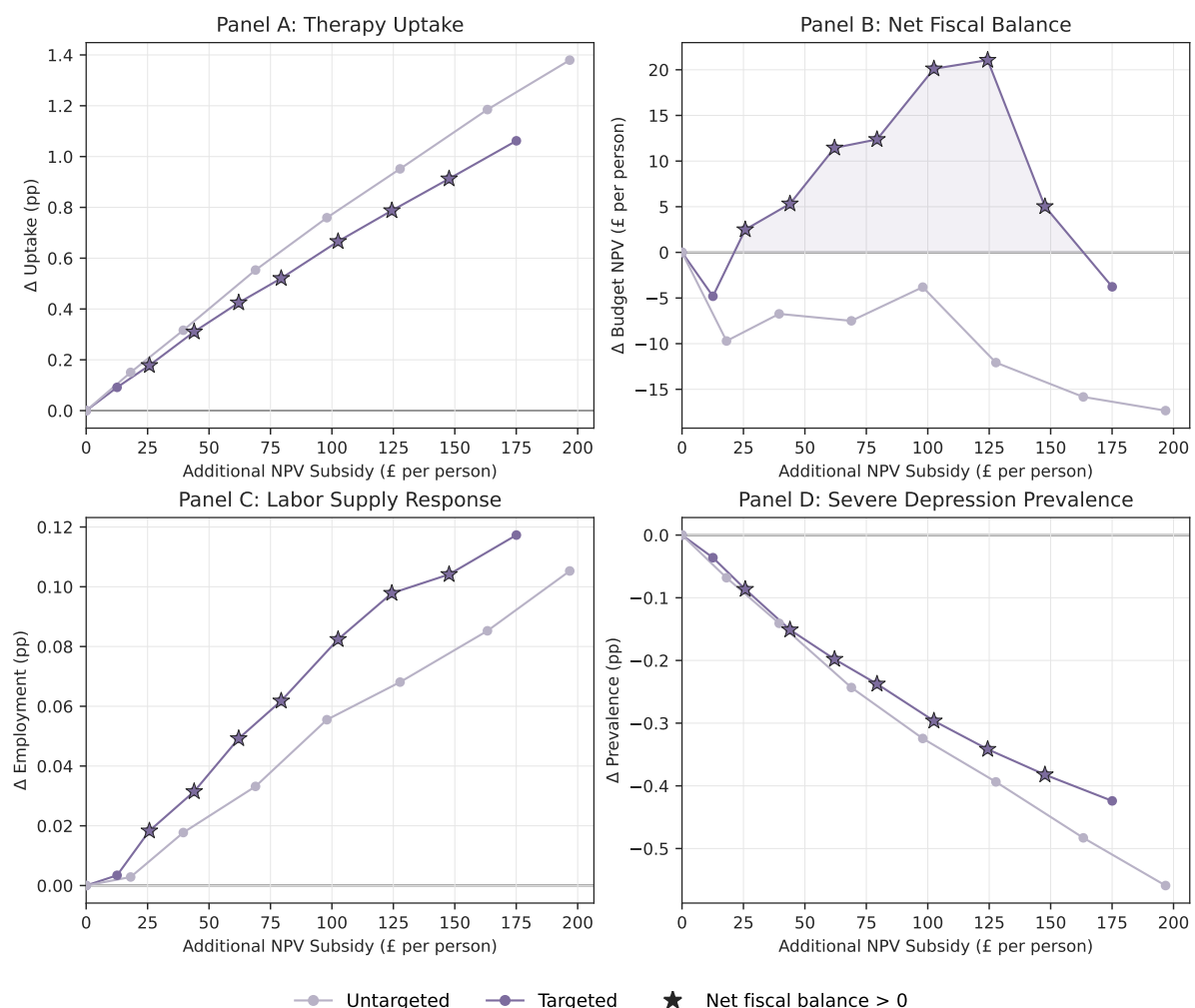
In the post-COVID environment, untargeted subsidies continue to generate a larger aggregate increase in therapy uptake compared to targeted interventions, although the gap between the two policies has narrowed (Panel A). Consistent with the baseline results, the higher overall uptake under the untargeted policy results in a more substantial reduction in the prevalence of severe depression for any given level of fiscal investment (Panel D). From a clinical perspective, universal subsidies remain a highly effective tool for lowering population-level morbidity, as they reach a broader segment of the population, including individuals who are nonetheless susceptible to relapse or recurrence.

⁵A concern is that the post-COVID exercise could reflect differential attrition rather than changes in behaviour. We therefore model attrition using a logit specification and test whether its correlates (including genetic risk and health) differ between the COVID and pre-COVID periods. Appendix Table 12 shows that we fail to reject the null of no differential attrition with respect to genetic risk or observed mental and physical health: the pre-COVID interaction terms for Genetic Risk, MH, and PH are all statistically indistinguishable from zero. Wealth is the main predictor of attrition in both periods.

Despite the clinical advantages of universal subsidies, targeted interventions exhibit clear fiscal dominance in the post-COVID scenario. As shown in Panel C, targeted subsidies generate a markedly stronger labor supply response, with employment gains significantly exceeding those of the untargeted policy across the investment distribution. This heightened responsiveness is reflected in the Net Fiscal Balance (Panel B), where targeted subsidies generate a substantial fiscal dividend, reaching a surplus of over £20 per person, a return that far exceeds the peak gains observed in the pre-pandemic baseline.

This shift is driven by the change in the initial state distribution; the post-COVID population features a higher concentration of individuals in the “depression trap”—the joint state of non-employment and poor mental health. Targeted subsidies are specifically calibrated to lower barriers for these high-risk individuals, whose recovery yields the largest fiscal returns through the dual mechanism of recovered tax revenue and the cessation of disability benefit payments.

Figure 14: Policy Frontier under Alternative Subsidy Schedules: Post-COVID Scenario



Notes: This figure plots policy frontiers for the COVID calibration. Each point corresponds to a subsidy schedule indexed by its implied additional net present value (NPV) subsidy cost on the horizontal axis (in £ per person), measured relative to the baseline untargeted policy at a 74% subsidy rate. Lines connect points within each policy regime: an untargeted subsidy (uniform across individuals) and a targeted subsidy (varying by risk group). Panel A reports the change in therapy uptake (percentage points). Panel B reports the change in the government budget NPV (in £ per person); shaded areas indicate combinations with positive net fiscal balance (above zero). Panel C reports the change in employment (percentage points). Panel D reports the change in the prevalence of severe depression (percentage points). Star markers highlight policy points with positive net fiscal balance (Panel B > 0), and the same points are marked with stars in the other panels for comparison.

The comparison between the baseline and post-COVID simulations reveals an evolving trade-off for policymakers. While untargeted spending remains the superior instrument for maximizing aggregate mental health improvements, targeted spending has become significantly more cost-effective in the wake of the pandemic. In the current landscape, the higher marginal returns from re-integrating high-risk individuals into the workforce mean that targeted subsidies not only mitigate the “depression trap” more effectively but also offer a self-financing path toward improving national labor supply and the government’s budget balance.

Overall, our structural results suggest that targeted therapy subsidies are a promising and cost-effective instrument for disrupting the “depression trap” and improving long-term labor market outcomes. While our current framework generates therapy utilization almost exclusively among those in a state of severe depression, the ex-ante observability of genetic risk presents a significant opportunity for preventative intervention. One potential advantage of early-life genetic screening is the mitigation of the high non-pecuniary costs associated with treatment-seeking. Just as depressive episodes increase the disutility of labor in our model, they likely increase the psychological burden of attending therapy. Encouraging uptake among high-risk individuals before the onset of clinical symptoms may therefore be more effective than attempting to induce entry once a patient has already entered a persistent non-employment spell.

However, the implementation of genetic screening is not without risk. Informing an individual they have a high genetic risk for depression could help them overcome it: “This is not your fault, you have a genetic vulnerability and our treatment strategy can overcome this” (Inglis et al., 2017). However, it could also hinder recovery, or even lead to the onset of depression, insofar as it is interpreted as deterministic and impossible to overcome. Study participants falsely told that they had a high genetic risk for depression reported higher levels of depressive symptoms (Lebowitz and Ahn, 2017) and reduced confidence about their ability to handle depression. This reduced confidence can, however, be fully mitigated by educating participants on the non-deterministic nature of genes’ effect on depression (Lebowitz and Ahn, 2018). This suggests the importance of developing and implementing an evidence-based model for psychiatric genetic counseling (Austin, 2020). More broadly, embedding genetic risk assessment within routine preventive care — rather than as a standalone psychiatric screen — would both mitigate the psychological costs of identification and enable timely intervention at symptom onset, a primary determinant of the clinical and economic effectiveness of programs like IAPT (Clark, 2011).

7 Conclusion

In this paper, we show that genetic predisposition to depressive symptoms strongly predict lifetime mental health and economic outcomes. Men with a high genetic risk for depression are 12 p.p. more likely to be depressed, 5 p.p. less likely to be employed and earn 0.15 s.d. less labor income. These results are robust to the inclusion of controls for socioeconomic background and broadly consistent with within-family estimates. We identify the “depression trap”, a self-reinforcing cycle of depression and non-employment, as a key mechanism explaining this disparity in labor market outcomes. Men with a high genetic risk for depression are more likely to enter the trap and less likely to leave it.

In this paper, we estimate a rich life-cycle model of mental health, benefits, and labor supply. The model incorporates endogenous treatment decisions and physical health dynamics. We find that latent genetic risk strongly influences life-cycle trajectories. Holding initial conditions fixed, genetic endowment drives inequalities in lifetime earnings, employment, asset accumulation, and overall health. Crucially, high genetic risk delays recovery from health shocks. It also transforms temporary wage losses into a persistent “depression trap”. We also evaluate targeted policy interventions. Specifically, we simulate paying high-risk men to attend psychotherapy. This policy improves both clinical and labor market outcomes. Notably, it achieves these gains at zero net cost to the government. These targeted subsidies prove exceptionally cost-effective in the post-COVID-19 era.

Our analysis builds on recent advances in psychiatric genetics (Baselmans et al., 2019). It highlights the untapped potential of Polygenic Indices to inform cost-effective precision public health strategies. Currently, PGIs explain only about 5% of the variation in depressive symptoms within our sample. Because of this, our estimates likely represent a lower bound of the true impact of genetic risk. Yet, even at this lower bound, the life-cycle effects and policy responses we uncover are non-trivial. Furthermore, the predictive power of GWAS is rapidly increasing. As larger-scale databases and higher-quality sequencing become available, genetic predictors could theoretically capture up to 40% of the variation in depression (Sullivan et al., 2000; Wainschtein et al., 2025). The UK National Health Service already plans to offer whole-genome sequencing to all newborns over the next decade (Davies, 2025). As these predictive tools mature, using ex-ante genetic screening to keep vulnerable individuals out of the “depression trap” will become a highly practical policy option.

The data and scope of this paper limits the applicability of our findings: we focus on the working-age men as we have very few observations below age 25 and to avoid modelling fertility decisions. Future research could use data focussed on early childhood development to explore in more detail the mechanisms by which genetic risk for depression affects the development of mental health and human capital. Early-life, targeted mental health interventions could plausibly be even more cost-effective, insofar as they could improve educational and occupational attainment. Follow-up research could also examine how genetic risk for depression shapes the career and mental health outcomes of women: women suffer from depression at higher rates than men (IHME, 2025) and have more elastic labor supply (Keane, 2011). This could interact with fertility decisions, as developing depression following fertility-induced labor supply reductions could make it more difficult to return to work. Furthermore, our analysis focusses on white men with european-like ancestry, despite the fact that ethnic minorities suffer from mental health issues at higher rates in the UK (Nazroo and Iley, 2011). This is due to data limitations, both in our discovery and target samples and due to the limited portability of PGIs across ancestry groups (Kim and Milliken, 2018). As more data becomes available, and better methods are developed for improving the portability of PGIs, exploring the role of genetic risk for depression in shaping the mental health and labor market outcomes of minority ethnic groups will be of increasing research interest.

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Appendices

A Genetic Data and Polygenic Indices

We summarize genetic predisposition using polygenic indices (PGIs) constructed from common genetic variants. The raw genotype data record variation at single-nucleotide polymorphisms (SNPs), which are genomic locations where individuals differ by a single DNA base. At each SNP j , an individual carries two alleles, one inherited from each biological parent. We code the genotype as an allele count $SNP_{i,j} \in \{0, 1, 2\}$, which equals the number of effect alleles carried by individual i at SNP j .

GWAS provide the weights used to aggregate SNPs into a scalar index. In a GWAS discovery sample, indexed by k , the outcome of interest Y_k is regressed on each SNP separately:

$$Y_k = \beta_j^{GWAS} SNP_{k,j} + \gamma \mathbf{X}_k + u_{k,j}, \quad (19)$$

where $\mathbf{X}_k$ includes standard controls such as sex, age polynomials, and genetic principal components to address population structure. The estimated SNP coefficients $\hat{\beta}_j^{GWAS}$ are then applied to individuals in our UKHLS analysis (target) sample, indexed by i , to form the PGI:

$$PGI_i = \sum_{j=1}^J \hat{\beta}_j^{GWAS} SNP_{i,j}. \quad (20)$$

Consistent with evidence that predictive performance improves when many variants are included, we construct the index using genome-wide SNP information rather than restricting to genome-wide significant hits (Okbay et al., 2016).

The UKHLS genetic subsample includes blood and saliva genotypes for 9,961 respondents. The released data contain 24,214,238 SNPs (hg38 build). After standard quality control, 9,920 individuals remain for analysis. We construct PGIs using PLINK2 and a standard workflow following Choi et al. (2020), combining UKHLS genotypes with publicly available GWAS summary statistics. In addition to the depressive-symptoms PGI used in the main analysis, we also construct PGIs for a broader set of traits for robustness and controls, including educational attainment, health, and related outcomes.

A.1 Measurement Error in PGIs and a Rank-Preserving Correction

Equation (20) uses estimated GWAS weights. These weights differ from the underlying effects due to sampling variation. Let the latent (population) index be

$$PGI_i^y = \sum_{j=1}^J \beta_j^y SNP_{i,j}. \quad (21)$$

Then the constructed index can be written as

$$\widehat{PGI}_i^y = PGI_i^y + \varepsilon_i^y, \quad (22)$$

where $\varepsilon_i^y = \sum_{j=1}^J (\widehat{\beta}_j^y - \beta_j^y) SNP_{i,j}$ captures measurement error in the estimated weights. Under standard conditions, this error is approximately classical (Becker et al., 2021). In our application, we often work with standardized or discretized PGIs. Measurement error can still matter because it may change an individual's rank in the cross-sectional distribution.

To limit rank misclassification, we follow the split-sample strategy in DiPrete et al. (2018) and Bolt et al. (2025) when suitable GWAS summary statistics are available. The key idea is to construct two PGIs for the same trait from non-overlapping discovery samples, so that their measurement errors are independent. Let $\widehat{PGI}_A$ and $\widehat{PGI}_B$ denote the two estimates, based on discovery samples A and B . We write

$$\widehat{PGI}_A = \alpha_1 PGI + \varepsilon_A, \quad (23)$$

$$\widehat{PGI}_B = \alpha_2 PGI + \varepsilon_B, \quad (24)$$

where PGI is the latent index and $(\varepsilon_A, \varepsilon_B)$ are independent and orthogonal to PGI . We then project one estimate onto the other:

$$\widetilde{PGI} = \frac{\text{Cov}(\widehat{PGI}_A, \widehat{PGI}_B)}{\text{Var}(\widehat{PGI}_A)} \cdot \widehat{PGI}_B. \quad (25)$$

This projection rescales the index and reduces the contribution of independent noise. It is therefore better aligned with the rank ordering of the latent PGI .

A.2 UKHLS Genetic Sample

We use data from Understanding Society: the UK Household Longitudinal Study (UKHLS). UKHLS is a nationally representative panel with rich information on income, employment, education, health, and well-being. A genetic subsample collected blood and saliva from 9,961 respondents. Genotypes are available on 24,214,238 SNPs (hg38 build). After standard quality control, 9,920 individuals remain.

We construct PGIs using PLINK2 and a standard pipeline following Choi et al. (2020). We combine UKHLS genotypes with publicly available GWAS summary statistics. In addition to the depressive-symptoms PGI used in the main analysis, we construct PGIs for a wider set of traits for robustness and controls, including education, health, and related outcomes.

The genetic data can be linked to family members within UKHLS. This feature allows us to characterize genetic relationships within households and, when needed, study patterns consistent with assortative mating or intergenerational links. Table 4 reports sample sizes for the main variables and the linked-relative subsamples after quality control.

Table 4: Sample Size by Information Availability

Sample Category	N
<i>Core Analysis Sample</i>	
Full genetic sample (post-QC)	9,920
Physical Health	9,920
Mental Health	9,888
Educational attainment	9,915
labor force status	9,920
Benefits	9,920
Saving information	7,178
Therapy	7,299
<i>Linked Biological Relatives</i>	
Biological parent genotype linked	550
Biological child genotype linked	392
Biological sibling genotype linked	223

Notes: This table reports sample sizes for key variables after applying standard genetic quality control (QC) protocols. “Full genetic sample (post-QC)” refers to individuals with valid genomic data. “Linked” categories indicate successful matching of genotype records between the primary respondent and their biological relatives within the dataset.

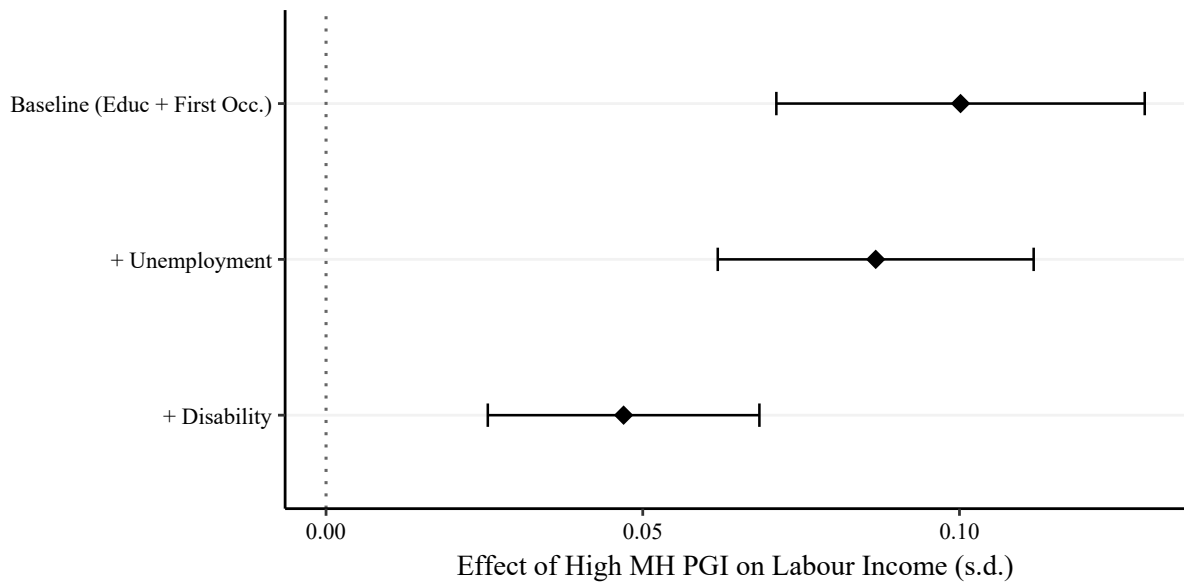
B Additional Empirical Results

Table 5: Mental Health PGI and Mental Health

Dependent Variable:	Mental Health (standardized)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
High MH PGI	0.31 (0.01)	0.30 (0.01)	0.30 (0.01)	0.30 (0.01)	0.30 (0.01)	0.28 (0.01)	0.29 (0.01)
<i>Controls</i>							
High EA PGI		✓	✓	✓	✓	✓	✓
Parental Occupation			✓	✓	✓	✓	✓
Parental Education			✓	✓	✓	✓	✓
Education				✓	✓	✓	✓
Occupation: First Job					✓	✓	✓
Employment						✓	✓
Current Occupation							✓
Observations	57,243	57,243	57,243	57,091	57,091	38,822	38,822

Notes: This table reports OLS regressions of standardized mental health (GHQ) on an indicator for having a mental health PGI above the median. All specifications include age, survey wave, and year fixed effects, as well as sex and the first 10 genetic principal components. Missing-value indicators are included for parental occupation and parental education. Standard errors are clustered and reported in parentheses.

Figure 15: Genetic Risk and Earnings: Decomposing the Role of Labor Force Status



Notes: This figure decomposes the role of current labor force status in accounting for the association between high mental health genetic risk and earnings. Each point reports the estimated coefficient on an indicator for being above-median in the mental health polygenic index (High MH PGI) across specifications that sequentially incorporate labor force status and its components. The decomposition shows that the attenuation attributed to labor force status is driven primarily by long-term sickness or disability rather than shorter-term unemployment. Standard errors are clustered at the household level.

Table 6: Descriptive Dynamics: Correlated Random Effects Logit Estimates

	Depression (t)	Employment (t)
	(1)	(2)
Employed (t-1)	-0.31 (0.10)	2.44 (0.12)
Depression (t)		-1.54 (0.09)
Depression (t-1)	0.48 (0.04)	-0.35 (0.10)
Earnings (t-1, £10k)	0.56 (0.18)	-1.03 (0.57)
Baseline Mental Health (Mean)	0.76 (0.01)	0.20 (0.02)
Baseline Employment (Mean)	0.43 (0.12)	7.10 (0.22)
Baseline Earnings (Mean)	-0.33 (0.21)	3.13 (0.76)
Age	0.04 (0.03)	0.16 (0.06)
Age ²	-0.00 (0.00)	-0.00 (0.00)
Number of Children	-0.01 (0.01)	-0.01 (0.03)
<i>Fixed Effects</i>		
Survey Wave	✓	✓
Year	✓	✓
Observations	46,681	46,681
Pseudo R ²	0.27	0.69

Notes: This table reports coefficients from a Mundlak-style correlated random effects (CRE) logit model. “Baseline” terms are within-person time averages of time-varying covariates, included to proxy individual unobserved heterogeneity. Column (1) models depression at time t and column (2) models employment at time t . Standard errors clustered at the individual level are in parentheses.

C Robustness: Within-Family Identification

A central challenge in identifying causal genetic effects in the genoeconomics literature is the correlation between an individual’s genotype and their developmental environment. Genetic variants may have a ‘direct genetic effect’, a causal effect on their mental health. However, ‘genetic risk for depression’ may instead reflect their environmental upbringing. In the literature, this is referred to as an ‘indirect genetic effect’ and occurs for two main reasons (Young et al., 2022). First, genetic variants may be more or less concentrated in certain areas of social classes. Norman ancestry remains a strong predictor of wealth in the UK a millennium following the conquest (Clark and Cummins, 2015). While we mitigate this in our primary analysis by controlling for the first 10 principal components of the genome (Price et al., 2006), residual confounding may persist. Second, ‘genetic nurture’ may occur as an individual’s genotype is inherited from their parents and thus mechanically correlated with their genetic predispositions (Kong

et al., 2018). A parent with a high genetic predisposition for depression may affect their child's risk of mental health issues through their interactions or the home environment they provide.

Many of the key conclusions of this paper do not require us to disentangle direct from indirect genetic effects. Even if our genetic associations purely capture indirect genetic effects, genetic variation remains a measurable, ex-ante predictor of depression risk, the persistence of the 'depression trap' and labor market outcomes. It may still be useful as a diagnostic tool in providing targeted mental health care, and doing so may be cost-effective in generating increased labor supply and reducing benefit payments. Nonetheless, it may suggest that rich socioeconomic indicators could be just as effective in targeting mental health interventions, and may have different implications regarding the design of mental health policy. If our results are closer to direct genetic effects, individuals in the same family may differ significantly in their risk profile and need for mental health care. We therefore aim to assess whether our key results reflect direct genetic effects by exploiting variation within family lineages.

C.1 Identification Strategy: Family vs. Sibling Designs

While family fixed effects in a between-sibling comparison provide the standard "lottery" of alleles within a shared household, the molecular genetic sample in the UKHLS is subject to significant attrition when restricted to genotyped sibling pairs. We identify only 153 siblings observed during working age with valid labor force status. When restricted to male sibling pairs, the resulting sample size is insufficient for meaningful statistical inference.

To maintain statistical power while adhering to causal identification standards, we expand our strategy to include all biological pedigrees. We identify 998 individuals connected by direct parent-child or sibling links, grouped into 538 unique family units. When we filter on all-male pedigrees, we are left with 418 men in 334 families. We identify the genetic effect by including family fixed effects, which purge the estimates of shared dynastic shocks and persistent social background factors. While variation between generations in a pedigree is influenced by assortative mating and environmental shifts, we include birth year fixed effects to capture cohort effects, and the stability of coefficients across these units provides a robust diagnostic for whether genetic nurture is a first-order driver of our results.

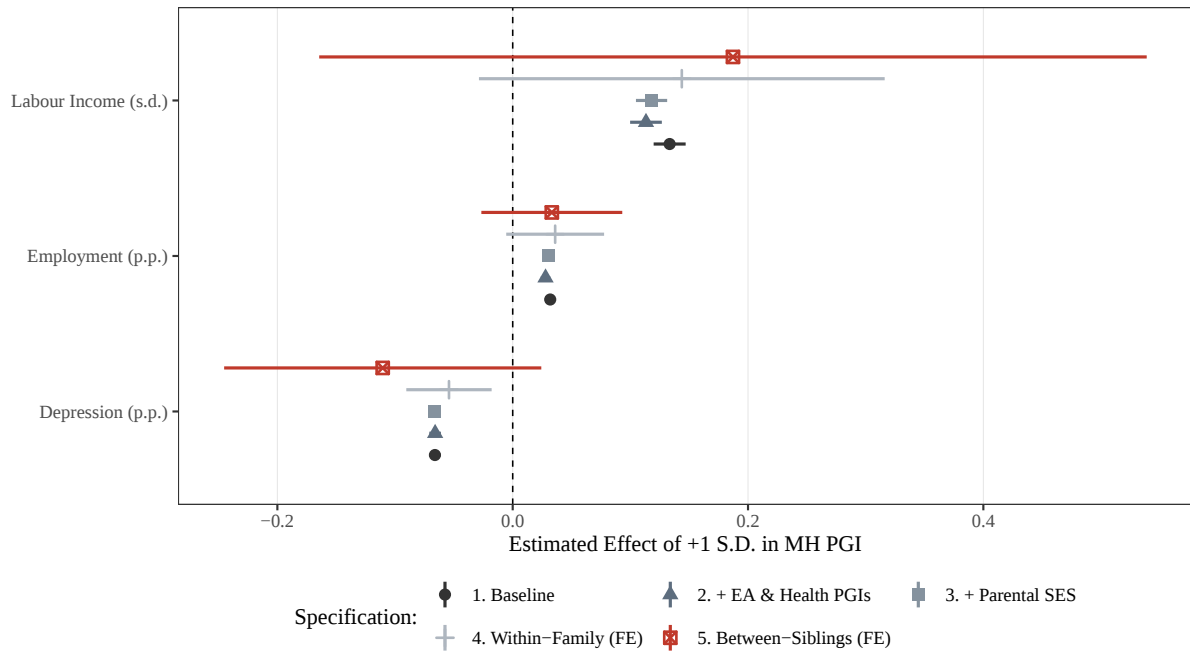
In these restrictive models, we transition from the binary measure used in our structural model to a continuous PGI to capture the smaller identifying variation within lineages. Furthermore, as the identifying variation within families is limited, we pool the sample and control for sex-genotype interactions to maximize power while maintaining a focus on the male "genetic penalty" established in Section 3. For employment and labor income, we report within-family results using the all-male family units.

C.2 Results and Coefficient Stability

Figure 16 displays the stability of the point estimates for our primary outcomes: labor income, employment, and clinical depression. To formally evaluate this stability, we perform a Z-test for the equality of coefficients between our socioeconomic mediation models (β_{med}) and these within-family models (β_{WF}).

As shown in Table 7, we fail to reject the hypothesis that the coefficients are statistically equivalent across all outcomes ($p > 0.50$). The stability of the point estimates—particularly for employment and income—suggests that the genetic penalty is rooted in individual genetic liability rather than purely environmental transmission or dynastic sorting. The point estimates in Figure 16 are also generally quite robust to inclusion of controls for socioeconomic background or EA or Health PGIs. For our main analyses, we therefore rely on between-family variation in MH PGIs for identification, which enables more comprehensive mediation analysis and dynamics. Given the robustness across specification, we generally interpret these as direct genetic effects. [Okbay et al. \(2022\)](#) test wellbeing, neuroticism and depression-related PGIs, comparing population-based estimates to within-family estimates with much larger sample sizes, and generally find consistent results.

Figure 16: Stability of MH PGI Estimates Across Specifications



Notes: This figure compares point estimates and 95% confidence intervals for the effect of a one standard deviation increase in the mental health polygenic index (MH PGI) across specifications. The “Socioeconomic Controls” specification includes parental occupation and parental education indicators. The “Within-Family” specification includes family fixed effects, identifying the association within extended family networks. Income is standardized; employment, disability, and depression are in probability units, with depression defined using GHQ-12 caseness (≥ 4). All models control for the first 10 genetic principal components and include age and year fixed effects. All within-sibling specification controls for sex and sex interacted with the MH PGI, and report the estimated coefficient for men. Within-family estimates are done comparing men for all outcomes except depression, which also includes sex and sex interacted with the MH PGI as a control. Standard errors are clustered at the household level in the socioeconomic-controls specifications and at the family level in the within-family specifications.

Table 7: Tests for Equality of Coefficients: Socioeconomic vs. Pedigree Specifications

Outcome	Socioeconomic Controls (β_{SES})	Family FE (β_{WF})	Z-stat	p-value (Diff)
Employment	0.03	0.04	0.12	0.91
Depression	-0.07	-0.05	0.67	0.50
Income (s.d.)	0.13	0.14	0.14	0.89

Notes: This table tests whether coefficient estimates differ across two specifications: one controlling for socioeconomic characteristics and one using family fixed effects (pedigree specification). The Z-statistic tests the null hypothesis $\beta_{SES} = \beta_{WF}$ for each outcome, and the final column reports the corresponding two-sided p-value.

D Estimation of individual heterogeneity with k-means clustering

Individuals have types x that take values on a discrete set $x \in \{1, \dots, K\}$. We estimate these types by building upon the approach of Bonhomme et al. (2022) (BLM) (see also Bonhomme et al. (2019)). BLM estimate individual heterogeneity classes by k -means clustering on relevant moments of outcome variables, with moments calculated at an individual level. Worker have types $x \in \{1, \dots, K\}$, there are individuals $i \in \{1, \dots, n\}$ and $\mathbf{m}_i$ is a vector of M moments of individual i 's outcomes. Workers are partitioned into K classes by finding the partition that solves

$$\min_{\{x_i\} \in \{1, \dots, K\}^N} \sum_{i=1}^N \|\mathbf{m}_i - \tilde{\mathbf{m}}(x_i)\|^2, \quad (26)$$

The consistency of BLM's approach relies on an injective map between individual unobserved heterogeneity and the vector of moments $\mathbf{m}_i$. In our application, productivity, mental health and physical health are the primary sources of individual heterogeneity. We therefore compute individual moments m using average wages, mental health status and physical health status.

In aiming to capture individual heterogeneity in productivity, we face an additional identification challenge. For a subset of individuals in our sample, we do not observe realized wages. These are the persistently non-employed, who we do not observe working, and retirees who we do not observe during their working lives. We address this challenge by generating a 'wage potential' (w_i^*) for each individual based on their ex-ante characteristics. We first estimate a log-wage regression on the subsample of employed workers⁶:

$$w_{it} = \gamma_{c(i)} + f(\text{Age}_{it}) + \delta_e \text{Educ}_i + \delta_o \text{Occ}_i + \delta_{pgi} \text{PGI}_i + \epsilon_{it} \quad (27)$$

where $\gamma_{c(i)}$ and $f(\text{Age}_{it})$ capture cohort fixed effects and a third-degree age polynomial, respectively. Educ_i and Occ_i represent educational attainment and first recorded occupation, while PGI_i includes indices for educational attainment and depressive symptoms. We calculate w_i^* for all individuals by predicting their log-wage at a fixed reference age (40) and anchoring them to a common cohort (1970):

$$w^i = \hat{\gamma}_{1970} + \hat{f}(40) + \hat{\delta}_e \text{Educ}_i + \hat{\delta}_o \text{Occ}_i + \hat{\delta}_{pgi} \text{PGI}_i \quad (28)$$

As we do not want our types to absorb variation attributable to genetic risk for depression, we residualize our vector of moments $\mathbf{m}_i$ on the depressive symptoms PGI. As in Jolivet and Postel-Vinay (2025), the data requires us to make an additional modification to the original BLM technique. In the UKLHS, individuals start the data observation period with differing ages, and we observe individuals for differing lengths of time. Estimating Equation 26 would lead to clustering based on cohort and age effects in addition to individual heterogeneity. We resolve this by first residualising physical and mental

⁶We filter out workers earning less than £1500 per year

health on birth-year dummies to remove cohort effects, and then adapting the partitioning to account for individual's ages:

$$\min_{\{x_i\} \in \{1, \dots, K\}^N} \sum_{i=1}^N \left[\sum_{H \in \{MH, PH\}} \|\bar{m}_{H,i} - g_H(x_i, Age_i)\|^2 + \|w_i^* - \bar{w}(x_i)\|^2 \right] \quad (29)$$

where Age_i is the average age of an individual through the periods observed and $g(1, \cdot), \dots, g(K, \cdot)$ are second-degree polynomials in age, aimed at capturing within-class moment differences due to age. The age polynomial is not included for the productivity moment, as it already accounts for age-effects. This approach mirrors [Jolivet and Postel-Vinay \(2025\)](#), who do not include cohort effects as they use the same observation window for all individuals.

D.1 Algorithm for the classification step

We present a simple iterative algorithm based on the one that has been developed by [Bonhomme and Manresa \(2015\)](#), which was in turn adapted by [Jolivet and Postel-Vinay \(2025\)](#) to implement their group fixed effect approach. Since this setting shares many features with theirs, we can use a similar, though slightly modified, algorithm for our classification step. The number of classes is set to $K \geq 1$.

1. Set $s = 0$ and let $\hat{k}_i^{(0)}$ be an initial partition.
2. In each class $k^{(s)}$ with at least two elements, regress by OLS each of the $M - 1$ individual moments (excluding w^i) on a constant, T, T^2 . Set the $g^{(s)}$ function parameters for this class to the resulting estimates. In each class with only one individual, set the $g^{(s)}$ slope parameters to zero and the constant to the value of the individual's moment.
3. If at least one class is empty:

- (a) Compute the “distance”

$$\sum_{H \in \{MH, PH\}} \|\bar{m}_{H,i} - g_H(x_i, Age_i)\|^2 + \|w_i^* - \bar{w}(x_i)\|^2$$

for each observation.

- (b) For each $k \in \{1, \dots, K\}$, if class $k^{(s)}$ is empty, assign to $k^{(s)}$ the observation with the highest distance and reset this distance to 0.
- (c) Do step 2 and replace the g_H function parameters with the new values.

4. Update the partition as follows:

$$k_i^{(s+1)} = \operatorname{argmin}_{k=1, \dots, K} \sum_{H \in \{MH, PH\}} \|\bar{m}_{H,i} - g_H(x_i, Age_i)\|^2 + \|w_i^* - \bar{w}(x_i)\|^2$$

5. If no observations changes class at Step 4 then stop. Otherwise, set $s = s + 1$ and go to Step 2.

As explained by Jolivet and Postel-Vinay (2025), this algorithm is a variant of one of the first heuristic clustering procedures, referred to as H-means algorithms. More precisely, a H-means algorithm would not include Step 3 and could thus terminate with empty classes. The addition of Step 3 overcomes this issue and leads to what is sometimes labelled a H-means+ algorithm. See (Brusco and Steinley, 2007) for more information about clustering algorithms.

E Model Estimates

Table 8: Estimated Health Transition Parameters

Parameter	Severe $\rightarrow$ Mild ($\mu_{S M}$)	Mild $\rightarrow$ Healthy ($\mu_{M H}$)
<i>Mental Health Transition Process (μ)</i>		
κ_{mh} : Intercept (Threshold)	-0.63	-0.24
$\mu_{1,1}$: Lagged Mental Health (Mild)	1.29	0.38
$\mu_{1,2}$: Lagged Mental Health (Healthy)	2.12	1.98
μ_3 : Employment (n_t)	0.20	0.11
$\mu_{4,1}$: Lagged Physical Health (Average)	-0.19	-0.19
$\mu_{4,2}$: Lagged Physical Health (Healthy)	-0.31	-0.31
$\mu_{5,g}$: Genetic risk (g_i)	0.54	0.43
$\mu_{5,t}$: Age (linear)	0.06	0.06
μ_{5,t^2} : Age (squared/100)	-0.07	-0.07
$\mu_{5,x,1}$: Health type 1	-0.13	-0.19
$\mu_{5,x,2}$: Health type 2	0.04	-0.07
$\mu_{5,x,3}$: Health type 3	-1.56	-1.61
$\mu_{5,x,4}$: Health type 4	0.37	0.19
Parameter	Poor $\rightarrow$ Average ($\pi_{P A}$)	Average $\rightarrow$ Healthy ($\pi_{A H}$)
<i>Physical Health Transition Process (π)</i>		
κ_{ph} : Intercept (Threshold)	1.18	2.76
$\pi_{1,1}$: Lagged Physical Health (Average)	1.42	0.78
$\pi_{1,2}$: Lagged Physical Health (Healthy)	2.28	2.07
$\pi_{2,1}$: Lagged Mental Health (Mild)	0.27	0.22
$\pi_{2,2}$: Lagged Mental Health (Healthy)	0.48	0.31
π_3 : Employment (n_t)	0.14	0.10
$\pi_{4,g}$: Genetic risk (g_i)	-0.34	-0.34
$\pi_{4,t}$: Age (linear)	-0.00	-0.00
π_{4,t^2} : Age (squared/100)	0.02	0.02
$\pi_{4,x,1}$: Health type 1	2.79	2.50
$\pi_{4,x,2}$: Health type 2	0.86	0.85
$\pi_{4,x,3}$: Health type 3	0.85	1.15
$\pi_{4,x,4}$: Health type 4	1.75	1.72

Notes: This table reports point estimates from partial proportional odds health transition models. Coefficients enter the latent health indices for mental health (mh^*) and physical health (ph^*); positive values indicate a higher probability of transitioning to a better health state. Parameters that take the same value across the two cutoffs within a process satisfy the proportional odds restriction. Health type 5 is the omitted (reference) category for health-type indicators. Coefficient subscripts follow the numbering used in the model equations 4 and 5.

Table 9: Survival Probability Parameters and Calibration Summary

Panel A: Logit Model (Pr(Death))			Panel B: UK Life Table Calibration		
Variable	Estimate	(S.E.)	Age	Shift (α_t)	Target (f)
ξ_0 : Intercept	-28.55	(22.07)	65	0.10	1.25%
ξ_1 : MH Healthy	49.64	(24.09)	70	0.36	2.15%
ξ_2 : MH Mild	27.67	(32.27)	75	0.38	3.46%
ξ_3 : PH Healthy	-16.20	(25.15)	80	0.38	5.98%
ξ_4 : PH Average	-7.64	(25.48)			
ξ_5 : Age	0.68	(0.61)			
ξ_6 : Age ² /100	-0.43	(0.42)			
<i>Interactions</i>					
MH Healthy $\times$ Age	-1.42	(0.66)			
MH Healthy $\times$ Age ² /100	0.99	(0.45)			

Notes: Panel A reports logit coefficients for the probability of death in the next wave, estimated on the retired UKHLS sample, with standard errors in parentheses. Panel B summarizes the life-table calibration: α_t is an age-specific shift applied to the latent index so that model-implied mortality matches the target rate f at each listed age.

Table 10: External Calibration: Benefits, Pensions, Taxes, and Therapy Costs

Panel A: Benefit Schedule ($b(mh, ph)$)				Panel B: Retirement & Therapy	
MH State	PH: Healthy	Average	Poor	Variable	Value (£)
Healthy	5,128	6,179	8,589	Pension (£)	
Mild	5,651	6,627	9,268	Type 1	23,438
Severe	5,889	8,433	10,126	Type 2	19,437
<i>Values in £ per annum</i>				Type 3	15,067
				Type 4	15,980
				Type 5	14,951
Panel C: Progressive Income Tax Schedule ($\tau(w)$)				Therapy Cost	
0% rate (personal allowance)			£0–£6,745	Unit Cost (£)	97.82
20% basic rate			£6,745–£37,400	Sessions	8.60
40% higher rate			> \$37,400	Private Share	0.26
				Total (P_τ)	218.63

Notes: This table summarizes external inputs used to calibrate the model. Panel A reports mean annual benefit receipt by mental health state (mh) and physical health state (ph). Panel B reports mean annual pension income by unobserved type and the components used to calibrate the out-of-pocket therapy cost P_τ . Panel C reports the progressive income tax schedule applied to labor earnings. All pecuniary amounts (benefits, pensions, and therapy costs) are expressed in 2015 £.

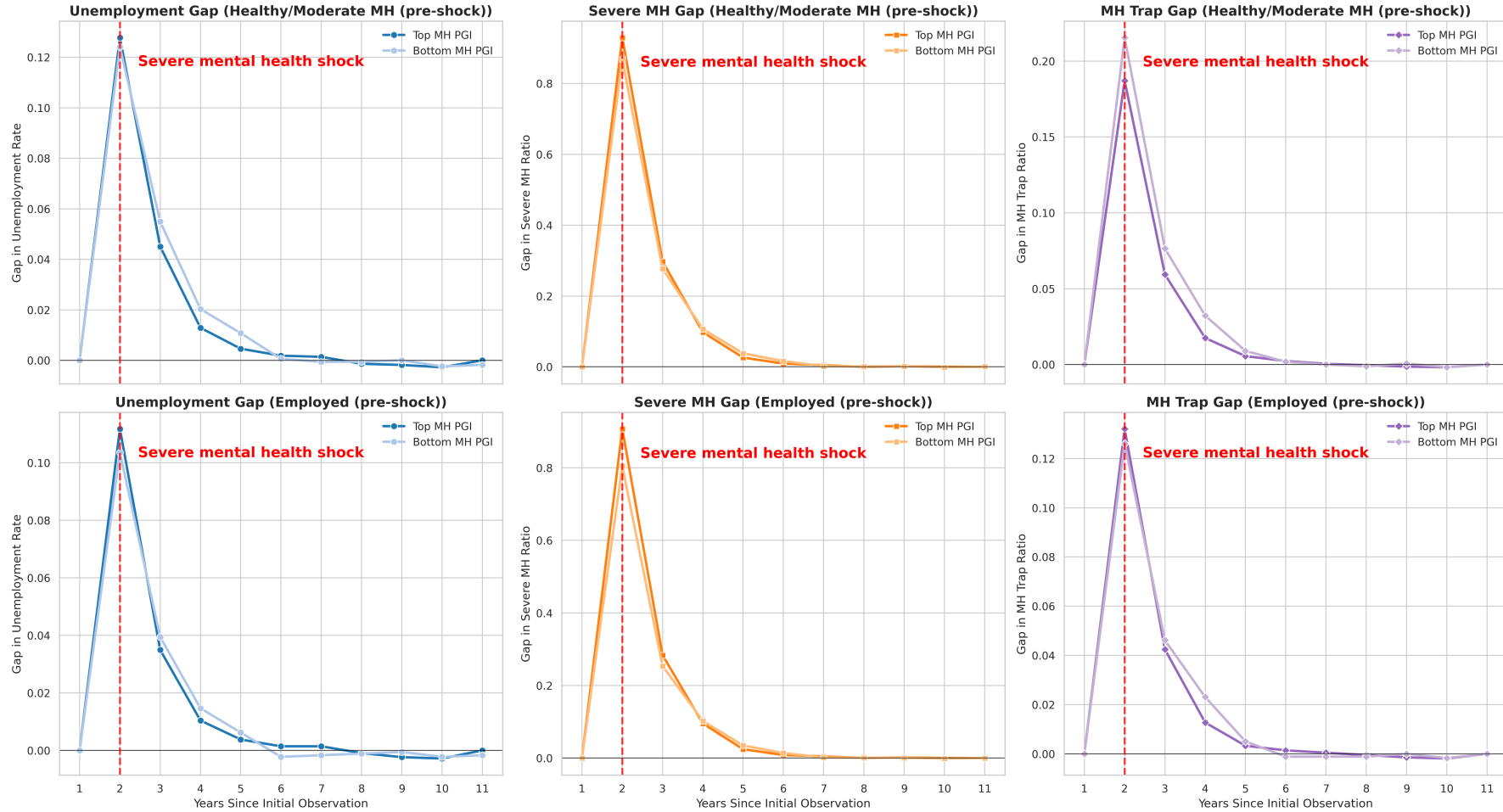
Table 11: Estimated Structural Parameters

Parameter	Estimate
<i>Preferences</i>	
β : Discount factor	0.95
γ : Consumption share of utility	0.53
<i>Wage Process</i>	
β_1 : Wage intercept	-0.06
β_g : Genetic return	0.07
$\beta_{2,1}$: Type 1 return	0.45
$\beta_{2,2}$: Type 2 return	0.52
$\beta_{2,3}$: Type 3 return	0.21
$\beta_{2,4}$: Type 4 return	0.15
$\beta_{3,1}$: Mental health return (Good)	0.15
$\beta_{3,2}$: Mental health return (Average)	0.14
$\beta_{4,1}$: Physical health return (Good)	0.21
$\beta_{4,2}$: Physical health return (Average)	0.02
β_5 : Age (linear)	0.04
β_6 : Age (squared)	-0.07
ρ : Wage persistence (AR1)	0.89
σ_η : Wage shock std. dev.	0.41
<i>Disutility of Labor</i>	
$\delta_{MH,Good}$: Disutility of labor (Good MH)	0.02
$\delta_{MH,Avg}$: Disutility of labor (Average MH)	0.05
$\delta_{MH,Bad}$: Disutility of labor (Severe MH)	0.08
$\delta_{PH,Good}$: Disutility of labor (Good PH)	0.03
$\delta_{PH,Avg}$: Disutility of labor (Average PH)	0.06
$\delta_{PH,Bad}$: Disutility of labor (Severe PH)	0.07
<i>Treatment Costs</i>	
ψ_{emp} : Therapy utility cost (Employed)	0.03
ψ_{unemp} : Therapy utility cost (Non-employed)	0.02

Notes: This table reports point estimates from the structural model. Health and labor-type returns are measured relative to omitted baseline categories in the log wage equation.

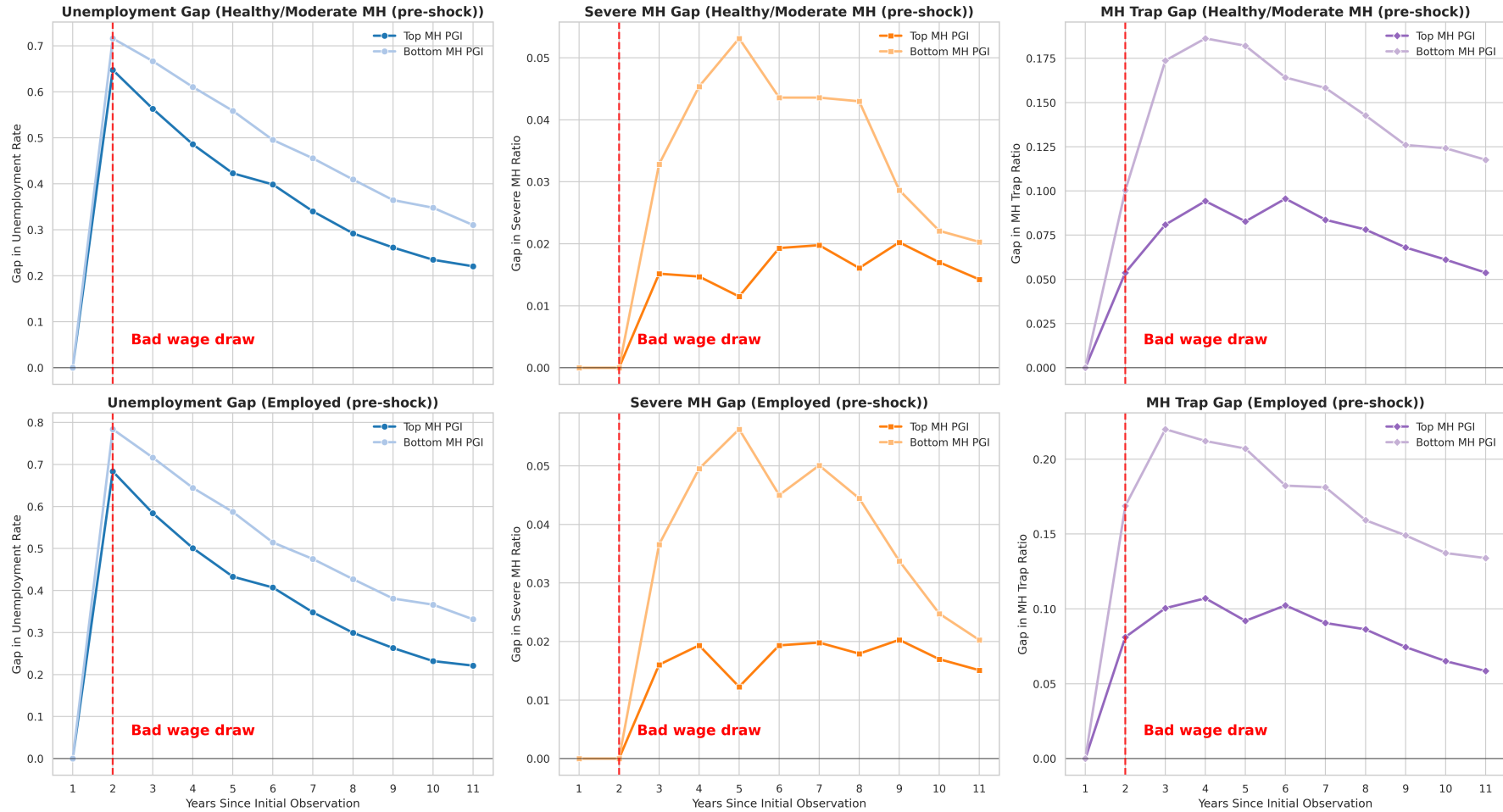
F Additional Counterfactual Results

Figure 17: Dynamics after a Severe Mental Health Shock by MH PGI Group



Notes: This figure extends Panel A of Figure 12. We start from the empirical initial distribution and select individuals first observed between ages 25–45. At the beginning of the year following an individual's first observation (Year 2), we exogenously force that individual into the severe mental health state, holding all other characteristics fixed, and simulate outcomes over the subsequent 10 years. Columns report (i) the full sample, (ii) individuals in healthy or moderate mental health prior to the severe mental health shock, and (iii) individuals employed prior to the severe mental health shock.

Figure 18: Dynamics after a Bad Wage Draw by MH PGI Group



Notes: This figure extends Panel B of Figure 12. We start from the empirical initial distribution and select individuals first observed between ages 25–45. At the beginning of the year following an individual's first observation (Year 2), we impose a bad wage draw by assigning the wage realization to the lowest of four discretized wage states. Under our discretization, this corresponds to a wage draw three standard deviations below the mean (approximately the bottom 0.14% of draws for individuals with the same characteristics). We then simulate and track employment and mental health over the subsequent 10 years (Years 1–11), separately for the Top and Bottom MH PGI groups. Columns report (i) the full sample, (ii) individuals in healthy or moderate mental health prior to the bad wage draw, and (iii) individuals employed prior to the bad wage draw.

Table 12: Logistic Regression: Predictors of Attrition (COVID vs. Pre-COVID Comparison)

Variable	Estimate	Std. Error	z-value	Pr(> z)
<i>Main Effects (COVID Wave 22→26)</i>				
(Intercept)	0.21	0.20	1.05	0.29
Wealth (per £10,000)	-0.03	0.01	-3.10	0.00
Genetic Risk (High DEP G)	-0.11	0.10	-1.15	0.25
Health Type 4	0.06	0.17	0.36	0.72
Health Type 3	0.13	0.24	0.53	0.60
Health Type 2	-0.19	0.16	-1.19	0.23
Health Type 1	-0.41	0.19	-2.21	0.03
PH: Average	-0.25	0.15	-1.72	0.09
PH: Healthy	-0.24	0.15	-1.57	0.12
MH: Mild	-0.21	0.21	-0.98	0.33
MH: Healthy	-0.13	0.17	-0.77	0.44
<i>Interactions (Pre-COVID Difference)</i>				
Period: Pre-COVID (15→22)	-0.24	0.63	-0.38	0.70
Period × Wealth (£10,000)	-0.02	0.04	-0.46	0.65
Period × Genetic Risk	0.11	0.23	0.46	0.65
Period × Health Type 4	0.32	0.44	0.72	0.47
Period × Health Type 3	-0.95	0.63	-1.52	0.13
Period × Health Type 2	-0.08	0.46	-0.17	0.87
Period × Health Type 1	0.15	0.48	0.31	0.76
Period × PH: Average	0.14	0.53	0.26	0.80
Period × PH: Healthy	-0.20	0.53	-0.39	0.70
Period × MH: Mild	0.37	0.48	0.77	0.44
Period × MH: Healthy	0.17	0.36	0.47	0.64
Model Diagnostics	AIC: 3,011 Null Deviance: 3,028.20 Residual Deviance: 2,967 $n = 2,245$			

Notes: This table reports logit estimates for attrition. The main-effects block uses the COVID comparison period (Wave 22→26). The interaction block reports how coefficients differ in the pre-COVID period (Wave 15→22) relative to the COVID period. Health Type 5 is the omitted (reference) category for the health-type indicators. For physical health (PH) and mental health (MH), the omitted (reference) categories are the non-listed baseline groups (i.e., neither “Average” nor “Healthy” for PH, and neither “Mild” nor “Healthy” for MH). Reported values are coefficient estimates, standard errors, z -statistics, and two-sided p -values.