
Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

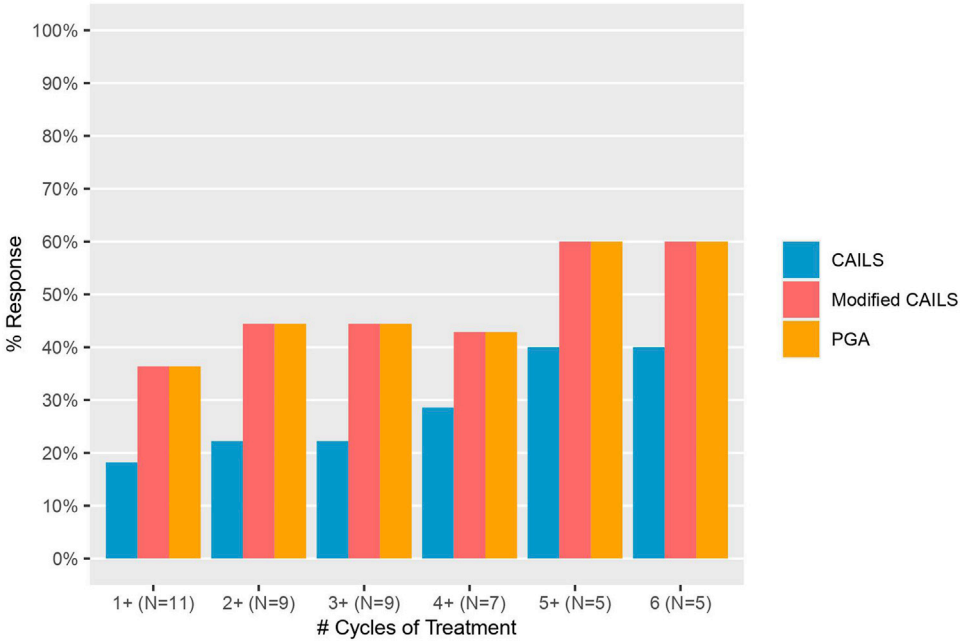


**Fig 1.** Response rates by Composite Assessment of Index Lesion Severity (*CAILS*), modified Composite Assessment of Index Lesion Severity (*mCAILS*), and Physician Global Assessment (*PGA*) according to number of treatment cycles completed.

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**Conflicts of interest**

Dr Mangold is an investigator for multiple studies in mycosis fungoides sponsored by Solagenix, MiRagen, Sun Pharma, and Elorac, and also serves on an advisory board for Kirin. Authors Severson, Brumfiel, Ginos, Kosiorek, Besch-Stokes, and Patel, and Drs Cumsky, Janeczek, Rule, DiCaudo, Rosenthal, and Pittelkow have no conflicts of interest to declare.

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## Genetic correlation analysis does not associate male pattern baldness with COVID-19

*To the Editor:* A recent study has reported an association between androgenetic alopecia in men, commonly known as male pattern baldness (MPB), and severe symptoms of COVID-19.[1] Here, we aimed to determine whether these epidemiological associations reflect shared genetic factors. Utilizing the genome-wide association study (GWAS) data, we estimated genetic correlations ($r_g$) between different phenotypes of MPB and COVID-19.

GWAS identifies genetic variants associated with differences in disease status among individuals.

**Table I.** Summary of genome-wide association study data analysed in this study and their estimated $P_{h2}$

| Phenotype | Study population | $h^2$ (SE) | $P_{h2}$ |
|---|---|---|---|
| MPB GWASs | | | |
| MPB* | 205,327 | 0.321 (0.026) | $9 \times 10^{-36}$ |
| MPB-2 vs MPB-1† | 38,044 vs 53,076 | 0.128 (0.014) | $1 \times 10^{-21}$ |
| MPB-3 vs MPB-1† | 44,304 vs 53,076 | 0.194 (0.023) | $3 \times 10^{-17}$ |
| MPB-4 vs MPB-1† | 30,225 vs 53,076 | 0.459 (0.037) | $7 \times 10^{-35}$ |
| MPB-2,3,4 vs MPB-1‡ | 112,573 vs 53,076 | 0.186 (0.017) | $2 \times 10^{-27}$ |
| COVID-19 GWASs | | | |
| COVID-19 positive vs population | 38,984 *vs* 1,644,784 | 0.001 ($3 \times 10^{-4}$) | $7 \times 10^{-6}$ |
| Hospitalized COVID-19 vs population | 8316 vs 1,549,095 | 0.003 ($6 \times 10^{-4}$) | $3 \times 10^{-7}$ |
| Very severe respiratory COVID-19 vs population | 5101 vs 1,383,241 | 0.004 ($7 \times 10^{-4}$) | $6 \times 10^{-8}$ |

*MPB*, Male pattern baldness; *GWAS*, genome-wide association study; $h^2$, observed scale heritability estimated by linkage disequilibrium score regression[4]; $P_{h2}$, *P* value for the $h^2$ estimate; *SE*, standard error of the $h^2$ estimate; *SNP*, single-nucleotide polymorphism.
*GWAS tested for association between "adjusted MPB pattern" and SNP genotypes using a linear mixed model.[2]
†GWASs tested for an association between a binary phenotype and SNP genotypes using a linear mixed model form http://www.nealelab.is/uk-biobank was utilized to obtain GWASs comparing MPB patterns with MPB-1 applying case-case GWAS.[3]
‡Directly resourced from http://www.nealelab.is/uk-biobank. Note that COVID-19 $h^2$ estimates appear small because they are on the observed scale as it is not possible to determine an appropriate population prevalence (required to estimate $h^2$ on the liability scale) for the COVID-19 binary phenotypes.

**Table II.** Linkage disequilibrium score regression genetic correlation results between male pattern baldness and COVID-19 phenotypes

| MPB phenotype | COVID-19 phenotype | $r_g$ (SE) | *P* |
|---|---|---|---|
| MPB | COVID-19 positive vs population | −0.078 (0.048) | .1048 |
| | Hospitalized COVID-19 vs population | −0.019 (0.042) | .6485 |
| | Very severe respiratory COVID-19 vs population | −0.026 (0.048) | .5846 |
| MPB-2 vs MPB-1 | COVID-19 positive vs population | −0.031 (0.072) | .6644 |
| | Hospitalized COVID-19 vs population | 0.058 (0.071) | .4146 |
| | Very severe respiratory COVID-19 vs population | 0.030 (0.068) | .6641 |
| MPB-3 vs MPB-1 | COVID-19 positive vs population | −0.120 (0.071) | .0929 |
| | Hospitalized COVID-19 vs population | −0.017 (0.057) | .7605 |
| | Very severe respiratory COVID-19 vs population | −0.004 (0.063) | .9552 |
| MPB-4 vs MPB-1 | COVID-19 positive vs population | −0.076 (0.054) | .1589 |
| | Hospitalized COVID-19 vs population | 0.035 (0.052) | .4973 |
| | Very severe respiratory COVID-19 vs population | 0.006 (0.052) | .9051 |
| MPB-2,3,4 vs MPB-1 | COVID-19 positive vs population | −0.091 (0.057) | .1087 |
| | Hospitalized COVID-19 vs population | 0.016 (0.051) | .7520 |
| | Very severe respiratory COVID-19 vs population | 0.004 (0.054) | .9374 |

*MPB*, Male pattern baldness; *P*, *P* value for the $r_g$ estimate; $r_g$, genetic correlation estimated by LDSC[4]; *SE*, standard error of the $r_g$ estimate.

GWAS data for MPB were sourced from the study by Yap et al[2] and http://www.nealelab.is/uk-biobank. The MPB GWAS enrolled male participants from the UK Biobank European population who were asked to choose a pattern from 4 options matching their baldness pattern: pattern 1 for no balding, pattern 2 for vertex balding, pattern 3 for crown balding, and pattern 4 for vertex plus crown balding.[2] In total, 5 GWASs testing for associations between single-nucleotide polymorphism (SNP) genotypes and [a] adjusted MPB patterns, [b] MPB-2 versus MPB-1, [c] MPB-3 versus MPB-1, [d] MPB-4 versus MPB-1, and [e] MPB-2,3,4 versus MPB1 were included in the present study. Data sets [b], [c] and [d] were obtained using the case-case GWAS approach.[3] GWAS results for COVID-19 phenotypes were obtained from the "The COVID-19 Host Genetics Initiative" resource (https://www.covid19hg.org/results/; round 5, Jan 18, 2021). These GWAS results show that susceptibility for COVID-19 and its severe symptoms is associated with genetic factors. Three COVID-19 GWAS phenotypes ("COVID-19 positive versus population," "Hospitalized COVID-19 versus population," and "Very severe respiratory COVID-19 versus population") were included. Table I provides a summary of the GWAS data sets used in this study.

Linkage disequilibrium score regression analysis was conducted to estimate heritability ($h^2$) and genome-wide genetic correlation ($r_g$) between MPB and COVID-19 phenotypes for approximately 1,000,000 autosomal SNPs.[4] All MPB and COVID-19 GWASs had significant heritability ($9 \times 10^{-36} < P_{b2} < 7 \times 10^{-6}$) and were subsequently utilized in the $r_g$ analyses (Table I).

We found no significant $r_g$ between the MPB and COVID-19 phenotypes (Table II). Furthermore, although not significant, the $r_g$ and previously reported associations are not directionally consistent.[1] For example, the increased risk for MPB has a negative $r_g$ with susceptibility ($r_g = -0.078$, $P = .1048$), hospitalization ($r_g = -0.019$, $P = .6485$), and severity ($r_g = -0.026$, $P = .5846$) of COVID-19.

Two possible limitations of this study need to be mentioned. First, the baldness pattern from UK Biobank is self-reported. Second, the linkage disequilibrium score regression $r_g$ was estimated utilizing autosomal SNPs and therefore did not evaluate X-linked genetic factors, including the androgen receptor gene, which has been implicated in both MPB and severe COVID-19.[5] However, the analysis of independent SNPs around androgen receptor gene found no correlation ($P > .05$) in risk effects between the MPB and COVID-19 GWAS phenotypes—consistent with the linkage disequilibrium score regression autosomal $r_g$ results.

Although we found no evidence for a global genetic correlation across MPB and COVID-19 phenotypes, given pleiotropic effects, where genetic variants influence multiple traits, are widespread in human complex traits (https://www.ebi.ac.uk/gwas/), it is possible that other/specific genes, including genes on chromosome X, could contribute to MPB and COVID-19 risk—noting many pleiotropic variants with consistent effect directions are required to produce a significant genetic correlation.

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**Conflicts of interest**

None disclosed.

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## An increase in respiratory protection device injuries associated with the COVID-19 pandemic

*To the Editor:* The COVID-19 pandemic abruptly changed many people's lives. While social distancing, quarantining, and personal protective equipment (PPE) have positively impacted the pandemic's progression, ancillary consequences have occurred.[1,2] Prior to the COVID-19 pandemic, the use of respiratory protection equipment was largely limited to health care and industrial settings. However, as PPE use by the general population increased, reports of dermatologic reactions have also increased.[3] This study reports on the epidemiology of respiratory protection equipment-related injuries in the United States associated with the COVID-19 pandemic.

The data for this study was obtained from the National Electronic Injury Surveillance System