FAQ's about "In and out unemployment – labour market transitions and the role of testosterone"

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This document provides information about the study:

Eibich et al. (2022). "In and out unemployment – labour market transitions and the role of testosterone" *Economics & Human Biology.*

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It draws from on the FAQs from Becker et al. (2021). "Resource Profile and User Guide of the Polygenic Index Repository" in *Nature Human Behaviour*.

For clarifications or additional questions, please contact Peter Eibich (eibich@demogr.mpg.de).

Quick Links

1. Background

1.1 Who conducted this study?

This research is conducted by a team of international researchers from different public research institutions. Peter Eibich and Julian Schmied are affiliated with the Max-Planck-Institute for Demographic Research in Germany; Ricky Kanabar is based at the University of Bath in the UK; and Alexander Plum is affiliated with the New Zealand Work Research Institute at the Auckland University of Technology.

1.2 What are the aims of the study?

We aim to improve scientific understanding of the factors that increase the risk for transitions into and out of unemployment. The authors have no financial interests in the results of this study, and there are no other conflicts of interest.

1.3 Who provided funding for this study?

Ricky Kanabar received funding from the University of Bath International Research Funding Scheme (Developing Networks in Europe, Grant ref: VB-SP3ARK) to support international collaboration activities for this study. The authors did not receive any further external funding for this study.

1.4 What is the research question and why is that relevant?

In general, we want to understand to what degree the testosterone level of British men can impact their labour market success. We are particularly interested in two groups: (i) does testosterone affect the likelihood of the employed in transiting into unemployment, and (ii) does testosterone levels impact unemployed men's chances to find a new job.

We know from previous studies that testosterone is related to personality traits and types of behaviour that plausibly might affect a person's risk to become unemployed. However, previous studies have only considered whether testosterone is related to a person's likelihood to work, and not whether it affects the transition from work to unemployment and vice versa. Our findings have important implications for labour market policy. They demonstrate that latent biological processes can affect job search behaviour and labour market outcomes without necessarily relating to illness and disability.

2 Data and Study Design

2.1 Where does the data for this study come from?

We use data from the UK Household Longitudinal Study (UKHLS). The UKHLS is an annual survey of households in the UK. The survey respondents are broadly representative of the wider UK population.

UKHLS participants are interviewed every year. For this study, we use data from the second, third and fourth waves of interviews conducted between 2010/11 (wave 2) and 2013/14 (wave 4). In addition, we use data from the "Health and Biomarker Survey" (also referred to as the "nurse visit" in the study). This is an additional survey wave of the UKHLS study. Participants who had completed a recent face-to-face interview in wave 2 or wave 3, who lived in Great Britain, completed their interview in English and (for women) were not pregnant were invited to participate in this additional data collection. Participants in the Health and Biomarker Survey were visited by a qualified nurse, which undertook several physical examinations and took a blood sample from consenting participants.

2.2 Which participants are included in this study?

In this study, we considered data from men aged 25-64 (broadly considered as "working age"). We only considered men if they were either employed or unemployed in the most recent interview before the nurse visit and if they were likewise either employed or unemployed in the next interview following the nurse visit (for 1,562 men these were waves 2 and 3, and for 553 men these were waves 3 and 4). We only included men with valid measures of testosterone that were taken between 8 a.m. and 8 p.m., and without missing information on age, their highest educational qualification, their self-rated health status, region of residence, household size, long-term disability status, legal marital status, % of body fat, smoking status, as well as whether they consumed beta-blockers or medication for the central nervous system. We require information on these characteristics because they have been identified in previous studies as factors that affect a person's testosterone levels or labour market success. Therefore, we include these characteristics in our statistical model to ensure that our estimate of the relationship between testosterone levels and labour market transitions are not affected by systematic differences in these characteristics.

2.3 Why are only men considered in this study?

Women are included in the Biomarker survey, and differences in labor market transitions are equally important for women. However, both the level and variation in the sample of tested women concerning testosterone is either very low or even undetectable to conduct a meaningful analysis.

2.4 Which labour market transitions are considered in this study?

We aim to understand how the individual's testosterone level impacts labour market changes between the nurse visit and the follow-up interview in the primary *Understanding Society* survey one year later.

We split our sample by individuals' reported labour market status at the time of the nurse visit, i.e. whether they are employed or unemployed. Then we analyse an individual's labour market position (unemployed or employed) at the first interview after the nurse visit.

2.5 How is testosterone measured?

Serum testosterone, the specific biomarker of interest for this study, was measured using an electrochemiluminescent immunoassay on the Roche Modular E170 analyser. Testosterone levels show wide variation among men and are considered within a normal range between 9-25 nmol/l.

Testosterone varies by time of day, such that values in the morning are higher than those found in the afternoon or evening. The level of testosterone also declines in age. To ensure comparability of testosterone, we adjust the circulating testosterone levels for age and time of the day when the blood sample was taken. We use the Health and biomarkers Survey to construct a sample of men with a positive level of testosterone in the age range 25 to 64 whose interview started between 8 am and 8 pm ($N = 3{,}597$). We form four age groups, spanning the following ages: 25-34, 35-44, 45-54, and 55-64. Next, for each age group, we estimate the diurnal change of testosterone by regressing the absolute level of testosterone (nmol/l) and controlling for the time difference of the nurse visit (hour and minute) to 10 am. We use the beta coefficients to correct the individual's testosterone level and standardise them to 10 am. Afterwards, we calculate the deviation by taking the difference between the estimated age-group specific and time-corrected sample mean and the individual's corrected testosterone level.

2.5 What study design is used to examine the effect of testosterone on labour market transitions.

Our primary aim is to understand how testosterone affects transitions to and from unemployment/employment. In order to do this, we use a probit regression. Such a framework is ideal given our dependent variable is dichotomous (employed or unemployed). Note that the labour market position refers to the first survey after the nurse visit, and we seek to understand the association with key sociodemographic and economic indicators, including testosterone. A limitation of using testosterone as an explanatory variable is that testosterone is not constant across the life-cycle and can also be affected by the labour-market position itself.

Thus, as a robustness check, we use the polygenic score provided by METADAC. To determine whether our main results hold, we use variation in genetic markers. These are known to be associated with testosterone and hence deal with potential endogeneity between testosterone measured at the time of nurse visit and observed employment status. Using such genetic markers is typically referred to as a Mendelian Randomisation approach. In this part of the analysis, we use an IV-probit regression framework that allows us to model our outcome of

interest (labour market status) in the presence of a potentially endogenous independent variable (testosterone) using the polygenic score as an instrument.

3 Genetic data

3.1 Why using genetic data?

We use genetic data to disentangle the effect of testosterone from the effect unemployment might have on testosterone. There is literature stating that being unemployed might affect testosterone levels. The genetic data is used to assess in a robustness test whether our primary findings are affected by reverse causality.

3.2 Where does the genetic data come from?

A genome-wide scan was conducted (Illumina human core exome array) on DNA provided by individuals who consented for their data to be collected. We have three genetic markers (rs12150660 and rs6258 in the SHGB gene on chromosome 17 and rs5934505 near FAM9B on the X chromosome), which are then linked to the individuals in the mainstage survey for whom we also have testosterone and sociodemographic information.

3.3 Who can access the genetic data and how?

Given the highly sensitive nature of the genetics data, access was provided by the METADAC secretariat, who judges and approves applications to use the UKHLS-linked genetics data for research purposes. Access to data is given to researchers who can show the research project and team meet all the criteria set out by the METADAC secretariat. More information can be found here.

3.4 What does Mendelian Randomisation mean, and how does it work?

Using genetic markers as instruments is typically referred to as a Mendelian Randomisation approach. Instead of using the three genetic markers separately in our regression model, we combine them into a single measure, the polygenic score. We use an IV-probit regression framework which allows us to model our outcome of interest (labour market status) in the presence of a potentially endogenous independent variable (testosterone) for which we instrument using the polygenic score.

3.4 What is a polygenic score (PGS)?

A polygenic score (PGS) is an index composed of several SNPs from across the genome. Each polygenic index is associated with a particular outcome. Because a polygenic index aggregates the information from many SNPs, it can "predict" far more of the variation among individuals than any single SNP. (Note that even polygenic indexes are not good predictors of outcomes for one person). The polygenic indexes with the most predictive power are often created using all the (millions of) SNPs measured in an SNP array. An SNP array is the current standard way of measuring common genetic differences across individuals. An SNP array data does not

measure the entire genetic sequence of each individual, but it does measure most of the places on the genome where individuals differ (see Becker et al. 2021)

3.5 How is a polygenic score constructed?

A polygenic score is constructed in three steps (see Becker et al 2021). First, a genome-wide association study (GWAS) is conducted, looking at SNPs measured across the entire human genome to see which of them are associated with higher or lower levels of some outcome. As explained above, SNPs are sites in the genome where single DNA base pairs commonly differ across individuals. SNPs usually have two different possible base pairs, or alleles. Although there are tens of millions of sites where SNPs are located in the human genome, GWASs typically investigate only SNPs that can be easily measured (or imputed) with a high level of accuracy. These days, we can quickly and accurately measure millions of SNPs, which together capture most of the common genetic variations across people. For each of these millions of SNPs, the GWAS generates an "effect size" corresponding to the (typically minuscule) magnitude of the association between that SNP and the outcome. (We use the term "effect size" because it is a common scientific shorthand for "magnitude of association". However, we emphasise that by using the term we do not intend to imply that the SNP, or polygenic index, causes the outcome)

3.6 Does the polygenic score "cause" unemployment?

No. The polygenic score does not 'cause' unemployment. Other factors are also likely to influence an individual's labour market position. Instead, we use the polygenic scores to support our primary analysis, which focuses on the relationship between testosterone and labour market transitions. Polygenic scores are useful in this context because they are associated with testosterone, which in itself may fluctuate for various reasons. The score is related to the 'stable' part of testosterone which does not change over time. So, the polygenic score allows us to check whether the 'stable' part of testosterone is related to labour market dynamics in a consistent way with our main findings. It is important to note that the three genetic markers we use only explain a small fraction of circulating testosterone.

4. Results and Implications

4.1 What are the main findings of this study?

The study shows that among unemployed individuals, those with medium and high testosterone levels are significantly more likely to leave unemployment compared to those with lower testosterone levels. In contrast, for our sample of employed men, we do not find significant effects of testosterone on the risk of entering unemployment.

4.2 How do you reconcile the findings from your main analysis with those findings using the genetic data?

The genetic data allow us to address endogeneity in testosterone levels. The results based on the genetic data are broadly supportive of our main findings. We find a negative effect of testosterone on unemployment risk, both among unemployed men and among employed men. Although we interpret the estimates from the Mendelian Randomisation as causal effects, the results should be interpreted with caution. Our sample size is limited, particularly for unemployed men, and our models likely lack statistical power. Moreover, the polygenic score only predicts a limited amount of variation in testosterone levels, which further reduces the precision of the IV estimates.

4.3 Do these findings mean that unemployed individuals should receive testosterone injections?

No. The analysis does not imply or recommend such an intervention. Instead, the findings highlight that latent biological processes can affect job search behaviour and labour market outcomes without necessarily relating to illness and disability.

4.4 What are the social and policy implications of these findings?

While we do not advocate injecting or determining the testosterone levels of unemployed men to improve their labour market outcomes, differences can still be taken into account. For example, it is crucial to recognise for employers and the unemployed that differences in labour market success are partly driven by biological processes outside the job seeker's control. For example, individuals with lower testosterone levels might benefit more from individual coaching rather than group sessions. Our results also suggest that individuals with high testosterone levels are at an advantage during the job search, although such hormonal differences do not necessarily translate into better productivity. In addition, awareness of the impact of personality and behavioural traits on performance during job interviews can potentially improve the quality of the job match.