Investigating genetic causal relationships between blood pressure and anxiety, depressive symptoms, neuroticism and subjective well-being

Lei Cai,1,2 Yonglin Liu,2 Lin He1,3

ABSTRACT

Background High blood pressure is a leading cardiovascular disease risk factor and considered to be associated with psychological factors. However, the causal relationships between blood pressure and anxiety, depressive symptoms, neuroticism and subjective well-being are not clear.

Aims The current study explored the genetic causal relationships between blood pressure and anxiety, depressive symptoms, neuroticism and subjective well-being.

Methods Mendelian randomisation (MR) analyses were performed using the generalised summary-data-based MR analysis method with eight large-scale genome-wide association study datasets for hypertension, systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse pressure, anxiety, depressive symptoms, neuroticism and subjective well-being.

Results A causal effect of DBP on neuroticism was found, and 1074 independent instrumental single nucleotide polymorphisms were identified by the incorporated Heterogeneity in Dependent Instruments-outlier test among the bidirectional causal relationship between blood pressure and the four psychological states.

Conclusions DBP has a causal effect on neuroticism. Appropriate management of blood pressure may reduce neuroticism, neuroticism-inducing mood disorders and cardiovascular diseases.

INTRODUCTION

According to the concepts of fluid mechanics, flowing blood exerts pressure against the blood vessels, inducing blood pressure (BP). BP is measured in millimetres of mercury (mm Hg) and expressed in terms of systolic BP (SBP, maximum pressure) representing the pressure in blood vessels when the heart contracts, and diastolic BP (DBP, minimum pressure) representing the pressure in the vessels when the heart rests between two continuous contractions. The human body is said to be in a state of hypertension when the SBP is >140 mm Hg and/or the DBP readings are >90 mm Hg on 2 continuous days. Hypertension affects more than a quarter of the global population1 and is a crucial risk factor in the development of whole-body disorders, particularly heart and circulatory diseases.2 Although the aetiology of hypertension is not fully understood, the comorbidity between hypertension and psychosocial and mental disorders has been investigated by several research groups. Nevertheless, the relationship between hypertension and psychosocial disorders remains unclear and is sometimes controversial.3

Anxiety is a feeling of fear, dread and uneasiness, which is a reaction to stress. Depressive symptoms include pessimism, negative affect, low energy, anxiety and bodily pains.4 Neuroticism is a personality trait characterised as being prone to experiencing negative emotions such as anxiety, fear, distress, dissatisfaction, depression, anger and guilt. Subjective well-being is measured by survey questions on life satisfaction, positive affect and happiness. A recent controversial systematic review and meta-analysis suggested an association between anxiety and increased risk of hypertension, based on the evidence.
from cross-sectional and prospective studies. While there is growing evidence of a relationship between psychosocial states and altered BP, the pathophysiological mechanisms underlying these relationships are unclear or contradictory. The causal effects of BP on anxiety, depressive symptoms, neuroticism and subjective well-being have not yet been elucidated.

Genome-wide association studies (GWASs) are powerful resources for identifying genetic variants contributing to a trait. There are GWASs that consider anxiety, depressive symptoms, neuroticism and subjective well-being, which can provide human genomic information about these psychological states. Mendelian randomisation (MR) analysis is currently a popular method to examine the bidirectional genetic causal effects between two traits based on GWAS summary data. This method can be used to explore the causal effects of BP on psychological states at the genetic level.

Therefore, this study aimed to explore the causal relationships between BP and anxiety, depressive symptoms, neuroticism and subjective well-being based on large samples of GWAS data.

METHODS AND MATERIALS

Study design

Bidirectional two-sample MR analyses were performed to explore the causal association between the exposure (a risk factor) and the outcome (a phenotype). Genetic variants associated with the exposure were selected as instrumental variables. For one direction of the MR analysis, the four BP traits (SBP, DBP, pulse pressure (PP) and hypertension) were treated as the exposure, and the four psychological states (anxiety, depressive symptoms, neuroticism and subjective well-being) were treated as the outcome. For the other direction of the MR analysis, this assignment was reversed. The study had three phases: (1) collecting summary-level GWAS data for the eight traits, (2) exploring genetic variants to serve as instrumental variables, and (3) estimating the causal effects of the exposure on the outcome.

Data collection and extraction

The flowchart of the current study is described in figure 1. First, the GWAS datasets from European populations for the four psychological states were obtained. The anxiety GWAS dataset was obtained from the MR-base database. The depressive symptoms GWAS dataset was obtained from the UK Biobank (UKB), Genetic Epidemiology Research on Adult Health and Aging (GERA), and Psychiatric Genetics Consortium (PGC). The neuroticism GWAS dataset was obtained from the UKB and Genetics of Personality Consortium (GPC). The subjective well-being GWAS dataset was obtained from a meta-analysis on summary association statistics from 59 cohorts. Subjective well-being was classified into four phenotypical panels: primary subjective well-being, life satisfaction, positive affect and post hoc subjective well-being.

GWAS data for DBP and SBP were retrieved from the study by Guo et al. The GWAS dataset for PP was included as PP has been proposed to describe the difference between SBP and DBP, and is viewed as an independent risk predictor for whole-body disorders (table 1). The hypertension GWAS dataset, with the largest sample size within the MR-base database, also included abnormal BP, defined as SBP >140 mm Hg and/or DBP >90 mm Hg on 2 continuous days. All participant samples in each
GWAS were of European ancestry. Ethical approval had been obtained in all original studies.

Detailed descriptions of participant characteristics can be found in each study. All participants were included in only one study. Genotyping was performed on genome DNA extracted from blood samples according to standard procedures from a range of commercially available genotyping arrays, as described in the original reports. Genotype imputation was performed using the 1000 Genomes Project reference panel and IMPUTE2 software. For each GWAS dataset, all biallelic single nucleotide polymorphisms (SNPs) and SNPs with an imputation score >0.9 were considered for the next analysis, while ambiguous SNPs were excluded. If an SNP was mapped to opposite strands in either dataset, its alleles in the second dataset were flipped.

**MR analysis**

MR analysis infers the credible causality of a relationship between the exposure and the outcome by leveraging instrumental variables, which are expected to be independent of confounding factors. In MR tests using GWAS data, genetic variants are treated as instrumental variables to test for causality. Due to the possibility of correlated or uncorrelated pleiotropy of genetic variants, exploiting GWAS data with large independent samples can greatly improve the power of an MR analysis. Genetic variants used as instrumental variables need to meet three assumptions; they should: (1) be associated with the exposure, (2) only affect an outcome via the exposure, and (3) be independent of confounders. The MR checklist for Strengthening the Reporting of Observational Study in Epidemiology is provided in the online supplemental materials.

The Genome-wide Complex Trait Analysis tool (V1.93.3 beta2) was used to explore bidirectional causal links between each psychological state and each BP trait in the framework of Generalised Summary-data-based MR (GSMR). This method is based on summary-level data, using independent genome-wide significant SNPs as instrumental variables, that is, an index of the exposure to test for putative causal associations between a risk factor (exposure) and an outcome. Instrumental variants were selected based on the default GWAS threshold of $p \leq 5 \times 10^{-8}$. An LD (linkage disequilibrium) threshold of $r^2 = 0.05$ was used to identify independent SNPs based on the European population as referenced within the 1000 Genomes Project (phase 3). Heterogeneity in Dependent Instruments (HEIDI)-outlier detection was used to filter genetic instruments that had obvious pleiotropic effects on the exposure and outcome. A threshold p value of 0.01 was used for the HEIDI analysis. We used an F-statistic $>10$ to define SNPs as valid instrumental variables. Ten was the minimum number of instrumental SNPs required. The power for the MR analysis was calculated using an online calculator (http://sb452.shinyapps.io/power/). P values were adjusted using the Bonferroni method, multiplying by 32 for multiple tests.

### RESULTS

The GWAS summary datasets obtained are listed in Table 1. The maximum sample size of BP traits was 736,650 for SBP, DBP, and PP, and the minimum was 463,010 for hypertension. The maximum sample size for psychological states was 463,010 for anxiety, and the minimum was 170,911 for neuroticism. There were no participants who appeared in both the BP and psychological state datasets.

With BP traits as exposure and psychological states as outcome, hypertension and DBP had significant causal effects on neuroticism ($p = 8.8 \times 10^{-6}$ and 0.026, respectively, Table 2). After adjusting for multiple tests, only DBP was significantly associated with neuroticism ($b_{x}=0.003\ 6$, Table 2; $p_{\text{bonferroni}} = 0.000\ 28$). There were 1074 independent instrumental SNPs, which were significantly associated with DBP but not with neuroticism (online supplemental table 1 and Figure 2). These instrumental SNPs, with F-statistic $>10$, were independent with an LD $r^2$ less than 0.05 and survived the HEIDI-outlier analysis that removes horizontal pleiotropic SNPs with $p < 0.01$. No significant causal effects were found for other BP traits and each psychological state.

<table>
<thead>
<tr>
<th>Traits</th>
<th>Sample size</th>
<th>SNPs</th>
<th>Reference (DOI)</th>
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<tr>
<td>Systolic blood pressure</td>
<td>736,650</td>
<td>7,070,522</td>
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<td>Diastolic blood pressure</td>
<td>736,650</td>
<td>7,142,798</td>
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<td>Pulse pressure</td>
<td>736,650</td>
<td>7,071,236</td>
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<td>Hypertension</td>
<td>463,010</td>
<td>9,851,867</td>
<td><a href="https://gwas.mrcieu.ac.uk/datasets/ukb-b-12493/">https://gwas.mrcieu.ac.uk/datasets/ukb-b-12493/</a></td>
</tr>
<tr>
<td>Anxiety</td>
<td>463,010</td>
<td>9,851,867</td>
<td><a href="https://gwas.mrcieu.ac.uk/datasets/ukb-b-11311/">https://gwas.mrcieu.ac.uk/datasets/ukb-b-11311/</a></td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>180,866</td>
<td>6,524,474</td>
<td>10.1038/ng.3552</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>170,911</td>
<td>6,524,432</td>
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<tr>
<td>Subjective well-being</td>
<td>298,420</td>
<td>2,268,675</td>
<td>10.1038/ng.3552</td>
</tr>
</tbody>
</table>

DOI, Digital Object Identifier; SNPs, single nucleotide polymorphisms.

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See Table 1 for a summary of European genome-wide association study data on various blood pressure, anxiety, depressive symptoms, neuroticism and subjective well-being traits.
The reverse causal effects analysis indicated that after clumping and HEIDI-outlier filtering of SNPs, less than the default threshold of 10 independent instrumental variants were retained in the analysis. Since anxiety, depressive symptoms, neuroticism and subjective well-being are complex traits, limited independent instrumental variants may provide biased results. However, without the limit of this threshold, no significant causal association of each psychological state with any BP trait was found. The power of our MR analysis in all pairs of exposure and outcomes was >90%, given the sample size for the relevant variant-outcome associations, at an alpha of 5%.

**DISCUSSION**

**Main findings**

As high BP is a leading risk factor for cardiovascular disease, the relationship between BP and psychological factors is undeniable. However, the causal effects between

<table>
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<th>Exposure trait</th>
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<th>bxy</th>
<th>SE</th>
<th>P value</th>
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<tr>
<td>DBP</td>
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<td>0.012 6</td>
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<tr>
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<td>−0.001 2</td>
<td>0.000 8</td>
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<tr>
<td>Hypertension</td>
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<td>0.697 7</td>
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<tr>
<td>PP</td>
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<td>SBP</td>
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<tr>
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<tr>
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<td>0.000 4</td>
<td>0.000 5</td>
<td>0.355</td>
<td>881</td>
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</table>

DBP, diastolic blood pressure; PP, pulse pressure; SBP, systolic blood pressure; SNPs, single nucleotide polymorphisms; SWB, subjective well-being.

*Figure 2*  The causal effects of DBP on neuroticism. The dotted lines denote effect sizes (bxy). DBP, diastolic blood pressure.
BP and anxiety, depressive symptoms, neuroticism and subjective well-being are not well distinguished. In this study, we have, for the first time, used GSMR analysis methods and found a causal effect of DBP on neuroticism.

In this study, MR use was based on the assumptions that genetic variants are associated with the exposure factor, are not related to confounding factors associated with the outcome, and must affect the outcome through the exposure factors. MR method is developed to infer the causal relationship between the exposure factors and outcome. Genetic variants from GWASs are usually treated as instrumental variants for their stability and randomness; however, they may cause horizontal pleiotropy where variants affect outcome and exposure traits via a shared heritable factor (correlated pleiotropy) or separate mechanisms (uncorrelated pleiotropy), other than a causal effect. The GSMR method is considered more powerful than other summary data-based MR approaches and incorporates the HEIDI-outlier test to identify loci that influence multiple phenotypes, such as pleiotropy effects on the exposure and outcome.

LIMITATIONS
Current large-scale GSMR analysis reduces the biases caused by confounding factors in observational studies, using random genetic variants as instrumental variables, which may represent lifelong influences and increase the precision of the analysis results. However, there are potential limitations of the current analysis: first, the incorporated HEIDI-outlier test in GSMR can identify loci affecting multiple phenotypes, such as pleiotropy effects on the exposure and outcome; nonetheless, the possibility of residual pleiotropy cannot be completely excluded. More methods are required to evaluate the independence of variants and analysis results. Second, current analyses were predominantly on populations of European ancestry, and therefore, the findings may not be generalisable to other populations. Third, a stringent Bonferroni correction was used to judge the positive MR findings, which may have caused false-negative findings and minimised the ratio of false-positive results.

Implications
BP is an important indicator of blood circulation and is one of the vital signs. BP has 30%–60% heritability, and over 1000 SNPs have been significantly associated with this complex trait by large-scale GWASs. Some psychological factors, such as mental stress and anxiety, may cause sudden high BP and increase blood fluidity by activating the sympathetic nervous system. Anxiety, anger and happiness increase BP, and emotional effects have greater variability in individuals with more labile BP. In pharmacological studies of hypertension, a reduction of BP in placebo groups is often found, which differs from spontaneous remission and regression to the mean effect of comparing placebo groups with untreated groups. The role of BP in psychosomatic medicine is implied as a link between the brain and the heart, and thus may promote the development of personality traits. Individuals with neuroticism can be sensitive to the criticism of others, often self-critical, and easily develop anxiety, anger, worry, hostility, self-consciousness, and depression. Neuroticism is viewed as a key causative factor for anxiety and mood disorders. Individuals with neuroticism more frequently experience high mental stress, which can lead to elevated BP and cardiovascular diseases. Thus, appropriate management of BP may reduce neuroticism, neurotism-inducing mood disorders and cardiovascular diseases.

In conclusion, using GWAS datasets with large sample sizes, we found that, among the causal relationship between BP and psychological states, DBP had a causal effect on neuroticism but not on the other psychological states of anxiety, depressive symptoms, or subjective well-being. Since the independent instrumental SNPs for these four psychological states are limited, future studies are required to explore the causal relationship between psychological states and BP.

Contributors
LC conceived and wrote the whole manuscript. LC and YL analysed the data. LC and LH proofread the manuscript. LC is the guarantor for the manuscript.

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Competing interests
None declared.

Patient consent for publication
Not required.

Ethics approval
This study involves human participants but the Bioethics Committee of Bio-X Institutes of Shanghai Jiao Tong University exempted this study. Participants gave informed consent to participate in the study before taking part.

Provenance and peer review
Not commissioned; externally peer reviewed.

Data availability statement
Data are available upon reasonable request.

Supplemental material
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REFERENCES
4 Okbay A, Baselmans BML, De Neve J-E, et al. Genetic variants associated with subjective well-being, depressive symptoms, and...

Lei Cai is an associate professor of Bio-X Institutes, Shanghai Jiao Tong University in China, and a member of Shanghai Jiao Tong University Institutional Review Board (IRB) for Human Research Ethics. He obtained his PhD degree from Fudan University, China, and has worked as a research assistant at the Harvard School of Public Health, USA. He has published over 50 papers and four chapters for three books, and has had two invention patents authorised. He has been the PI on several projects and research tasks, such as the National Natural Science Foundation of China, Shanghai, the Ministry of Education (MOE) Scientific Research Foundation, and the Key Research and Development Program of the Ministry of Science and Technology. His main research interests include the molecular basis of mind-body interactions and big data analysis.
STROBE-MR checklist of recommended items to address in reports of Mendelian randomization studies

<table>
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<tr>
<th>Item No.</th>
<th>Section</th>
<th>Checklist item</th>
<th>Page No.</th>
<th>Relevant text from manuscript</th>
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<tr>
<td>1</td>
<td>TITLE and ABSTRACT</td>
<td>Indicate Mendelian randomization (MR) as the study’s design in the title and/or the abstract if that is a main purpose of the study</td>
<td>1</td>
<td>(based on) Mendelian randomization analyses (were performed)</td>
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<td>2</td>
<td>INTRODUCTION</td>
<td>Explain the scientific background and rationale for the reported study. What is the exposure? Is a potential causal relationship between exposure and outcome plausible? Justify why MR is a helpful method to address the study question</td>
<td>4</td>
<td>A systematic review and meta-analysis on controversial results suggests that there is an association between anxiety and increased risk of Hypertension based on the evidence from cross-sectional and prospective studies at that moment</td>
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<td>3</td>
<td>Objectives</td>
<td>State specific objectives clearly, including pre-specified causal hypotheses (if any). State that MR is a method that, under specific assumptions, intends to estimate causal effects</td>
<td>4</td>
<td>the aim of this paper was to study causal relationships between blood pressure and anxiety, depressive symptoms, neuroticism and subjective well-being based on GWAS data with the large sample size.</td>
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<td>4</td>
<td>METHODS</td>
<td>Present key elements of the study design early in the article. Consider including a table listing sources of data for all phases of the study. For each data source contributing to the analysis, describe the following:</td>
<td>4</td>
<td>the GWAS datasets on European population for four psychological states, i.e. anxiety, depressive symptoms, neuroticism and subjective well-being were collected respectively</td>
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<td>Study design and data sources</td>
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<td>Detailed description of subjects’ characters can be checked in each study.</td>
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<td></td>
<td>a) Setting: Describe the study design and the underlying population, if possible. Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection, when available.</td>
<td>4</td>
<td>For each GWAS dataset, all bi-allelic SNPs and imputation score (INFO score) above</td>
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<td>4</td>
<td>d) For each exposure, outcome, and other relevant variables, describe methods of assessment and diagnostic criteria for diseases</td>
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<td>5</td>
<td>e) Provide details of ethics committee approval and participant informed consent, if relevant</td>
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<td>Assumptions</td>
<td>Explicitly state the three core IV assumptions for the main analysis (relevance, independence and exclusion restriction) as well assumptions for any additional or sensitivity analysis</td>
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<td>Statistical methods: main analysis</td>
<td>Describe statistical methods and statistics used</td>
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<td>6</td>
<td>a) Describe how quantitative variables were handled in the analyses (i.e., scale, units, model)</td>
<td>explore bidirectional causal links between each psychological state of blood pressure and anxiety, depressive symptoms, neuroticism and subjective well-being and hypertension in frame of Generalized Summary-data-based Mendelian Randomization (GSMR)</td>
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<td>6</td>
<td>b) Describe how genetic variants were handled in the analyses and, if applicable, how their weights were selected</td>
<td>And r2 = 0.05 as the LD threshold to identify independent SNP based on European population as reference within1000 genome project (phase3)</td>
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<td>6</td>
<td>c) Describe the MR estimator (e.g. two-stage least squares, Wald ratio) and related statistics. Detail the included covariates and, in case of two-sample MR, whether the same covariate set was used for adjustment in the two samples</td>
<td>in frame of Generalized Summary-data-based Mendelian Randomization (GSMR) [19]. This method based on summary-level data utilized independent genome-wide significant SNPs as instrumental variables, i.e an index of the exposure to test for</td>
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putative causal associations between a risk factor (exposure) and an outcome. Instrumental variants were selected based on the default GWAS threshold of $P \leq 5 \times 10^{-8}$ and $\text{r}_2 = 0.05$ as the LD threshold to identify independent SNP based on European population as reference within 1000 genome project (phase 3). HEIDI outlier detection was used to filter genetic instruments that played obvious pleiotropic effects on the exposure and outcome. A threshold $P$ value of 0.01 was used for the outlier detection analysis in HEIDI.

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<td>d)</td>
<td>Explain how missing data were addressed</td>
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<tr>
<td>e)</td>
<td>If applicable, indicate how multiple testing was addressed</td>
<td>6</td>
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<td>7</td>
<td><strong>Assessment of assumptions</strong></td>
<td>Describe any methods or prior knowledge used to assess the assumptions or justify their validity</td>
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<td>8</td>
<td><strong>Sensitivity analyses and additional analyses</strong></td>
<td>Describe any sensitivity analyses or additional analyses performed (e.g. comparison of effect estimates from different approaches, independent replication, bias analytic techniques, validation of instruments, simulations)</td>
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<td>9</td>
<td><strong>Software and pre-registration</strong></td>
<td>Name statistical software and package(s), including version and settings used</td>
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<td>b)</td>
<td>State whether the study protocol and details were pre-registered (as well as when and where)</td>
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<td><strong>RESULTS</strong></td>
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<td>10</td>
<td>Descriptive data</td>
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<td>a)</td>
<td>Report the numbers of individuals at each stage of included studies and reasons for exclusion. Consider use of a flow diagram</td>
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<tr>
<td>b)</td>
<td>Report summary statistics for phenotypic exposure(s), outcome(s), and other relevant variables (e.g. means, SDs, proportions)</td>
</tr>
<tr>
<td>c)</td>
<td>If the data sources include meta-analyses of previous studies, provide the assessments of heterogeneity across these studies</td>
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| d) | For two-sample MR:  
  i. Provide justification of the similarity of the genetic variant-exposure associations between the exposure and outcome samples  
  ii. Provide information on the number of individuals who overlap between the exposure and outcome studies | 7 |
| **Main results** |   |
| a) | Report the associations between genetic variant and exposure, and between genetic variant and outcome, preferably on an interpretable scale | 8 |
| b) | Report MR estimates of the relationship between exposure and outcome, and the measures of uncertainty from the MR analysis, on an interpretable scale, such as odds ratio or relative risk per SD difference | 8 |

The maximum sample size for BP traits for SBP, DBP and PP is 736,650, the minimum one for BP traits is 463,010 for hypertension. The maximum sample size for psychological states is 463,010 for anxiety, the minimum one is 170,911 for neuroticism.

There were no subjects overlapping between BP studies and psychological state studies.

1074 independent instrumental SNPs, which are significantly associated with DBP but not with neuroticism.

With the BP traits as exposure and psychological states as outcome, hypertension and DBP had significant
c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period

<table>
<thead>
<tr>
<th>12</th>
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<td>a) Report the assessment of the validity of the assumptions</td>
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<tr>
<td>b) Report any additional statistics (e.g., assessments of heterogeneity across genetic variants, such as $I^2$, Q statistic or E-value)</td>
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13 Sensitivity analyses and additional analyses

| a) Report any sensitivity analyses to assess the robustness of the main results to violations of the assumptions | n.a. |
| b) Report results from other sensitivity analyses or additional analyses | n.a |
| c) Report any assessment of direction of causal relationship (e.g., bidirectional MR) | 8 |

The reverse causal effects analysis indicated that after clumping and HEIDI-outlier filtering SNPs, less than the default threshold of 10 independent instrumental variants were retained for analyzing the causal effects of each psychological state on BP.

causal effects on neuroticism (P=8.8 E-6 and 0.026, respectively). After adjustment for multiple tests, only DBP is significantly associated with neuroticism ($b_{xy}=0.0036$, $P_{bonferroni}=0.00028$, Table 2)

n.a

If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period

Consider plots to visualize results (e.g., forest plot, scatterplot of associations between genetic variants and outcome versus between genetic variants and exposure)

These instrumental SNPs with F-statistic >10 are independent with the LD $r^2$ less than 0.05 and are remained through HEIDI-outlier analysis that can remove horizontal pleiotropic SNPs with $P$ value less than 0.01.
<table>
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<td><strong>14</strong> Key results</td>
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<td><strong>15</strong> Limitations</td>
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<td><strong>16</strong> Interpretation</td>
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**OTHER INFORMATION**

<p>| <strong>18</strong> Funding | Describe sources of funding and the role of funders in the present study and, if applicable, sources of funding for the databases and original study or studies on which the present study is based | 15 | Acknowledgement |
| <strong>19</strong> Data and data sharing | Provide the data used to perform all analyses or report where and how the data can be accessed, and reference these sources in the article. Provide the statistical code needed to reproduce the results in the article, or report whether the code is publicly accessible and if so, where | n.a |</p>
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Blood pressure highly likely to cause neurotic personality trait

Keeping it under control can help curb neuroticism, anxiety, and cardiovascular disease

Diastolic blood pressure—the lower of the two numbers in a blood pressure reading—is highly likely to cause neurotic personality trait, finds research published in the open access journal *General Psychiatry*.

And keeping it under control can help curb neurotic behaviours, anxiety, and heart and circulatory diseases, conclude the researchers.

High blood pressure is a major risk for cardiovascular disease and thought to be associated with psychological factors, such as anxiety, depression, and neuroticism—a personality trait characterised by susceptibility to negative emotions, including anxiety and depression.

But which causes which isn't entirely clear.

In a bid to find out, the researchers used a technique called Mendelian randomisation. This uses genetic variants as proxies for a particular risk factor—in this case, blood pressure—to obtain genetic evidence in support of a causal relationship, reducing the biases inherent in observational studies.

Between 30% and 60% of blood pressure is down to genetic factors, and over 1000 genetic single nucleotide polymorphisms, or SNPs for short, are associated with it. SNPs help predict a person’s response to certain drugs, susceptibility to environmental factors, and their risk of developing diseases.

The researchers drew on 8 large-scale study datasets containing whole genome DNA extracted from blood samples from people of predominantly European ancestry (genome-wide association studies).

They applied Mendelian randomisation to the 4 traits of blood pressure—systolic blood pressure (736,650 samples), diastolic blood pressure (736,650), pulse pressure (systolic minus diastolic blood pressure; 736,650), and high blood pressure (above 140/90 mm Hg; 463,010) with 4 psychological states—anxiety (463,010 samples), depressive symptoms (180,866), neuroticism (170,911) and subjective wellbeing (298,420).

The analysis revealed that high blood pressure and diastolic blood pressure had significant causal effects on neuroticism, but not on anxiety, depressive symptoms, or subjective wellbeing.

But after adjusting for multiple tests, only diastolic blood pressure was significantly associated with neuroticism (over 90%), based on 1074 SNPs.
The researchers acknowledge certain limitations to their findings. For example, it wasn’t possible to completely exclude pleiotropy—where one gene can affect several traits. And the findings may not be more widely applicable beyond people of European ancestry.

But blood pressure links the brain and the heart, and so may promote the development of personality traits, they explain.

“Individuals with neuroticism can be sensitive to the criticism of others, are often self-critical, and easily develop anxiety, anger, worry, hostility, self-consciousness, and depression.

“Neuroticism is viewed as a key causative factor for anxiety and mood disorders. Individuals with neuroticism more frequently experience high mental stress, which can lead to elevated [blood pressure] and cardiovascular diseases,” they write.

And they suggest: “Appropriate surveillance and control of blood pressure can be beneficial for the reduction of neuroticism, neuroticism-inducing mood disorders, and cardiovascular diseases.”