Chapter 6

Investigating the causal relationship of C-reactive protein with 32 complex somatic and psychiatric outcomes: A large scale cross-consortia Mendelian randomization study


* Equal contribution

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Abstract

**Background:** C-reactive protein (CRP) is associated with immune, cardiometabolic (somatic) and psychiatric traits and diseases. Yet it is inconclusive whether these associations are causal.

**Methods:** We performed Mendelian randomization (MR) analyses using two genetic risk scores (GRS) as instrumental variables (IVs). The first consisted of four single nucleotide polymorphisms (SNPs) in the CRP gene (GRS\textsubscript{CRP}), and the second of eighteen SNPs that were significantly associated with CRP levels in the largest genome-wide association study (GWAS) to date (GRS\textsubscript{GWAS}). To optimize power we used summary statistics from GWAS consortia and tested association of these two GRSs with 32 complex somatic and psychiatric outcomes comprising up to 123,865 participants per outcome from populations of European ancestry. We performed heterogeneity tests to disentangle pleotropic effect of IVs. A Bonferroni corrected significance level of less than 0.0016 was considered statically significant.

**Results:** The strengths (F statistics) of IVs were between 27.6-2527.8 and 83.4-6519.2 for GRS\textsubscript{CRP} and GRS\textsubscript{GWAS}, respectively. CRP GRS\textsubscript{GWAS} showed a statistically significant protective relationship of a 10% genetically elevated lnCRP levels with the risk of schizophrenia (OR 0.86 [95% CI 0.79-0.94]; P<0.001). CRP GRS\textsubscript{GWAS} showed nominal association with risk of knee osteoarthritis (1.17 [1.01-1.36]; P<0.04), bipolar disorder (1.21 [1.05-1.40]; P<7.17x10\textsuperscript{-3}), with an increase of 0.72 (0.11-1.34; P<0.02), and 0.39 (-0.01 - 0.78; P<0.05) mmHg in systolic and diastolic blood pressure, respectively. After adjustment for heterogeneity when necessary, none of the GRSs showed a significant effect on any of the other 27 studied complex diseases.

**Conclusions:** Genetically elevated CRP levels showed a significant potentially protective causal relationship with risk of schizophrenia. We observed nominal, yet to be confirmed, evidence for a causal relation between elevated CRP levels and SBP, DBP, knee osteoarthritis and bipolar disorder. Our analyses did not support any causal effect of CRP on 27 other common somatic and neuropsychiatric outcomes investigated in the present study. This implies that interventions lowering CRP levels are unlikely to result in decreased risk for the majority of common complex outcomes.
Introduction

Emerging evidence suggests that the persistent dysregulation of the inflammatory response is linked to a plethora of complex somatic and neuropsychiatric disorders (1–18). Epidemiological studies have shown that C-reactive protein (CRP), a well-studied biomarker of inflammation, is associated with and exhibited a reliable predictive value for cardiovascular disease (19, 20), type 2 diabetes (21), and immunity-related disorders such as inflammatory bowel disease (IBD) (22), rheumatoid arthritis (23) and all-cause mortality (20, 24). Nevertheless, the evidence for a causal involvement of CRP from traditional experimental or observational studies remains controversial (25, 26), fuelling the debate whether CRP contributes to the chain of causality in disease mechanisms (27). The use of genetically informed instrumental variables (IVs) termed Mendelian randomization is a complementary approach to epidemiological observations and allows investigating whether the effect of an exposure (i.e. CRP levels) on observed outcome phenotypes is likely to be causal (28).

Recent large-scale Mendelian randomization studies, focussing mainly on cardiovascular disease and metabolic traits, failed to show a causal association between CRP and these outcomes (Supplementary Table S1). This has led to the notion that elevated CRP levels do not causally contribute to these traits and disorders. However, these studies have used either a single CRP-associated single nucleoid polymorphism (SNP), or a very limited set of CRP-associated SNPs (Supplementary Table S1). Common SNPs serving as proxies for CRP levels represent only a small effect on CRP levels per se requiring a large enough sample size to detect causal effects on the outcome. Moreover, most studies have generally included a limited range of common, complex diseases, often not more than two or three outcomes, or they have been performed in a single or small population yielding inadequate study power (Supplementary Table S1). In other words, existing evidence for a causal relationship between CRP and a broad range of common traits or diseases remains inconclusive. This is mostly due to the lack of well-powered Mendelian randomization studies that use optimally informative genetic IVs for CRP. Here, we sought to comprehensively examine the hypothesis that genetically increased CRP levels directly contribute to common somatic and psychiatric outcomes. To optimize IV power, we applied a Mendelian randomization approach using summary statistics of outcomes data from large-scale genome-wide association study (GWAS) consortia for the largest known set of
independent SNPs known to be associated with CRP and for the four variants representing 98% of the common variation in the CRP gene.

**Methods**

**Study design and rationale**

The present Mendelian randomization study consists of two key components: first, we used established variants associated with CRP levels, and combined them to build two genetic risk scores (GRS) for CRP: The first one consisted of only four SNPs in the CRP gene (GRS\textsubscript{CRP}) selected from the largest recent Mendelian randomization study of CRP\textsuperscript{(29)}, and the second of eighteen SNPs that were genome-wide significantly associated with CRP levels in the largest GWAS to date (GRS\textsubscript{GWAS})\textsuperscript{(30)}. Second, we obtained summary association statistics from GWAS consortia for a panel of 32 common somatic and psychiatric outcomes (Table 1). The corresponding authors have selected the studies, and contacted each consortium with a standardized data request for study data, including the name of study/consortium, number of cases and controls, number of available CRP SNPs for GRS\textsubscript{CRP} and GRS\textsubscript{GWAS}, and the estimated effects for each SNP (or its proxy) on outcome, i.e. per allele regression coefficient with standard errors or odds ratio and corresponding 95% confidence interval.

Data were available for 32 different outcomes in five broad disease classes, (i.e. autoimmune-inflammatory, cardiovascular, metabolic, neuro-degenerative and psychiatric), including at least 2,525 up to 123,865 participants per outcome from populations of European ancestry (Table 1). These outcomes were selected based on the following two inclusion criteria: (i) having been associated with CRP levels in epidemiological studies and (ii) availability of large meta-GWAS analyses for the outcome, as our study design relies on summary association statistics of GWAS level data (Table 1).

**Genetic instruments**

Weak IVs yielding insufficient statistical power may have hampered estimation of causal effects of CRP on the outcomes in previous analyses (Supplementary Table S1). Our Mendelian randomization approach, by using GWAS data, and combining multiple independent SNPs into a GRS (i.e. IV), has the potential to greatly increase power. These IVs were used to test the combined effect of the associations of CRP level influencing alleles with the outcomes. The selected SNPs have been described
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elsewhere(30–32), and are further detailed in Supplementary Tables S2 and S3. Our approach was implemented in such a way that both the effects of independent SNPs in the CRP gene (GRS\textsubscript{CRP})(31, 32) and independent SNPs known to be genome-wide significantly associated with CRP levels (GRS\textsubscript{GWAS})(30), as well as pleiotropic effects of SNPs could be discriminated(33). Pleiotropy exists if CRP SNPs influence exposures (risk factors) other than CRP levels and therefore would violate Mendelian randomization assumptions. We selected all independent SNPs for GRS\textsubscript{CRP} and assessed if they influenced outcome risk only \textit{via} changes in CRP levels.

\textbf{Table 1} Diseases and traits included in this study.

<table>
<thead>
<tr>
<th>Disease / Trait Class</th>
<th>Abbreviation</th>
<th>Cases</th>
<th>Controls</th>
<th>Total</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Autoimmune/Inflammatory</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Celiac Disease</td>
<td>CED</td>
<td>4533</td>
<td>10750</td>
<td>15283</td>
<td>(34)</td>
</tr>
<tr>
<td>Inflammatory Bowel Disease (all types)</td>
<td>IBD</td>
<td>13020</td>
<td>34774</td>
<td>47794</td>
<td>(35, 36)</td>
</tr>
<tr>
<td>Crohn's Disease</td>
<td>CD</td>
<td>6333</td>
<td>15056</td>
<td>21389</td>
<td>(35)</td>
</tr>
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<td>Ulcerative Colitis</td>
<td>UC</td>
<td>6687</td>
<td>19718</td>
<td>26405</td>
<td>(36)</td>
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<tr>
<td>Psoriasis Vulgaris\textsuperscript{a}</td>
<td>PSV</td>
<td>4007</td>
<td>4934</td>
<td>8941</td>
<td>(37, 38)</td>
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<tr>
<td>Psoriatic Arthritis\textsuperscript{a}</td>
<td>PSA</td>
<td>1946</td>
<td>4934</td>
<td>6880</td>
<td>(37, 38)</td>
</tr>
<tr>
<td>Psoriasis Cutaneous\textsuperscript{a}</td>
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<td>1363</td>
<td>3517</td>
<td>4880</td>
<td>(37, 38)</td>
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<tr>
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<td>20167</td>
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<tr>
<td>Knee Osteoarthritis</td>
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<td>18505</td>
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<td><strong>Cardiovascular</strong></td>
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<td>Coronary Artery Disease</td>
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<td>—</td>
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<td>(45)</td>
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<td>Diastolic Blood Pressure</td>
<td>DBP</td>
<td>—</td>
<td>—</td>
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<td>(45)</td>
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<td>Ischemic Stroke (all types)</td>
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<td>5972</td>
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<td>—</td>
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<td>15872</td>
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Chapter 6

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<th>Disease / Trait Class</th>
<th>Abbreviation</th>
<th>Cases</th>
<th>Controls</th>
<th>Total</th>
<th>Reference</th>
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<td>—</td>
<td>—</td>
<td>74354</td>
<td>(49)</td>
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<td>Serum Albumin Levels</td>
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<td>—</td>
<td>—</td>
<td>53189</td>
<td>(50)</td>
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<tr>
<td>Serum Protein Levels</td>
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<td>—</td>
<td>—</td>
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<td>(50)</td>
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<td>Alzheimer's Disease</td>
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<table>
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<td>Autism</td>
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<td>Bipolar Disorder</td>
</tr>
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<td>Major Depressive Disorder</td>
</tr>
<tr>
<td>Schizophrenia</td>
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</table>

#results from individual cohorts combined in meta-analysis, which is, yet unpublished data.

Statistical analysis

All analyses were done using the GRS function implemented in the grs.summary module as part of the Genetics ToolboX R (version 2.15.1 for Windows; Vienna, Austria). The grs.summary module approximates the regression of an outcome onto an additive GRS, using only single SNP association summary statistics extracted from GWAS results. The method is described in more detail elsewhere (58). In brief, we performed Mendelian randomization analyses using GRS IVs in two steps: first, we used individual CRP gene SNPs (i.e. IVs) associated with CRP levels (32, 59) (Supplementary Tables S2 and S3) to create a weighted GRS, named GRS\textsubscript{CRP}, corresponding to the joint effect of four SNPs within the CRP gene (31). We extracted \( \omega \) (the estimated coefficient or weight) for individual SNPs from the association results as reported by the CRP Coronary Heart Disease Genetics Collaboration (CCGH) (31), which represent one unit (in mg/L) increase of the natural log of CRP (lnCRP) per dose of the coded allele. These four tagging SNPs represent 98% of the common variation in the CRP gene assuming minor allele frequency \( \geq 0.05 \) and an \( r^2 \) threshold of \( \geq 0.8 \), and aggregately explain \( \sim 2% \) of the total variation (i.e. phenotypic variance) in serum CRP levels in populations of European descent (31, 59). Second, we constructed a multilocus GRS, named...
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GRS\textsubscript{GRS}, that combined 18 SNPs associated with serum CRP levels at genomewide significance (P<5×10\textsuperscript{−6}; Supplementary Tables S2 and S3), derived from a large meta-GWAS analysis for CRP conducted by the CHARGE consortium\textsuperscript{(30)}. This multilocus GRS explains approximately ~5% of the total variation in serum CRP levels\textsuperscript{(30)}.

We integrated ω for each CRP SNP from the reference data of CCGH\textsuperscript{(31)} or meta-analysis of GWASs\textsuperscript{(30)} for CRP levels with the summary association statistics extracted from GWAS consortia for each outcome. This Mendelian randomization approach using meta-GWAS summary statistics data is equivalent to an inverse-variance weighted meta-analysis and has previously been validated in comparison to individual level data\textsuperscript{(33, 60)}. To estimate the causal effect of CRP on an outcome, we obtained the β values (estimated effects from regression analysis) for CRP SNPs on the outcome with standard errors of se from the corresponding discovery GWAS results. When no summary statistics for the SNPs of in genetic instruments were available in the look-up dataset, we chose a proxy SNP that had the highest linkage disequilibrium (LD) with the initial SNP (r\textsuperscript{2}>0.9 in HapMap release 22). If several proxy SNPs had the exact same r\textsuperscript{2} values, we chose the proxy nearest to the original SNP in the instrument. Separate regressions of outcomes on GRSs were performed to calculate α\textsubscript{IV} estimators (causal IV estimator) for each outcome. Correspondingly, the value of a GRS is the sum of the ω values, which is multiplied by the allele dosage (i.e. 0, 1 or 2) for each CRP SNP in the CCGC or in the CHARGE CRP consortium\textsuperscript{(30, 31)}. For uncorrelated SNPs, when maximizing the likelihood function, the α\textsubscript{IV} value and its standard error, se\textsubscript{α}, can be approximated with the formula: \{α≅(Σω×β×se\textsubscript{β}\textsuperscript{2})/(Σω\textsuperscript{2}×se\textsubscript{β}\textsuperscript{2})\} with its \{se\textsubscript{α}≅\sqrt{1/(Σω\textsuperscript{2}×se\textsubscript{β}\textsuperscript{2})}\}. Since lnCRP was used as the outcome in reference studies\textsuperscript{(30)(31)} to obtain the ω values (i.e. effect sizes) for each of the CRP SNPs, a unit increase in lnCRP equals to a 10 symmetric percentage (s%) increase in CRP levels, which corresponds to a unit change in level of a continuous outcome or logit of risk-estimate (i.e. beta coefficient) for a dichotomous outcome\textsuperscript{(61)}. The α\textsubscript{IV} value (i.e. causal estimate) for each CRP SNP was, therefore, presented for each outcome as corresponding to a 10 s% increase in actual CRP levels.

To assess which SNP might have violated one of the main Mendelian randomization assumptions of pleiotropy, we performed goodness-of-fit tests to correct both GRSs for heterogeneity of effects on outcome. Heterogeneity, which indicates potential presence of pleiotropy, was measured using Q statistics, and was considered statistically significant at a conservative uncorrected P value <0.05.
Although heterogeneity could be an indicator of pleiotropy; there are other factors that could introduce heterogeneity in the analyses. Therefore, even though the adopted adjustments for heterogeneity that we have taken could be overconservative, we have taken this method in order to minimize false positives. After stepwise removal of SNPs with potential pleiotropic effects, we repeated the analyses until significant heterogeneity was no longer observed.

To further ensure the strength of these two GRSs as IVs, we generated an F statistic for each outcome. We used variance in lnCRP levels explained by each set of CRP SNPs (2% and 5% respectively for GRS_{CRP}, GRS_{GWAS}), to calculate F statistics using the formula as $F_{\text{statistic}}=\frac{R^2 \times (n-1-K)}{(1-R^2 \times K)}$, where “R$^2$” represents proportion of variability in the CRP that is explained by GRS, “n” represents sample size, and “K” represents number of IVs included in model (i.e. for this study K=1) (62). As a rule of thumb, an F value above 10 indicates that a causal estimate is unlikely to be biased due to weak instruments (33).

Multiple testing. The present study included 32 independent sample-sets. Per each sample-set, we did one statistical test, for which a global nominal significance level of 0.05 would be considered as satisfactory to derive conclusions. The need for correction for multiple testing is debatable. Nevertheless, to ensure the validity of our conclusions, we took a conservative approach, and applied a Bonferroni corrected significance threshold calculated as 0.05 divided by 32 (i.e. 0.0016). We present our results and discussion at three different levels of confidence for corresponding causal estimates; we considered a statistical test with an observed P value more than 0.05 as a definite non-significant result yielded no association; an observed P value equal or less than 0.05 as nominal significant evidence for a potential causal association but yet to be confirmed; and an observed P value equal or less than 0.0016 as statistically significant evidence for a causal association.

Results

Using the GRS_{CRP}, we first tested whether a CRP gene determined increase in lnCRP levels was associated with the outcome risks. In Table 2, the causal effects of lnCRP estimated for each outcome are summarized. We found no heterogeneity in the IV analyses ($P_{\text{heterogeneity}} \geq 0.11$ for all outcomes) while the GRS_{CRP} was a strong instrument ($F \geq 31$). IV analyses provided nominal evidence for potential causal relationships of lnCRP with risk for Crohn’s disease (odd ratio [OR] 0.78 [95%CI 0.65-0.94];P<0.009), psoriatic arthritis (1.45 [1.04-2.04];P<0.03), schizophrenia (0.90 [0.82-0.99];P<0.03), and increase in SBP (mean increase 1.23
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(0.45—2.01);P<0.002), and DBP (0.70 (0.20—1.19);P<0.006) in mmHg per 10 s% increase in CRP levels. The GRS_{CRP} showed no significant effect on any of the other outcomes (Table 2, Supplementary Figure S1).

**Table 2** The effect of the CRP Genetic Risk Score instrument of four SNPs in CRP (GRS_{CRP}) with somatic and neuropsychiatric outcomes.

<table>
<thead>
<tr>
<th>Disease / Trait Class</th>
<th>M</th>
<th>N</th>
<th>Effect size (95% CI) *</th>
<th>P-value</th>
<th>P-het</th>
<th>F-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Autoimmune/Inflammatory</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Celiac Disease</td>
<td>3</td>
<td>15283</td>
<td>0.96 (0.77-1.21)</td>
<td>0.750</td>
<td>0.19</td>
<td>311.86</td>
</tr>
<tr>
<td>Inflammatory Bowel Disease (all)</td>
<td>3</td>
<td>47794</td>
<td>0.97 (0.84-1.13)</td>
<td>0.700</td>
<td>0.30</td>
<td>975.35</td>
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<td>Crohn's Disease</td>
<td>4</td>
<td>21389</td>
<td>0.78 (0.65-0.94)</td>
<td>0.009</td>
<td>0.25</td>
<td>436.47</td>
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<tr>
<td>Ulcerative Colitis</td>
<td>4</td>
<td>26405</td>
<td>1.10 (0.92-1.31)</td>
<td>0.290</td>
<td>0.92</td>
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<td>8941</td>
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<td>0.95</td>
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<td>0.92</td>
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<td>1.10 (0.76-1.59)</td>
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<td>0.60</td>
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<td>25702</td>
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<td>0.550</td>
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<td>Systemic Lupus Erythematosus</td>
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<td>4651</td>
<td>1.20 (0.80-1.81)</td>
<td>0.380</td>
<td>0.19</td>
<td>94.88</td>
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<td>0.85</td>
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<td>0.34</td>
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<td>0.94 (0.78-1.13)</td>
<td>0.500</td>
<td>0.23</td>
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<td><strong>Cardiovascular</strong></td>
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<tr>
<td>Coronary Artery Disease</td>
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<td>86995</td>
<td>1.07 (0.95-1.20)</td>
<td>0.280</td>
<td>0.41</td>
<td>177.37</td>
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<td>Systolic Blood Pressure **</td>
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<td>69372</td>
<td>1.23 (0.45-2.01)</td>
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<td>0.51</td>
<td>1415.63</td>
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<td>Diastolic Blood Pressure **</td>
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<td>0.70 (0.2-1.19)</td>
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<td>Ischemic Stroke (all types)</td>
<td>4</td>
<td>9520</td>
<td>1.19 (0.93-1.53)</td>
<td>0.160</td>
<td>0.93</td>
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<td>Ischemic Stroke (Cardioembolic)</td>
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<td>6762</td>
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<td>0.940</td>
<td>0.96</td>
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<td>1.44 (0.93-2.21)</td>
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<td>0.31</td>
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<td>Ischemic Stroke (Small Vessel)</td>
<td>4</td>
<td>6552</td>
<td>1.18 (0.71-1.95)</td>
<td>0.520</td>
<td>0.36</td>
<td>133.06</td>
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<tr>
<td><strong>Metabolic</strong></td>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Body Mass Index ***</td>
<td>4</td>
<td>123864</td>
<td>-0.017 (-0.06-0.02)</td>
<td>0.410</td>
<td>0.50</td>
<td>2527.82</td>
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<tr>
<td>Type 2 Diabetes</td>
<td>4</td>
<td>22570</td>
<td>1.11 (0.94-1.32)</td>
<td>0.230</td>
<td>0.50</td>
<td>460.57</td>
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<td>Chronic Kidney Disease</td>
<td>4</td>
<td>74354</td>
<td>1.04 (0.88-1.22)</td>
<td>0.670</td>
<td>0.90</td>
<td>1517.39</td>
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<td>eGFR for creatinine ****</td>
<td>4</td>
<td>74354</td>
<td>0.004 (-0.01-0.02)</td>
<td>0.400</td>
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<td>Serum Albumin Levels *****</td>
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<td>-0.002 (-0.02-0.01)</td>
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<td>0.88</td>
<td>1085.45</td>
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<td>Serum Protein Levels *****</td>
<td>4</td>
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<td>0.008 (-0.02-0.04)</td>
<td>0.640</td>
<td>0.12</td>
<td>521.12</td>
</tr>
</tbody>
</table>
### Disease / Trait Class

<table>
<thead>
<tr>
<th>Disease / Trait Class</th>
<th>M</th>
<th>N</th>
<th>Effect size (95% CI) *</th>
<th>( P )-value</th>
<th>( P )-het</th>
<th>F-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Neurodegenerative</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Amyotrophic Lateral Sclerosis</td>
<td>2</td>
<td>12263</td>
<td>0.79 (0.60-1.04)</td>
<td>0.090</td>
<td>0.23</td>
<td>258.39</td>
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<tr>
<td>Alzheimer’s Disease</td>
<td>2</td>
<td>13020</td>
<td>1.26 (0.89-1.78)</td>
<td>0.200</td>
<td>0.11</td>
<td>265.67</td>
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<tr>
<td>Parkinson’s Disease</td>
<td>3</td>
<td>17352</td>
<td>1.00 (0.85-1.17)</td>
<td>0.960</td>
<td>0.33</td>
<td>354.08</td>
</tr>
<tr>
<td><strong>Psychiatric</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Autism</td>
<td>3</td>
<td>1566</td>
<td>1.02 (0.97-1.07)</td>
<td>0.380</td>
<td>0.69</td>
<td>31.92</td>
</tr>
<tr>
<td>Bipolar Disorder</td>
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<td>16731</td>
<td>1.17 (0.97-1.42)</td>
<td>0.110</td>
<td>0.49</td>
<td>341.41</td>
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<tr>
<td>Major Depressive Disorder</td>
<td>3</td>
<td>18759</td>
<td>0.98 (0.81-1.18)</td>
<td>0.810</td>
<td>0.86</td>
<td>382.80</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>3</td>
<td>79845</td>
<td>0.90 (0.82-0.99)</td>
<td>0.030</td>
<td>0.79</td>
<td>1629.45</td>
</tr>
</tbody>
</table>

Abbreviations: M: number of markers used in the genetic instrument; N: number of samples in the disease/trait meta-analysis; Effect size (95% CI): Effect size (95% CI) per mg/L increase in lnCRP serum levels; \( P \)-value: \( P \)-value of goodness of fit test; \( P \)-het: \( P \)-value of heterogeneity of effect test; F-value: F-statistic value for the used genetic instrument;

* For risk of disease, effect size is given in odds ratios, otherwise in the specific units in which the outcome was measured. Derived from the IV causal estimator \( \alpha \).

** Effect size unit is mm Hg per increase in ln serum CRP (mg/L).

*** Effect size unit is 1 standard deviation per ln mg/L increase in serum CRP (the BMI results were inverse normal transformed to a distribution with \( \mu = 0 \) and \( \sigma = 1 \)).

**** Effect size unit is ml per min per 1.73 m\(^2\), per ln mg/L increase in serum CRP.

***** Effect size unit g/dL, per ln mg/L increase in serum CRP.

#results from individual cohorts combined in meta-analysis which is yet unpublished data

Second, the GRS\(_{GFA}\) showed a statistically significant protective effect of lnCRP on the risk of schizophrenia (OR 0.86 [95%CI 0.79-0.94];\( P<0.0010 \)) per 10 s% increase in CRP levels (Figure 1, Table 3). The GRS\(_{GFA}\) also showed moderate but nominally significant effects of lnCRP on the risk of IBD (OR 0.85 [95%CI 0.74-0.98];\( P<0.03 \)), Crohn’s disease (0.81[0.70-0.94];\( P<0.005 \)), psoriatic arthritis (1.36 [1.00-1.84];\( P<0.049 \)), knee osteoarthritis (1.17 [1.01-1.36];\( P<0.04 \)), coronary artery disease (CAD) (0.86 [0.79-0.95];\( P<0.002 \)), and bipolar disorder (1.21 [1.05-1.40];\( P<0.007 \)) (Table 3, Figure 1, Supplementary Figure S1). GRS\(_{GFA}\) revealed a nominally significant increase of 0.72 (95%CI 0.11-1.34;\( P<0.02 \)), and 0.45 (0.06-0.84;\( P<0.02 \)) mmHg in SBP and DBP respectively (Table 3; Supplementary Figure S1). Likewise, genetically 10 s% increase in CRP levels was nominally associated
with a 0.01 ml/min/1.73m² (0.003-0.02;P<0.005) higher estimated glomerular filtration rate from serum creatinine (eGFRₐ), 0.01 g/dl (0.0004-0.02;P<0.04) higher albumin, and 0.03 g/dl (0.008-0.05;P<0.009) higher serum protein levels. The remaining outcomes tested for causal associations using GRSₚ were not significant, though the corresponding GRSₚ proved to be a strong IV with F values ≥82 (Table 3, Supplementary Figure S1).

Figure 1 Genetic Risk Score GRSₚ for bipolar disorder (A) and schizophrenia (B). Genetic risk score plots for bipolar disorder and schizophrenia. Horizontal axes: effect size for up to 18 SNPs comprising the GRSₚ influencing levels of CRP, with corresponding standard error bars. Vertical axes: Log odds ratio for the GRSₚ SNPs in bipolar disorder (A) or schizophrenia (B) with corresponding standard error bars. The effect estimate of CRP levels on disease risk or trait level is represented by a red solid line with gradient ?. The 95% CI of this ? estimate is represented by grey dashed lines.

The included SNPs are shown by Arabic numbering as: #1 rs2847281 (gene:PTPN2; chr:18;basepair position:12811593); #2: rs340029 (ROA;15;58682257); #3: rs6091250 (GPRC6A;6;11720218); #4: rs10745954 (ASCL1;12;102007224); #5: rs4705952 (IRF1;1;131867517); #6: rs12037222 (PABPC1;1;39837548); #7: rs12239046 (NLRP3;1;246668218); #8: rs6734238 (IL1F3;2;113555701); #9: rs12323571 (BCL7;7;72609167); #10: rs987289 (PPP1R3B;8;9220768); #11: rs1260326 (GCKR;2;27584444); #12: rs4129267 (IL6R;1;25692888); #13: rs1800961 (HNF4A;20;42475778); #14: rs4420065 (LEPR;1;6;59340049); #15: rs10521222 (SALL1;1;6;49716211); #16: rs1183910 (HNF1A;12;119952190); #17: rs2794520 (CRP;1;157945440); #18: rs4420638 (APOC1;19;50114786).

Using the GRSₚ, there was no significant evidence of heterogeneity of the effect sizes for knee osteoarthritis, bipolar disorder, schizophrenia, and SBP,
while the heterogeneity test was statistically significant for psoriatic arthritis, IBD, Crohn's disease, CAD, DBP, eGFR_{cr}, serum albumin and serum protein. These heterogeneities in the effects of GRS_{GWAS} may be attributable to pleiotropic effects of SNPs used to build the GRS_{GWAS}. We subsequently performed a stepwise removal of SNPs from GRS_{GWAS} until no significant heterogeneity remained and presented the results in Table 4. This adjustment in the GRS_{GWAS} resulted in the removal of three SNPs from the GRS_{GWAS} for IBD (in GCKR, IRF1, PTPN2), five SNPs for Crohn's disease (in GCKR, IL6R, IRF1, PABPC4, PTPN2), one SNP for psoriatic arthritis (in GCKR), five SNPs for CAD (in APOC1, ASCL1, HNF1A, IL6R, IL1F10), one SNP for DBP (in PABPC4), two SNPs for eGFR (in LEPR and GCKR), eight SNPs for serum albumin (in APOC1, BCL7B, GCKR, IL6R, LEPR, PPP1R3B, PTPN2, SALL1), and one SNP for serum protein levels (in GCKR). After removal of these variants from the GRS_{GWAS}, we found no significant association between genetically increased lnCRP levels and any of these outcomes (Table 4). However, for DBP, 17 SNPs remained in the GRS_{GWAS} and yielded a slightly lower causal estimate (when compared to the values before adjustment) of 0.39 mmHg (-0.01 to 0.78) increase in DBP per 10 s% increase in lnCRP levels with a nominal significance of P<0.05.

<table>
<thead>
<tr>
<th>Disease / Trait Class</th>
<th>M</th>
<th>Effect size (95% CI) *</th>
<th>P-value</th>
<th>P-het</th>
<th>F-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autoimmune/Inflammatory</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Celiac Disease</td>
<td>18</td>
<td>0.99(0.85-1.16)</td>
<td>0.930</td>
<td>7.2x10^{-4}</td>
<td>804.26</td>
</tr>
<tr>
<td>Inflammatory Bowel Disease (all)</td>
<td>15</td>
<td>0.85(0.74-0.98)</td>
<td>0.030</td>
<td>1.4x10^{-5}</td>
<td>2515.37</td>
</tr>
<tr>
<td>Crohn's Disease</td>
<td>17</td>
<td>0.81(0.70-0.94)</td>
<td>0.005</td>
<td>4.4x10^{-7}</td>
<td>1125.63</td>
</tr>
<tr>
<td>Ulcerative Colitis</td>
<td>17</td>
<td>1.05(0.91-1.21)</td>
<td>0.490</td>
<td>0.01</td>
<td>1389.63</td>
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<tr>
<td>Psoriasis Vulgaris#</td>
<td>17</td>
<td>1.12(0.90-1.40)</td>
<td>0.310</td>
<td>0.19</td>
<td>470.47</td>
</tr>
<tr>
<td>Psoriatic Arthritis#</td>
<td>17</td>
<td>1.36(1.00-1.84)</td>
<td>0.049</td>
<td>0.04</td>
<td>362.00</td>
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<tr>
<td>Psoriasis Cutaneous#</td>
<td>17</td>
<td>1.00(0.72-1.39)</td>
<td>0.990</td>
<td>0.16</td>
<td>256.74</td>
</tr>
<tr>
<td>Rheumatoid Arthritis</td>
<td>18</td>
<td>0.93(0.80-1.08)</td>
<td>0.350</td>
<td>1.8x10^{-4}</td>
<td>1352.79</td>
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<tr>
<td>Systemic Lupus Erythematosus</td>
<td>11</td>
<td>1.06(0.71-1.58)</td>
<td>0.780</td>
<td>0.27</td>
<td>244.68</td>
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<tr>
<td>Systemic Sclerosis</td>
<td>11</td>
<td>0.84(0.62-1.14)</td>
<td>0.280</td>
<td>0.63</td>
<td>396.89</td>
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<tr>
<td>Type 1 Diabetes</td>
<td>15</td>
<td>1.10(0.92-1.31)</td>
<td>0.310</td>
<td>3.47x10^{-3}</td>
<td>1415.16</td>
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<tr>
<td>Knee Osteoarthritis</td>
<td>18</td>
<td>1.17(1.01-1.36)</td>
<td>0.040</td>
<td>0.10</td>
<td>1276.74</td>
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</table>
Mendelian randomization of CRP in 32 somatic and psychiatric outcomes

<table>
<thead>
<tr>
<th>Disease / Trait Class</th>
<th>M</th>
<th>Effect size (95% CI) *</th>
<th>P-value</th>
<th>P-het</th>
<th>F-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary Artery Disease</td>
<td>18</td>
<td>0.86 (0.79-0.95)</td>
<td>0.002</td>
<td>2.08x10^{-8}</td>
<td>4578.58</td>
</tr>
<tr>
<td>Systolic Blood Pressure **</td>
<td>18</td>
<td>0.72 (0.11-1.34)</td>
<td>0.020</td>
<td>0.14</td>
<td>3650.84</td>
</tr>
<tr>
<td>Diastolic Blood Pressure **</td>
<td>18</td>
<td>0.45 (0.06-0.84)</td>
<td>0.020</td>
<td>0.02</td>
<td>3651.05</td>
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<tr>
<td>Ischemic Stroke (all types)</td>
<td>18</td>
<td>1.06 (0.87-1.29)</td>
<td>0.570</td>
<td>0.37</td>
<td>500.95</td>
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<tr>
<td>Ischemic Stroke (Cardioembolic)</td>
<td>18</td>
<td>0.98 (0.69-1.39)</td>
<td>0.920</td>
<td>0.35</td>
<td>355.79</td>
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<tr>
<td>Ischemic Stroke (Large Vessel)</td>
<td>18</td>
<td>1.30 (0.92-1.82)</td>
<td>0.140</td>
<td>0.97</td>
<td>358.63</td>
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<tr>
<td>Ischemic Stroke (Small Vessel)</td>
<td>18</td>
<td>0.85 (0.58-1.25)</td>
<td>0.420</td>
<td>0.76</td>
<td>343.16</td>
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<tr>
<td>Metabolic</td>
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<td></td>
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<tr>
<td>Body Mass Index ***</td>
<td>18</td>
<td>-0.005 (-0.03-0.02)</td>
<td>0.740</td>
<td>0.11</td>
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<tr>
<td>Type 2 Diabetes</td>
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<td>1.09 (0.95-1.24)</td>
<td>0.210</td>
<td>1.8x10^{-5}</td>
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<tr>
<td>Chronic Kidney Disease</td>
<td>18</td>
<td>0.96 (0.84-1.09)</td>
<td>0.500</td>
<td>0.07</td>
<td>3913.26</td>
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<tr>
<td>eGFR for creatinine ****</td>
<td>18</td>
<td>0.011 (0.003-0.02)</td>
<td>0.005</td>
<td>7.2x10^{-9}</td>
<td>3913.26</td>
</tr>
<tr>
<td>Serum Albumin Levels *****</td>
<td>18</td>
<td>0.011 (0.0004-0.02)</td>
<td>0.041</td>
<td>2.3x10^{-18}</td>
<td>2799.32</td>
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<tr>
<td>Serum Protein Levels *****</td>
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<td>0.031 (0.008-0.05)</td>
<td>0.009</td>
<td>0.03</td>
<td>1343.95</td>
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<td>Neurodegenerative</td>
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<tr>
<td>Amyotrophic Lateral Sclerosis</td>
<td>8</td>
<td>1.01 (0.79-1.29)</td>
<td>0.960</td>
<td>0.56</td>
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<td>Alzheimer's Disease</td>
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<td>1.26 (0.99-1.61)</td>
<td>0.060</td>
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<td>0.500</td>
<td>0.50</td>
<td>913.16</td>
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<td>Psychiatric</td>
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<tr>
<td>Autism #</td>
<td>9</td>
<td>0.89 (0.70-1.13)</td>
<td>0.350</td>
<td>0.99</td>
<td>82.32</td>
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<tr>
<td>Bipolar Disorder</td>
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<td>1.21 (1.05-1.40)</td>
<td>0.007</td>
<td>0.15</td>
<td>880.47</td>
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<tr>
<td>Major Depressive Disorder</td>
<td>15</td>
<td>1.14 (0.96-1.36)</td>
<td>0.140</td>
<td>0.84</td>
<td>987.21</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>15</td>
<td>0.86 (0.79-0.94)</td>
<td>0.001</td>
<td>0.66</td>
<td>4202.26</td>
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</tbody>
</table>

Abbreviations: M: number of markers used in the genetic instrument; Effect size (95% CI): Effect size (95% CI) per mg/L increase in lnCRP serum levels; P-value: P-value of goodness of fit test; P-het: P-value of heterogeneity of effect test; F-value: F-statistic value for the used genetic instrument

* For risk of disease, effect size is given in odds ratios, otherwise in the specific units in which the outcome was measured. Derived from the IV causal estimator α.

** Effect size unit is mm Hg per increase in ln serum CRP (mg/L).

*** Effect size unit is 1 standard deviation per ln mg/L increase in serum CRP (the BMI results were inverse normal transformed to a distribution with μ = 0 and σ = 1).

**** Effect size unit is ml per min per 1.73 m², per ln mg/L increase in serum CRP.

***** Effect size unit g/dL, per ln mg/L increase in serum CRP.

# results from individual cohorts combined in meta-analysis which is yet unpublished data.
Table 4: The effect of the CRP Genetic Risk Score instrument of 18 SNPs associated to CRP (GRS\textsubscript{GWAS}) with somatic and neuropsychiatric outcomes after correcting for heterogeneity.

<table>
<thead>
<tr>
<th>Disease / Trait Class</th>
<th>M</th>
<th>Effect size (95% CI)*</th>
<th>P-value</th>
<th>P-het</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Autoimmune/Inflammatory</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Celiac Disease</td>
<td>16</td>
<td>1.05 (0.90-1.23)</td>
<td>0.56</td>
<td>0.10</td>
</tr>
<tr>
<td>Inflammatory Bowel Disease</td>
<td>12</td>
<td>0.92 (0.79-1.06)</td>
<td>0.24</td>
<td>0.14</td>
</tr>
<tr>
<td>Crohn’s Disease</td>
<td>12</td>
<td>0.93 (0.79-1.08)</td>
<td>0.34</td>
<td>0.12</td>
</tr>
<tr>
<td>Ulcerative Colitis</td>
<td>16</td>
<td>1.11 (0.96-1.28)</td>
<td>0.16</td>
<td>0.12</td>
</tr>
<tr>
<td>Psoriatic Arthritis</td>
<td>16</td>
<td>1.25 (0.91-1.72)</td>
<td>0.16</td>
<td>0.07</td>
</tr>
<tr>
<td>Rheumatoid Arthritis</td>
<td>12</td>
<td>0.98 (0.81-1.19)</td>
<td>0.85</td>
<td>0.09</td>
</tr>
<tr>
<td>Type 1 Diabetes</td>
<td>14</td>
<td>1.06 (0.89-1.27)</td>
<td>0.52</td>
<td>0.07</td>
</tr>
<tr>
<td><strong>Cardiovascular</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary Artery Disease</td>
<td>13</td>
<td>1.03 (0.91-1.18)</td>
<td>0.55</td>
<td>0.07</td>
</tr>
<tr>
<td>Diastolic Blood Pressure **</td>
<td>17</td>
<td>0.385 (-0.008-0.78)</td>
<td>0.05</td>
<td>0.09</td>
</tr>
<tr>
<td><strong>Metabolic</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 2 Diabetes</td>
<td>17</td>
<td>0.95 (0.82-1.10)</td>
<td>0.52</td>
<td>0.09</td>
</tr>
<tr>
<td>eGFR for creatinine *****</td>
<td>16</td>
<td>0.001 (-0.007-0.01)</td>
<td>0.74</td>
<td>0.11</td>
</tr>
<tr>
<td>Serum Albumin *****</td>
<td>10</td>
<td>-0.005 (-0.02-0.01)</td>
<td>0.49</td>
<td>0.15</td>
</tr>
<tr>
<td>Serum Protein *****</td>
<td>17</td>
<td>0.021 (-0.002-0.05)</td>
<td>0.07</td>
<td>0.31</td>
</tr>
</tbody>
</table>

Abbreviations: M: number of markers used in the genetic instrument; Effect size (95% CI): Effect size (95% CI) per mg/L increase in lnCRP serum levels; P-value: P-value of goodness of fit test; P-het: P-value of heterogeneity of effect test.

* For risk of disease, effect size is given in odds ratios, otherwise in the specific units in which the outcome was measured. Derived from the IV causal estimator $\alpha$.

** Effect size unit is mm Hg per increase in ln serum CRP (mg/L).

**** Effect size unit is ml per min per 1.73 m\textsuperscript{2}, per ln mg/L increase in serum CRP.

***** Effect size unit g/dL, per ln mg/L increase in serum CRP.

Likewise, we hypothesized that a non-significant effect of CRP using GRS\textsubscript{GWAS} on celiac disease, ulcerative colitis, rheumatoid arthritis, type 1 diabetes and type 2 diabetes can be to some extent explained by significant heterogeneity observed for these outcomes (Table 3). This adjustment in the GRS\textsubscript{GWAS} resulted in the removal of two SNPs from the GRS\textsubscript{GWAS} for celiac disease (in PABPC4, PTPN2), one SNP for ulcerative colitis (in GCKR), six SNPS for rheumatoid
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arthritis (in APOC1, GCKR, HNF4A, IL6R, PPP1R3B, SALL1), one SNP for type 1 diabetes (in PTPN2), one SNP for type 2 diabetes (in APOC1). However, after adjusting for heterogeneity, the association of GRS

Discussion

In this large scale cross-consortia Mendelian randomization study of 32 complex outcomes, we provide evidence for a potential causal relationship between elevated CRP levels, and schizophrenia in both genetic IVs (i.e. GRS

\textit{GWAS}) with these outcomes remained non-significant (Table 4).
may fuel the debate about whether the observed elevated levels of CRP in schizophrenia are a by-product of the pathogenesis of schizophrenia or directly contribute to the clinical manifestations of the disorder(6). This finding may also point out potential biases in previous studies regarding the causes of elevated CRP levels in patients with schizophrenia such as reverse causality and/or pleiotropic effects within chosen instruments. As for bipolar disorder, though the nominal predisposing effect needs to be confirmed, our finding corroborates epidemiological observations suggesting that higher levels of CRP are associated with the disease(76). In bipolar disorder, our findings support a potential causal predisposing association between this disorder with inflammation in general. Our results failed to pass a multiple testing correction, though they may be biologically sensible, nevertheless independent confirmations on cohort level, and by functional follow-up analyses and with the use of a stronger CRP GRGWAS from upcoming studies are required to make a factitive conclusion.

We found nominally significant evidence for up to ~0.70 mmHg increased blood pressure for a 10 s% increase in CRP levels and no evidence for heterogeneity for SBP. Additionally, there was nominally borderline evidence of a causal association for DBP after we adjusted for heterogeneity. These nominally significant findings, on the one hand, are in line with numerous epidemiological studies that have highlighted an association between higher CRP levels and decreased risk of hypertension. For instance, one study found an association between CRP loci and hypertension in Asians(77). An additional line of support for a possible causal association of CRP and blood pressure comes from an experimental study where the authors found that when in mice CRP gene expression increased, and subsequently, CRP protein levels, this led to a rise in particularly SBP (78). Moreover an ex-vivo study by Zhou et al. has shown that combining IL-6 treatment and mechanical strain has led to a consistent increase in CRP expression at protein and mRNA levels in smooth muscle cells(79). Both inflammatory factors and local mechanical strains are abundant in blood vessels and are well known risk factors for high blood pressure. However, on the other hand, our finding did not reach a statically significant level after correction for multiple testing; thus it may echo previous Mendelian randomization studies which have failed to find a causal relationship between CRP levels and blood pressure or hypertension in Europeans(80, 81). However our systematic review showed previous studies had some limitations (Supplementary Table S1). For instance, no study used a refined GWAS set of 18 CRP associated SNPs rather they tested single or a limited set of CRP SNPs. Using such instruments might have led to biased
estimates as their corresponding effects on CRP levels have been found to be small(30, 33). A combination of weak instruments and low sample sizes might lead to type II error(28, 33), and hence to a conclusion of no causal association between CRP and blood pressure traits in previous studies. Taken together, a direct link between CRP and blood pressure remains to be elucidated, though our nominal association between GRS\textsubscript{CRP}, GRS\textsubscript{GWAS} and blood pressure do add to a line of findings from experimental studies suggesting a potential causal relationship between CRP and blood pressure.

Our nominally significant findings that CRP might be a potential causal factor for knee osteoarthritis (using GRS\textsubscript{GWAS}), should be interpreted with caution. In line with our findings, we have shown earlier that the levels of CRP were higher in women with early radiological knee osteoarthritis (i.e. Kellgren-Lawrence grade 2+), and in women whose disease progressed(82). Additionally, another study showed that genetically elevated CRP levels contribute to osteoarthritis severity(83). However, other studies found contrasting results(84)(85). One systematic review provided evidence that the relationship between CRP and osteoarthritis does exist, but is dependent on BMI(85). It remains to be further investigated whether weight gain over the lifetime mediates the potential causal association between genetically elevated CRP and knee osteoarthritis.

The present study was able to calculate nominal causal estimates for IBD, Crohn’s disease, psoriatic arthritis, CAD, eGFRcr, serum albumin or protein using a CRP GRS\textsubscript{GWAS} but they were altered by removal of SNPs from GRS\textsubscript{GWAS} based on heterogeneity tests resulting in non-significant associations. These outcomes appeared therefore to have heterogeneity in causal estimates suggesting these observed estimates were biased likely due to pleiotropic effects of CRP loci. These results corroborate negative findings of previous studies (Supplementary Table S1), suggesting a causal role of CRP in these traits and diseases is unlikely.

A detailed evaluation of pleiotropic SNPs in our study showed that our applied method to identify heterogeneity sources was able to indicate and exclude several already known pleiotropic loci from the GRS\textsubscript{GWAS} IV. For instance, the use of a SNP in \textit{IL6R} (rs4129267) amongst others resulted in heterogeneity of effects on CAD risk. The same variant contributed to heterogeneity of effects for Crohn’s disease and serum albumin levels in our study, and it has been shown that this SNP is associated with levels of biomarkers other than CRP[56]. Further, a Mendelian randomization study found that \textit{IL6R} SNPs, specifically, the non-synonymous SNP rs8192284, are associated with CAD risk and CRP levels(86). Our selected \textit{IL6R}
SNPs, namely rs4537545 or rs4129267, are in extremely high LD with rs8192284 ($r^2 \geq 0.96$ for both SNPs). Carriers of the risk allele of rs8192284 have higher CRP, IL6 and fibrinogen levels(86). Fibrinogen is also a well-known risk factor for CAD. Therefore, it is unclear so far which biomarker(s) mediates the effect of IL6R SNPs on CAD. Besides the IL6 locus, APOC1 and PABPC4 have been indicated as pleiotropic in three, and PTPN2 in four and GCKR in six out of 32 our investigated outcomes. Taken together, we were able to disentangle at least part of the pleiotropic effect on the causal estimates of CRP with outcomes. Again, we found no significant association of CRP GRS with outcomes. Again, we found no significant association of CRP GRS with outcomes.

Even though our results were mostly based on very large sample sizes of GWAS consortia and therefore much better powered than most previous Mendelian randomization studies investing causal involvement of CRP, we found that genetically elevated CRP levels approximated by powerful instruments appear not to contribute directly to most of the studied somatic and psychiatric outcomes. It is of utmost interest whether the observed effect of CRP as a risk predictor is causal and thus whether reduction of CRP levels will lower the risk of disease. Our findings are consistent with previous Mendelian randomization studies that suggest null associations of genetically elevated CRP levels with inflammation-related outcomes including CAD(32, 59, 87), type 2 diabetes(88), BMI(89), Alzheimer’s disease and depression(90). All previous Mendelian randomization studies were substantially limited to a single or a few outcomes, used only SNPs in the CRP gene or had sample sizes much smaller than the present study (Supplementary Table S1). In addition to these studies, the use of current GWAS data do not corroborate epidemiological observations suggesting that elevated CRP levels are associated with amyotrophic lateral sclerosis(91), Alzheimer’s disease(92), Parkinson’s disease(93), and major depressive disorder(94). Furthermore, patients with immunity-related disorders frequently have a very high CRP level (as high as 100 mg/L) due to their disease status. Our findings may therefore more favorably indicate reverse causality. Taken together, we showed that CRP is highly unlikely to contribute causally to most of the major common somatic and neuropsychiatric outcomes that are investigated in the present study.

**Strengths and limitations of the study**

Results presented in table 2 show that our GRScrP is not a weak instrument, as indicated by its high F values owing to the available large sample sizes of outcomes from GWAS consortia. The strength of our instruments increased considerably in all disease classes when we used variants of multiple loci associated with CRP in
Mendelian randomization of CRP in 32 somatic and psychiatric outcomes

GWAS. However, the variants comprising the CRP GRS_{GWAS} explain on average only a moderate ~5% of the total variance in baseline CRP levels(30). Moreover, the possibility of effect modification by non-genetic CRP related factors on the outcomes remains to be investigated. We may be able to create even stronger instruments based on ongoing efforts to identify additional variation influencing CRP levels. Even if larger sample sizes and stronger instruments can be realized, the overwhelming lack of causal effects observed for most of outcomes in our current study also implies that CRP specific lowering therapies will not directly result in decreased risk of the investigated outcomes, or a better efficacy(95, 96). Here we used summary associations statistics obtained from previously conducted meta-GWASs in order to maximize our study power. One may argue this may induce bias, compared to when one uses the individual level data. Nevertheless, several studies showed a high agreement in results for both the GWAS summary data and individual level data Mendelian randomization methods(60, 97).

Conclusion

We showed that elevated CRP levels driven by genetic factors are causally associated with schizophrenia. We observed nominal evidence that genetically elevated CRP is causally associated with SBP, DBP, knee osteoarthritis and bipolar disorder. Our analyses did not support any causal effect of CRP on the other 27 common somatic and neuropsychiatric outcomes investigated in the present study. Therefore, disease associated rise in CRP levels in these 27 outcomes may be interpreted as a response to the disease process rather than a cause for it. This implies that interventions lowering CRP levels are unlikely to result in decreased risk for the majority of common complex outcomes.

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We thank for Dan Arking for providing the genome-wide summary statistics data on CRP polymorphisms for the Autism GWAS. We also would like to acknowledge the Rheumatoid Arthritis and Systemic Lupus Erythematosus consortia, the Genetic Investigation of ANthropometric Traits consortium (GIANT), and the Psychiatric Genomics Consortium (PGC) who made their data publicly available from which we extracted summary association statistics for CRP SNPs.

The PAGE consortium is grateful to Alex Tsoi for adding the consortium information and membership list.
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Weblinks

Software

Genetics ToolboX R (version 2.15.1 for Windows; Vienna, Austria): [http://cran.r-project.org/web/packages/gtx/index.html](http://cran.r-project.org/web/packages/gtx/index.html)

Publicly downloaded GWAS summary statistics


2. CARDIoGRAM CAD summary statistics: [www.cardiogramplusc4d.org](http://www.cardiogramplusc4d.org)
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3. Rheumatoid arthritis summary statistics:
   www.broadinstitute.org/ftp/pub/rheumatoid_arthritis/Stahl_etal_2010NG/

4. PGC consortium (psychiatric) summary statistics:
   www.med.unc.edu/pgc/downloads

5. Systemic Lupus Erythematosus
   Data was download through dbGaP: http://www.ncbi.nlm.nih.gov/gap
   Study name: Whole Genome Association Study of Systemic Lupus Erythematosus
   dbGaP Study Accession: phs000122.v1.p1
   Analysis Name and Accession
   Name: Whole Genome Association Study of Systemic Lupus Erythematosus
   Accession: pha002848.1

References


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### Supplementary Table 1  Previous Mendelian randomization analyses using CRP variants as instruments

<table>
<thead>
<tr>
<th>Disease/Trait Class</th>
<th>Reference (PMID)</th>
<th>Year</th>
<th>Sample size</th>
<th>Indication of CRP causality</th>
<th>Variants used as instruments (Nearby) genes</th>
<th>Findings</th>
<th>Notes / Study Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>15731495</td>
<td>2005</td>
<td>&gt;3500 women</td>
<td></td>
<td>rs1800947 CRP</td>
<td>The polymorphism was associated with a robust difference in CRP, but the predicted causal effects of CRP on blood pressure, pulse pressure, and hypertension using instrumental variables methods were close to null, although with wide CIs.</td>
<td>CRP levels are associated with blood pressure, pulse pressure, and hypertension, but adjustment for life course confounding and a Mendelian randomization approach suggest the elevated CRP levels do not lead to elevated blood pressure.</td>
</tr>
<tr>
<td>Coronary Artery Disease</td>
<td>21325005 (including 46557 patients with prevalent or incident coronary heart disease)</td>
<td>2011</td>
<td>194418 (including 46557 patients with prevalent or incident coronary heart disease)</td>
<td></td>
<td>rs3093077 rs1205 rs1130864 rs1800947 CRP</td>
<td>CRP variants were each associated with up to 30% per allele difference in concentration of C reactive protein (P&lt;10^-34) and were unrelated to other risk factors. Risk ratios for coronary heart disease per additional copy of an allele associated with raised C reactive protein were 0.93 (95% confidence interval 0.87 to 1.00) for rs3093077; 1.00 (0.98 to 1.02) for rs1205; 0.98 (0.96 to 1.00) for rs1130864; and 0.99 (0.94 to 1.03) for rs1800947. In a combined analysis, the risk ratio for coronary heart disease was 1.00 (0.90 to 1.13) per 1 SD higher genetically raised natural log (ln) concentration of C reactive protein. The genetic findings were discordant with the risk ratio observed for coronary heart disease of 1.33 (1.23 to 1.43) per 1 SD higher circulating ln concentration of C reactive protein in prospective studies (P=0.001 for difference).</td>
<td>Human genetic data indicate that C reactive protein concentration itself is unlikely to be even a modest causal factor in coronary heart disease.</td>
</tr>
<tr>
<td>Disease/Trait Class</td>
<td>Reference (PMID)</td>
<td>Year</td>
<td>Sample size</td>
<td>Indication of CRP causality</td>
<td>Variants used as instruments (Nearby) genes</td>
<td>Findings</td>
<td>Notes / Study Conclusions</td>
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<tr>
<td>Coronary Artery Disease</td>
<td>19567438</td>
<td>2009</td>
<td>28112 cases and 10823 controls</td>
<td>SNP rs7553007 and published data for SNPs rs1130864 and rs1205 for 18 cohorts. SNP rs7553007 was not significantly associated with CHD, estimated OR was 0.98 (95% CI, 0.94–1.01) per 20% lower CRP. For rs1130864, OR was 1.00 (95% CI, 0.86–1.15) and for rs1205, OR was 1.03 (95% CI, 0.99–1.07). There was no association of CHD with CRP variants (per 20% lower CRP) when results for all three SNPs were combined, OR 1.00 (95% CI, 0.97–1.02).</td>
<td>SNPs were not in themselves associated with an increased risk of ischemic vascular disease.</td>
<td>The lack of concordance between the effect on coronary heart disease risk of CRP genotypes and CRP levels argues against a causal association of CRP with coronary heart disease.</td>
<td></td>
</tr>
<tr>
<td>Coronary Artery Disease</td>
<td>18714384</td>
<td>2008</td>
<td>18637 (including 4610 cases)</td>
<td>SNP rs1130864 suggested that circulating CRP was not associated with CHD: the odds ratio for a doubling of CRP level was 1.04 (95% CI: 0.61, 1.80). No association of this SNP, which is known to be related to CRP levels, and having CHD.</td>
<td>An instrumental variables analysis by rs1130864 would not be associated with CHD, the odds ratio for a doubling of CRP level was 1.04 (95% CI: 0.61, 1.80).</td>
<td>These findings do not support a causal association between circulating CRP and CHD risk, but very large, extended, genetic association studies would be required to rule this out.</td>
<td></td>
</tr>
<tr>
<td>Coronary Artery Disease</td>
<td>16365153</td>
<td>2006</td>
<td>6201 for MR</td>
<td>SNP rs1130864 suggested that circulating CRP was not associated with CHD: the odds ratio for a doubling of CRP level was 1.04 (95% CI: 0.61, 1.80). No association of this SNP, which is known to be related to CRP levels, and having CHD.</td>
<td>A difference in CRP of 0.68 mg/l (95% CI 0.31–1.10) would confer an OR for non-fatal MI of 1.25 (95% CI 1.09–1.43) or 1.20 (95% CI 1.07–1.38) using data from a meta-analysis of prospective cohort studies. However, for men with the TT genotype, the point estimate of the OR for non-fatal MI was 1.01 (95% CI 0.74–1.38).</td>
<td>The null association of CRP variant with coronary events suggests possible residual confounding (or reverse causation) in the CRP coronary event association in observational studies, though the confidence limits are still compatible with a modest causal effect.</td>
<td></td>
</tr>
<tr>
<td>Ischemic Heart Disease</td>
<td>18971492</td>
<td>2008</td>
<td>6586 cases, 41996 controls</td>
<td>SNP rs3093077 and published data for SNPs rs3091244 and rs1130864 for 18 cohorts. SNP rs3093077 was significantly associated with CHD, estimated OR was 1.10 (95% CI, 1.04–1.15) per 20% lower CRP. For rs3091244, OR was 1.00 (95% CI, 0.86–1.15) and for rs1130864, OR was 1.03 (95% CI, 0.99–1.07). There was no association of CHD with CRP variants (per 20% lower CRP) when results for all three SNPs were combined, OR 1.00 (95% CI, 0.97–1.02).</td>
<td>Genotype combinations of the four CRP polymorphisms were associated with an increase in CRP levels of up to 64%, resulting in a theoretically predicted increased risk of up to 32% for ischemic heart disease and up to 25% for ischemic cerebrovascular disease. However, these genotype combinations were not associated with an increased risk of ischemic vascular disease.</td>
<td>Polymorphisms in the CRP gene are associated with marked increases in CRP levels and thus with a theoretically predicted increase in the risk of ischemic vascular disease. However, these polymorphisms are not in themselves associated with an increased risk of ischemic vascular disease.</td>
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<tr>
<td>Disease/Trait</td>
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<tr>
<td><strong>Body Mass Index</strong></td>
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<table>
<thead>
<tr>
<th>Reference (PMID)</th>
<th>Year</th>
<th>Sample Size</th>
<th>Indication of CRP Causality</th>
<th>Variants Used as Instruments</th>
<th>Findings</th>
<th>Notes / Study Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>20714329</td>
<td>2011</td>
<td>37,027</td>
<td></td>
<td>rs3091244 CRP</td>
<td>Observational analyses showed change in BMI of 1.03 kg/m² (95% CI: 1.00, 1.07), P &lt; 0.0001, for a doubling in logCRP. Analysis using rs3091244 CRP to re-estimate the causal effect of circulating CRP on BMI yielded null effects (change in BMI for a doubling in logCRP of −0.24 kg/m² (95% CI: −0.58, 0.11), P = 0.2). These data suggest that the observed association between circulating CRP and measured BMI is likely to be driven by BMI, with CRP being a marker of elevated adiposity.</td>
<td>Greater adiposity conferred by FTO and MC4R SNPs led to higher CRP levels, with no evidence for any reverse pathway.</td>
</tr>
<tr>
<td>19906786</td>
<td>2010</td>
<td>5804</td>
<td></td>
<td>rs1800947 CRP, rs1205 CRP</td>
<td>With increasing CRP allele score, there was a stepwise decrease in CRP levels (P for trend &lt; 0.0001) and a 1.98 mg/liter difference between extremes of the allele score distribution, but there was no associated change in BMI or leptin levels (P &gt; or = 0.89). Adiposity allele score was associated with increased CRP levels (1.24 mg/liter difference between extremes; P for trend 0.02).</td>
<td></td>
</tr>
<tr>
<td>19584180</td>
<td>2009</td>
<td>5,363 (2,526 men and 2,836 women)</td>
<td></td>
<td>rs7553007, rs1805096 LEPR</td>
<td>Log-transformed CRP explained by the rs7553007 CRP gene was significantly associated with BMI (regression coefficient: 1.22 (0.18; 2.25), P = 0.02) and fat mass [2.67 (0.65; 4.68), P = 0.01] but not with lean mass in women, whereas no association was found in men. Log-transformed CRP explained by the combined CRP-LEPR instrument was significantly associated with BMI [0.98 (0.32; 1.63), P = 0.004], fat mass [2.07 (0.79; 3.34), P = 0.001], and waist [2.09 (0.91; 3.78), P = 0.01] in women but not men.</td>
<td>CRP is causally and positively related to BMI in women and that this is mainly due to fat mass. Results on the combined CRP-LEPR instrument suggest that leptin may play a role in the causal association between CRP and adiposity in women. Results in men were not significant.</td>
</tr>
<tr>
<td>Disease/Trait Class</td>
<td>Reference (PMID)</td>
<td>Year</td>
<td>Sample size</td>
<td>Indication of CRP causality</td>
<td>Variants used as instruments (Nearby) genes</td>
<td>Findings</td>
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<tr>
<td>Type 2 Diabetes</td>
<td>18700811</td>
<td>2008</td>
<td>5,274</td>
<td></td>
<td>rs1130864, rs1205, rs3093077</td>
<td>CRP</td>
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<tr>
<td>Psychiatric</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Major Depression</td>
<td>24246360</td>
<td>2014</td>
<td>7,809</td>
<td></td>
<td>rs1130864, rs1205, rs3093077</td>
<td>CRP</td>
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<tr>
<td>Schizophrenia</td>
<td>23996346</td>
<td>2013</td>
<td>7,810</td>
<td></td>
<td>rs1130864, rs1205, rs3093077</td>
<td>CRP</td>
</tr>
</tbody>
</table>
### Supplementary Table 2 CRP lead variants used in the genetic risk score as instrumental variables

**A) CRP gene genetic risk score (ie. GRS\textsubscript{CRP})**

<table>
<thead>
<tr>
<th>SNP ID</th>
<th>Chr</th>
<th>Position (build 36)</th>
<th>Alleles</th>
<th>AF</th>
<th>(Nearby) genes</th>
<th>Effect of lead variant on ln (CRP) levels</th>
<th>Standard Error of Effect</th>
<th>P-value in respective CRP meta-analysis</th>
<th>Reference (PMID)</th>
</tr>
</thead>
<tbody>
<tr>
<td>rs3093077</td>
<td>1</td>
<td>157946260</td>
<td>G/T</td>
<td>0.0500</td>
<td>CRP</td>
<td>0.207</td>
<td>0.033</td>
<td>5.40E-35</td>
<td>21325005</td>
</tr>
<tr>
<td>rs1205</td>
<td>1</td>
<td>157948857</td>
<td>C/T</td>
<td>0.6667</td>
<td>CRP</td>
<td>0.169</td>
<td>0.016</td>
<td>1.00E-40</td>
<td>21325005</td>
</tr>
<tr>
<td>rs1130864</td>
<td>1</td>
<td>157949715</td>
<td>T/C</td>
<td>0.3091</td>
<td>CRP</td>
<td>0.127</td>
<td>0.014</td>
<td>1.00E-40</td>
<td>21325005</td>
</tr>
<tr>
<td>rs1800947</td>
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**B) CRP multilocus genetic risk score (ie. GRS\textsubscript{GWA,3})**

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SNP indicates single nucleotide polymorphism; Chr, chromosome; Alleles, raising/lowering alleles respectively; AF, allele frequency of raising Allele in European populations; and CRP, C-reactive protein.
**Supplementary Table 3** Proxy SNPs of CRP lead variants used in the genetic risk scores as instrumental variables.

A) CRP gene genetic risk score (GRS<sub>CRP</sub>) - proxy SNPs of CRP lead variants.

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### B) CRP multilocus genetic risk score (GRS\textsubscript{GWAS})- proxy SNPs of CRP lead variants

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SNP indicates single nucleotide polymorphism; Chr, chromosome; CRP, C-reactive protein; bp, base pairs; and LD, linkage disequilibrium ($r^2$) with lead SNP.

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### Supplementary Table 4

Individual association summary statistics of CRP lead SNPs and / or proxies with traits and diseases.

**A) CRP gene genetic risk score (GRS<sub>CRP</sub>)**

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**Metabolic**

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**Neurodegenerative**

<p>| ALS           | rs3093077     | T/G     | 1   | CRP          | −          | −/−              | − (−)              | −         | −          | −       |
| ALS           | rs1205        | T/C     | 1   | CRP          | rs2794520  | C/T              | 1.019 (0.961-1.08) | 0.019 (0.03) | 5.29E-01 | −         | 12260  |
| ALS           | rs1130864     | C/T     | 1   | CRP          | rs12093699 | G/A              | 0.943 (0.889-1)    | -0.059 (0.03) | 4.87E-02 | −         | 12252  |
| ALS           | rs1800947     | C/G     | 1   | CRP          | −          | −/−              | − (−)              | −         | −          | −       |
| ALZ           | rs3093077     | T/G     | 1   | CRP          | rs11265260 | G/A              | 0.888 (0.775-1.018) | -0.119 (0.058) | 8.79E-02 | 4.663-8357 | 13020  |
| ALZ           | rs1205        | T/C     | 1   | CRP          | −          | −/−              | − (−)              | −         | −          | −       |
| ALZ           | rs1130864     | C/T     | 1   | CRP          | rs12093699 | G/A              | 1 (0.943-1.06)     | 0 (0.029)  | 9.88E-01 | 4.663-8357 | 13020  |</p>
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**Supplementary Table 4** Individual association summary statistics of CRP lead SNPs and / or proxies with traits and diseases.

B) CRP multilocus genetic risk score (GRS<sub>GWA</sub>)- CRP lookup SNPs and association summary statistics in consortia.

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Cardiometabolic

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Note: The table above lists various SNPs, genes, and their associated genetic markers, along with odds ratios and p-values for different diseases and traits.
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Mendelian randomization of CRP in 22 somatic and psychiatric outcomes
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Neurodegenerative

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| AUT rs12037222 | G/A     | 1   | PABPC4       | rs2293476 | G/C    | 0.974 (—/—)      | -0.027 (0.04) | 6.77E-01 | 90-1476              | 1566   |
| AUT rs4420065  | T/C     | 1   | LEPR         | —         | —/—    | — (-)             | (−)       | —            | 15                   | 1566   |
| AUT rs4129267  | T/C     | 1   | IL6R         | rs4537545 | C/T    | 1.071 (—/—)      | 0.069 (0.368) | 1.87E-01 | 90-1476              | 1566   |
| AUT rs2794520  | T/C     | 1   | CRP          | rs7553007 | C/T    | 1.03 (—/—)       | 0.03 (0.05)  | 5.87E-01 | 90-1476              | 1566   |
| AUT rs12239046 | T/C     | 1   | NLRRP3       | —         | —/—    | — (-)             | (−)       | —            | 15                   | 1566   |
| AUT rs1260326  | C/T     | 2   | GCKR         | rs780094  | C/T    | 0.911 (—/—)      | -0.094 (1.154) | 8.12E-02 | 90-1476              | 1566   |
| AUT rs6734238  | A/G     | 2   | IL1F10       | rs10176274| G/C    | 1.05 (—/—)       | 0.049 (0.134) | 3.64E-01 | 90-1476              | 1566   |
| AUT rs4705952  | A/G     | 5   | IRF1         | —         | —/—    | — (-)             | (−)       | —            | 15                   | 1566   |
| AUT rs6901250  | G/A     | 6   | GPRC6A       | rs6924002 | A/T    | 0.985 (—/—)      | -0.015 (0.019) | 7.86E-01 | 90-1476              | 1566   |
| AUT rs13233571  | T/C     | 7   | BCL7B        | rs17145738| C/T    | 0.951 (—/—)      | -0.05 (0.095) | 5.27E-01 | 90-1476              | 1566   |
| AUT rs9987289  | A/G     | 8   | PPP1R3B      | —         | —/—    | — (-)             | (−)       | —            | 15                   | 1566   |
| AUT rs10745954  | G/A     | 12  | ASCL1        | rs7972145 | G/A    | 0.995 (—/—)      | -0.005 (0.005) | 9.20E-01 | 90-1476              | 1566   |
| AUT rs1183910  | A/G     | 12  | HNF1A        | —         | —/—    | — (-)             | (−)       | —            | 15                   | 1566   |
| AUT rs340029   | C/T     | 15  | RORA         | —         | —/—    | — (-)             | (−)       | —            | 15                   | 1566   |
| AUT rs10521222 | T/C     | 16  | SALL1        | —         | —/—    | — (-)             | (−)       | —            | 15                   | 1566   |</p>
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</table>

SNP indicates single nucleotide polymorphism; Requested SNP Alleles, lowering allele/increasing allele respectively; Chr, chromosome; Lookup SNP Alleles, non-coded allele/coded allele respectively; Odds Ratio, Odds Ratio (95% Confidence Interval) for coded allele; and Beta (SE), effect size (standard error) for coded allele.

**Disease/trait abbreviations:** CED indicates Celiac Disease; IBD, Inflammatory Bowel Disease (all types); CD, Crohn's Disease; UC, Ulcerative Colitis; PSV, Psoriasis Vulgaris; PSA, Psoriatic Arthritis; PSC, Psoriasis Cutaneous; RA, Rheumatoid Arthritis; SLE, Systemic Lupus Erythematosus; SSC, Systemic Sclerosis; T1D, Type 1 Diabetes; KOA, Knee Osteoarthritis; CAD, Coronary Artery Disease; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; IS, Ischemic Stroke (all types); IS (CS), Ischemic Stroke (Cardioembolic); IS (LVS), Ischemic Stroke (Large Vessel); IS (SVD), Ischemic Stroke (Small Vessel); BMI, Body Mass Index; T2D, Type 2 Diabetes; CKD, Chronic Kidney Disease; eGFR, eGFR for creatinine; SA, Serum Albumin Levels; SP, Serum Protein Levels; ALS, Amyotrophic Lateral Sclerosis; ALZ, Alzheimer's Disease; PKD, Parkinson's Disease; AUT, Autism; BPD, Bipolar Disorder; MDD, Major Depressive Disorder; and SCZ, Schizophrenia.
Supplementary Figure S1 GRS plots. For each disease / trait we show the plots for two genetic risk scores; on the left the GRS\textsubscript{CRP} and on the right GRS\textsubscript{GWAS}. For every graph, the estimated effects on disease risk (log odds) or trait level (vertical axis) are plotted against estimated effects on the natural log CRP levels (mg/ml) (horizontal axis), for either the GRS\textsubscript{CRP} SNPs or GRS\textsubscript{GWAS} SNPs that are associated with CRP levels. The grey vertical lines indicate the 95% confidence interval (CI) for each individual SNP. The effect estimate of CRP levels on disease risk or trait level is represented by a solid line with gradient $\alpha$. The 95% CI of this $\alpha$ estimate is represented by dashed lines.

1 Celiac disease
Supplementary Figure S1

GRS plots. For each disease / trait we show the plots for two genetic risk scores; on the left the GRS\_CRP and on the right GRS\_GWAS. For every graph, the estimated effects on disease risk (log odds) or trait level (vertical axis) are plotted against estimated effects on the natural log CRP levels (mg/ml) (horizontal axis), for either the GRS\_CRP SNPs or GRS\_GWAS SNPs that are associated with CRP levels. The grey vertical lines indicate the 95% confidence interval (CI) for each individual SNP. The effect estimate of CRP levels on disease risk or trait level is represented by a solid line with gradient α. The 95% CI of this α estimate is represented by dashed lines.

1 Celiac disease
2 Inflammatory Bowel Disease (all types)
3 Crohn's Disease
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4 Ulcerative Colitis
5 Psoriasis vulgaris

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6 Psoriatic Arthritis
7 Psoriasis Cutaneous
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8 Rheumatoid Arthritis
9 Systemic Lupus Erythematosus
10 Systemic Sclerosis
11 Type I Diabetes

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12 Knee Osteoarthritis

13 Coronary Artery Disease

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14 Systolic Blood Pressure

15 Diastolic Blood Pressure
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12 Knee Osteoarthritis

13 Coronary Artery Disease

14 Systolic Blood Pressure

15 Diastolic Blood Pressure
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16 Ischemic Stroke (all types)

17 Ischemic Stroke (Cardioembolic Stroke)

18 Ischemic Stroke (Large Vessel Disease)

19 Ischemic Stroke (Small Vessel Disease)
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Body Mass Index

Type II Diabetes

Mendelian randomization of CRP in 32 somatic and psychiatric outcomes

Chronic Kidney Disease

eGFR for Creatinine
Mendelian randomization of CRP in 32 somatic and psychiatric outcomes

22 Chronic Kidney Disease

23 eGFR for Creatinine
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24 Serum Albumin Levels

25 Serum Protein Levels

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26 Amyotrophic Lateral Sclerosis

27 Alzheimer's Disease
Mendelian randomization of CRP in 32 somatic and psychiatric outcomes

26. Amyotrophic Lateral Sclerosis

27. Alzheimer's Disease
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Mendelian randomization of CRP in 32 somatic and psychiatric outcomes

28 Parkinson’s Disease

30 Bipolar Disorder

29 Autism

31 Major Depressive Disorder
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Bipolar Disorder

Major Depressive Disorder
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32 Schizophrenia
Supplementary text S1. Extended methods.

1.1 Calculation of the Alzheimer's Disease GWAS summary statistics

We received summary statistics (SNP, OR, SE, P, Reference Allele, Other Allele, OR 95 L, OR 95 U) from the Genetic and Environmental Risk in Alzheimer's Disease (GERAD) consortium for three separate Alzheimer’s datasets; from the TGEN consortium, from the ADNI consortium, and from the GERAD consortium for up to 4663 cases and 8357 controls. We next performed an inverse variance weighted fixed effects analysis using GWAMA\(^1\) to calculate combined effect sizes and standard errors, which were subsequently used in our genetic risk scores.

1.2 Calculation of the BMI GWAS summary statistics

We downloaded sex-stratified summary statistics for BMI from Randall et.al\(^2\). From:


We next performed an inverse variance weighted fixed effects analysis using GWAMA\(^1\) to calculate combined effect sizes and standard errors, which were subsequently used in our genetic risk scores.


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Supplementary text S2 Additional acknowledgments and consortia related information.

T1D GC study

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Supplementary text S3 Consortia co-author lists.

The Genetic and Environmental Risk for Alzheimer's disease (GERAD1) Consortium

Data used in the preparation of this article were obtained from the Genetic and Environmental Risk for Alzheimer's disease (GERAD1) Consortium. As such, the investigators within the GERAD1 consortia contributed to the design and implementation of GERAD1 and/or provided data but did not participate in analysis or writing of this report.


Affiliations: 1Medical Research Council (MRC) Centre for Neuropsychiatric Genetics and Genomics, Neurosciences and Mental Health Research Institute, Department of Psychological Medicine and Neurology, School of Medicine, Cardiff University, Cardiff, UK. 2Kings College London, Institute of Psychiatry, Department of Neuroscience, De Crespigny Park, Denmark Hill, London. 3Institute of Public Health, University of Cambridge, Cambridge, UK. 4Cambridge Institute for Medical Research, University of Cambridge, Cambridge, UK. 5Mercers Institute for Research on Aging, St. James Hospital and Trinity College, Dublin, Ireland. 6Institute of Genetics, Queens Medical Centre, University of Nottingham,
UK. 7Ageing Group, Centre for Public Health, School of Medicine, Dentistry and Biomedical Sciences, Queens University Belfast, UK. 8Division of Clinical Neurosciences, School of Medicine, University of Southampton, Southampton, UK. 9Clinical Neuroscience Research Group, Greater Manchester Neurosciences Centre, University of Manchester, Salford, UK. 10Oxford Project to Investigate Memory and Ageing (OPTIMA), University of Oxford, Department of Pharmacology, Mansfield Road, Oxford, UK. 11University of Bristol Institute of Clinical Neurosciences, School of Clinical Sciences, Frenchay Hospital, Bristol, UK. 12Department of Molecular Neuroscience and Reta Lilla Weston Laboratories, Institute of Neurology, UCL, London, UK. 13Laboratory of Neurogenetics, National Institute on Aging, National Institutes of Health, Bethesda, Maryland, United States of America. 14MRC Prion Unit, Department of Neurodegenerative Disease, UCL Institute of Neurology, London, UK. 15Dementia Research Centre, Department of Neurodegenerative Diseases, University College London, Institute of Neurology, London, UK. 16Department of Psychiatry, University of Bonn, Sigmund-Freud-Straße 25, 53105 Bonn, Germany. 17Institute for Molecular Psychiatry, University of Bonn, Bonn, Germany. 18Institute of Primary Medical Care, University Medical Center Hamburg-Eppendorf, Germany. 19Department of Psychiatry, Charité Berlin, Germany. 20Department of Psychiatry, Friedrich-Alexander-University Erlangen-Nürnberg, Germany. 21Department of Psychiatry and Psychotherapy, University Medical Center (UMG), Georg-August-University, Göttingen, Germany. 22Institute for Stroke and Dementia Research, Klinikum der Universität München, Marchioninistr. 15, 81377, Munich, Germany. 23Department of Neurology, Klinikum der Universität München, Marchioninistr. 15, 81377, Munich, Germany. 24Central Institute of Mental Health, Medical Faculty Mannheim, University of Heidelberg, Germany. 25Institute for Memory and Alzheimers Disease & INSERM, Sorbonne Universités, Pierre and Marie Curie University, Paris ; Institute for Brain and Spinal Cord Disorders (ICM), Department of Neurology, Hospital of Pitié-Salpêtrière. 26Centre for Geriatric Medicine and Section of Gerontopsychiatry and Neuropsychology, Medical School, University of Freiburg, Germany. 27Department of Psychiatry, University of Halle, Halle, Germany. 28Departments of Psychiatry, Neurology and Genetics, Washington University School of Medicine, St Louis, MO 63110, US. 29Department of Biology, Brigham Young University, Provo, UT, 84602, USA. 30Department of Mental Health Sciences, University College London, UK. 31The Wellcome Trust Sanger Institute, Wellcome Trust Genome Campus, Hinxton, Cambridge, UK. 32Department of Genomics, Life & Brain Center, University of Bonn, Bonn,
Mendelian randomization of CRP in 32 somatic and psychiatric outcomes

Germany. 39Department of Molecular Neuroscience, Institute of Neurology, University College London, Queen Square, London WC1N 3BG, UK. 34Deutsches Zentrum für Neurodegenerative Erkrankungen (DZNE), Bonn.

The Psoriasis and Psoriatic Arthritis Genetics Consortium

(name of co-author: affiliation(s))

James T Elder: Department of Dermatology, University of Michigan Ann Arbor, MI 48109, USA;
Ann Arbor Veterans Affairs Hospital, Ann Arbor, MI 48105 USA
Philip E Stuhr: Department of Dermatology, University of Michigan Ann Arbor, MI 48109, USA
Rajan P Nair: Department of Dermatology, University of Michigan Ann Arbor, MI 48109, USA
Trilokraj Tejasvi: Department of Dermatology, University of Michigan Ann Arbor, MI 48109, USA.
Johann E Gudjonsson: Department of Dermatology, University of Michigan Ann Arbor, MI 48109, USA
John J Voorhees: Department of Dermatology, University of Michigan Ann Arbor, MI 48109, USA.
Lam C Tsoi: Department of Biostatistics, Center for Statistical Genetics, University of Michigan Ann Arbor, MI 48109, USA
Jun Ding: Department of Biostatistics, Center for Statistical Genetics, University of Michigan Ann Arbor, MI 48109, USA
Yanming Li: Department of Biostatistics, Center for Statistical Genetics, University of Michigan Ann Arbor, MI 48109, USA
Hyun M Kang: Department of Biostatistics, Center for Statistical Genetics, University of Michigan Ann Arbor, MI 48109, USA;
Goncalo R Abecasis: Department of Biostatistics, Center for Statistical Genetics, University of Michigan Ann Arbor, MI 48109, USA
André Franke: Institute of Clinical Molecular Biology, Christian-Albrechts-University of Kiel, 24105 Kiel, Germany
Eva Ellinghaus: Institute of Clinical Molecular Biology, Christian-Albrechts-University of Kiel, 24105 Kiel, Germany
Stefan Schreiber: Institute of Clinical Molecular Biology, Christian-Albrechts-University of Kiel, 24105 Kiel, Germany
Chapter 6

Ulrich Mrowietz: Department of Dermatology, University Hospital, Schleswig-Holstein, Christian-Albrechts-University of Kiel, 24105 Kiel, Germany
Stephan Weidinger: Department of Dermatology, University Hospital, Schleswig-Holstein, Christian-Albrechts-University of Kiel, 24105 Kiel, Germany
Michael Weichenthal; Department of Dermatology, University Hospital, Schleswig-Holstein, Christian-Albrechts-University of Kiel, 24105 Kiel, Germany
Sören Mucha: Institute of Clinical Molecular Biology, Christian-Albrechts-University of Kiel, 24105 Kiel, Germany
Dafna D Gladman: Department of Medicine, Division of Rheumatology, University of Toronto, Toronto Western Hospital, Toronto, Ontario M5T 2S8, Canada
Fawnda J Pellett: Department of Medicine, Division of Rheumatology, University of Toronto, Toronto Western Hospital, Toronto, Ontario M5T 2S8, Canada
Vinod Chandran: Department of Medicine, Division of Rheumatology, University of Toronto, Toronto Western Hospital, Toronto, Ontario M5T 2S8, Canada
Cheryl F Rosen: Department of Medicine, Division of Dermatology, University of Toronto, Toronto Western Hospital, Toronto, Ontario M5T 2S8
Proton Rahman: Department of Medicine, Memorial University, St. John’s, Newfoundland A1C 5B8, Canada
Sulev Koks: Department of Physiology, Centre of Translational Medicine and Centre for Translational Genomics, University of Tartu, 50409 Tartu, Estonia
Külli Kingo: Department of Dermatology and Venerology, University of Tartu, 50409 Tartu, Estonia
Tonu Esko: Estonian Genome Center and Center of Translational Genomics; Estonian Biocenter; Institute of Molecular and Cell Biology, University of Tartu, 50409 Tartu, Estonia
Andres Metspalu: Estonian Genome Center and Center of Translational Genomics; Estonian Biocenter; Institute of Molecular and Cell Biology, University of Tartu, 50409 Tartu, Estonia
Peter Gregersen: Robert S. Boas Center for Genomics and Human Genetics, The Feinstein Institute for Medical Research, Manhasset, NY 11030
Andrew Henschel: National Psoriasis Foundation, Portland, OR 97223 USA
Marin Aurand National Psoriasis Foundation, Portland, OR 97223 USA
Bruce Bebo: National Psoriasis Foundation, Portland, OR 97223 USA
Mendelian randomization of CRP in 32 somatic and psychiatric outcomes

H. Erich Wichmann: Institute of Epidemiology I, Helmholtz Centre Munich, German Research Center for Environmental Health, 85764 Neuherberg, Germany; Institute of Medical Informatics, Biometry and Epidemiology, Ludwig-Maximilians-University, 81377 Munich, Germany; Klinikum Grosshadern, 81377 Munich, Germany

Christian Gieger: Institute of Genetic Epidemiology, Helmholtz Centre Munich, German Research Center for Environmental Health, 85764 Neuherberg, Germany

Thomas Illig: Research Unit Molecular Epidemiology, Helmholtz Centre Munich, German Research Center for Environmental Health, 85764 Neuherberg, Germany

Juliane Winkelmann: Department of Neurology, Technische Universität München, Munich, Germany; Institute of Human Genetics, Technische Universität München, Munich, Germany; Institute of Human Genetics, Helmholtz Zentrum Munich, German Research Center for Environmental Health, Munich, Germany

Susanne Moebus: Institute for Medical Informatics, Biometry and Epidemiology (IMIBE), University of Duisburg-Essen, Hufelandstr. 55, 45122 Essen, Germany

Karl-Heinz Jöckel: Institute for Medical Informatics, Biometry and Epidemiology (IMIBE), University of Duisburg-Essen, Hufelandstr. 55, 45122 Essen, Germany

Raimund Erbel: Clinic of Cardiology, West German Heart Centre, University Hospital of Essen, University Duisburg-Essen, Essen, Germany

Markus M. Nöthen: Institute of Human Genetics, University of Bonn, Bonn, Germany; Department of Genomics, Life & Brain Center, University of Bonn, Bonn, Germany

Henry W Lim Henry Ford Hospital, Detroit, Michigan, 48202, USA
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