**Greater risk of severe COVID-19 in non-White ethnicities is not explained by cardiometabolic, socioeconomic, or behavioural factors, or by 25(OH)-vitamin D status: study of 1,326 cases from the UK Biobank**

Dr. Zahra Raisi-Estabragh1,2, Ms. Celeste McCracken1, Dr. Mae S. Bethell3, Ms. Jackie Cooper1, Prof. Cyrus Cooper4,5,6, Prof Mark J. Caulfield1, Prof Patricia B. Munroe1, Prof. Nicholas C. Harvey4,5, Prof. Steffen E. Petersen\*1,2

1. William Harvey Research Institute, NIHR Barts Biomedical Research Centre, Queen Mary University of London, London, UK

2. Barts Heart Centre, St Bartholomew’s Hospital, Barts Health NHS Trust, London, UK

3. North West Anglia NHS Foundation Trust, Hinchingbrooke Hospital, Huntingdon, UK

4. MRC Lifecourse Epidemiology Unit, University of Southampton, Southampton, UK

5. NIHR Southampton Biomedical Research Centre, University of Southampton and University Hospital Southampton NHS Foundation Trust, Southampton, UK

6. NIHR Biomedical Research Centre, University of Oxford, Oxford, UK

**\*Corresponding author:** Professor Steffen E. Petersen. William Harvey Research Institute, NIHR Barts Biomedical Research Centre, Queen Mary University of London, London, UK; Email: s.e.petersen@qmul.ac.uk; Telephone: +44-2078826902

**Abstract**

**Background**

We examined whether the greater severity of coronavirus disease 2019 (COVID-19) amongst men and non-White ethnicities is explained by cardiometabolic, socio-economic, or behavioural factors.

**Methods**

We studied 4,510 UK Biobank participants tested for COVID-19 (positive, *n*=1,326). Multivariate logistic regression models including age, sex, and ethnicity were used to test whether addition of: 1)cardiometabolic factors (diabetes, hypertension, high cholesterol, prior myocardial infarction, smoking, BMI); 2)25(OH)-vitamin D; 3)poor diet; 4)Townsend deprivation score; 5)housing (home type, overcrowding); or 6)behavioural factors (sociability, risk taking) attenuated sex/ethnicity associations with COVID-19 status.

**Results**

There was over-representation of men and non-White ethnicities in the COVID-19 positive group. Non-Whites had, on average, poorer cardiometabolic profile, lower 25(OH)-vitamin D, greater material deprivation, and were more likely to live in larger households and flats/apartments. Male sex, non-White ethnicity, higher BMI, Townsend deprivation score, and household overcrowding were independently associated with significantly greater odds of COVID-19. The pattern of association was consistent for men and women; cardiometabolic, socio-demographic and behavioural factors did not attenuate sex/ethnicity associations.

**Conclusions**

Sex and ethnicity differential pattern of COVID-19 is not adequately explained by variations in cardiometabolic factors, 25(OH)-vitamin D levels, or socio-economic factors. Investigation of alternative biological pathways and different genetic susceptibilities is warranted.

**Introduction**

The coronavirus disease 2019 (COVID-19) pandemic has to date resulted in over 6 million cases and 376,000 deaths worldwide1. Growing reports highlight men and Black and Minority Ethnic (BAME) cohorts as at higher risk of adverse COVID-19 outcomes2,3. Variations in cardiometabolic disease burden4, oestrogen pathway activity5, vitamin D levels6, and angiotensin‐converting enzyme (ACE) 2 receptor expression7 have been proposed as potential explanations for the differential pattern of disease severity. Furthermore, disparities in socio-economic standards, housing conditions, socialisation habits, and risk perception have potential implications for risk of exposure and transmission. Understanding the significance of these factors is urgently needed to inform public health and research efforts.

We therefore investigated, in the UK Biobank (UKB) cohort, whether differential patterns of COVID-19 incidence and severity by sex and ethnicity might be explained by cardiometabolic, socio-economic, lifestyle, and behavioural exposures.

**Methods**

**Setting and study population**

UKB is a prospective cohort study of over half a million men and women from across the UK covering a range of urban and rural settings. Recruitment was between 2006-2010 through postal invite of individuals aged 40-69 years-old identified through National Health Service (NHS) registers. All individuals living within 10 miles of one of 22 UK Biobank assessment centres were invited to participate. Individuals who were unable to consent were not recruited. Baseline assessment included detailed characterisation of socio-demographics, lifestyle, health, a series of physical measures, and blood biochemistry. The protocol is publicly available8. Data linkage with Hospital Episode Statistics (HES) enables prospective tracking of health outcomes for all participants with conditions recorded according to international classification of disease (ICD). Incidence of key events, such as myocardial infarction (MI), are algorithmically defined by cross-checking over multiple data sources9. Linkage with Public Health England has enabled rapid release of linked COVID-19 test results of UKB participants to researchers10. The latest data release (29/05/2020) included test results from 16/03/2020 to 18/05/2020. As UK testing during this period was almost entirely restricted to hospitalised patients, researchers have been advised that COVID-19 positive status can be taken as surrogate for severe disease11.

**Exposures**

We considered relevant demographic (age, sex, ethnicity), biological (cardiometabolic, 25(OH)-vitamin D status), socio-economic (material deprivation, type of home, household overcrowding, poor diet quality), and behavioural (sociability, attitude to risk) exposures (Supplementary Table 1).

We used age and sex as recorded at baseline. For consistency with wider UK classification, we document ethnicity as White and non-White. For the latter we report breakdown of ethnicities as per existing UKB categories: Black (Caribbean, African, any other Black background), Asian (Indian, Pakistani, Bangladeshi, any other Asian background), Chinese, Mixed (White and Black Caribbean, White and Black African, White and Asian, Any other mixed background), and “other”. Townsend deprivation score is reported by the UKB as a measure of material deprivation calculated at baseline: zero, positive, and negative scores correspond to average, higher and lower levels of deprivation respectively, relative to national averages12. We used type of housing as a binary variable comprising communal living spaces (flat, apartment, sheltered accommodation) vs stand-alone housing (house, bungalow). We considered household overcrowding based on self-report of household size and intergenerational cohabitation. Socialisation habits were defined per self-reports of frequency of family/friend visits and participation in regular leisure activities outside the home. Attitude to risk was assessed using self-report of tendency “to take risks”. BMI was calculated from height and weight recorded at baseline. Smoking status was based on self-report. Hypertension, diabetes, and hypercholesterolaemia were defined through cross-checking across self-report and HES data. A list of ICD codes used is available in Supplementary Table 2. Prior MI was obtained from UKB algorithmically defined health outcomes. We used serum 25(OH)-vitamin D levels measured at baseline (CLIA analysis on a DiaSorin Ltd. LIASON XL), limiting to results between 10-375 nmol/L based on the manufacturer’s analytic range13. We adjusted for seasonality by regressing vitamin D on month of sampling as a factor, this allowed derivation of vitamin D adjusted to the same month for each participant. There were differences in vitamin D levels and degree of seasonal variation by ethnicity (Figure 1D). We therefore performed seasonality adjustment separately for White and non-White ethnicities and added the intercept to the adjusted variables to maintain the difference between the two groups. We considered processed meat intake as a marker of poor diet quality. We converted self-reported weekly intake frequencies into probabilities of daily intake and multiplied by portion size to derive a continuous measure of daily consumption in grams, as previously published using this dataset14,15.

**Ethics**

This study was covered by the ethics approval for UKB studies from the NHS National Research Ethics Service on 17th June 2011 (Ref 11/NW/0382) and extended on 10th May 2016 (Ref 16/NW/0274).

**Statistical analysis**

Statistical analysis was performed using R Version 3.6.2 [R Core Team (2019). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. URL [https://www.R-project.org/](https://www.r-project.org/)], and RStudio Version 1.2.5019 [RStudio Team (2015). RStudio: Integrated Development for R. RStudio, Inc., Boston, MA URL <http://www.rstudio.com/>].

UKB participants were grouped according to COVID-19 status: test positive, test negative, and untested. In analysis of an earlier data release, we demonstrated similar associations when comparing the untested cohort with both the test negatives and test positives; suggesting that comparison with the whole cohort reveals associations with general hospitalisation rather than specifically with COVID-1916. Therefore, to avoid bias relating to hospitalisation, in the present study, we limited to modelling within the tested cohort. We performed analyses in the whole tested sample, and separately in men and women. Logistic regression models were first used to examine univariate associations. We then undertook individual multivariate models for each hypothesis tominimise loss of participants due to missingness from adding multiple variables simultaneously. We defined a final model using variables noted to be important from previous model permutations. We tested for multicollinearity setting a variance inflation factor (VIF) cut-off of 2.5. We present odds ratio (OR) for each exposure with the corresponding 95% confidence interval (CI) and p-value.

**RESULTS**

**Population characteristics**

*Sex and ethnicity*

Test results for 4,510 participants were available (positive, *n*=1,326; negative, *n*=3,184). Baseline characteristics are summarised in Table 1. Comparisons with the untested cohort (*n*=497,996) and characteristics by sex and ethnicity are summarised in Supplementary Tables 3, 4, and 5. There was over-representation of men and non-White ethnicities in the test positive cohort (Figure 1A, Figure 1B). Individuals of Black and Asian ethnicity were most disproportionately affected with Black ethnicities contributing over 3.5 times the number of positive cases than their representation in the untested cohort (Supplementary Table 3, Figure 1B).

*Cardiometabolic factors and vitamin D*

Men and non-White ethnicities had overall greater burden of cardiometabolic morbidities compared to women and White cohorts respectively (Figure 1E, Figure 1F). Serum 25(OH)-vitamin D levels were, on average, higher in White ethnicities than non-White cohorts (Figure 1D).

*Socio-demographic and behavioural factors*

In comparison to the test negatives, those with a positive test had greater levels of material deprivation and were more likely to live in crowded households (Figure 1C). Non-Whites had higher levels of material deprivation by Townsend score compared to those of White ethnicity (Supplementary Table 4). The frequency of family/friend visits and leisure activities outside the home were similar between the test positive and test negative groups. There was greater tendency to risk taking behaviour in the test positive cohort, which was greater in men vs women and in non-White vs White ethnicities.

**Univariate associations of exposures with COVID-19 positive status**

We tested the univariate association of all defined exposures with COVID-19 positive status within the tested cohort (Supplementary Table 6). Male sex, non-White ethnicity, higher BMI, greater material deprivation, and greater household overcrowding (household size, generations in household) were associated with increased odds of COVID-19 positive test. More frequent visits from family/friends were associated with lower risk of COVID-19 hospitalisation, perhaps reflecting the role of social support in enabling individuals to remain at home when ill (given that a positive test implied hospital attendance). There was a negative association between age and COVID-19 positivity, which may reflect the narrow range and distribution of ages in the sample. Testing separately in men, non-White ethnicity, greater material deprivation, and higher BMI were the only statistically significant exposures. For women, additionally, lower 25(OH)-vitamin D status, greater household overcrowding (household size, generations in household), and greater risk taking behaviour were associated with COVID-19 positivity.

**Independent associations of specific exposures with COVID-19 status**

*Cardiometabolic factors*

We undertook multivariate logistic regression models incorporating sex, age, ethnicity, smoking, BMI, diabetes, hypertension, high cholesterol, and prior MI (Table 2, Model 1). Male sex and non-White ethnicity were associated with greater odds of COVID-19 positive status with OR 1.28 (1.12, 1.46) and 1.78 (1.43, 2.20) respectively. Every 1kg/m2 of BMI was associated with 1.03 (1.01, 1.04) greater odds of COVID-19 positivity. There was a borderline negative association with age 0.99 [0.98, 1.00], which remained significant for women in sex-stratified analysis. There was no evidence of attenuation (compared with the crude models) in the associations with non-White ethnicity and higher BMI, consistent across men and women.

*25(OH)-vitamin D status and poor diet quality*

In multivariate logistic regression models incorporating sex, age and ethnicity, there was no significant association between season-adjusted 25(OH)-vitamin D status and COVID-19 positivity (Table 2, Model 2). Similarly, in a separate model, adjustment for sex, age, and ethnicity demonstrated no statistically significant association between processed meat consumption and COVID-19 status (Table 2, Model 3). In both models, male sex and non-White ethnicity were associated with higher odds of COVID-19 positive test across men and women, with no evidence of attenuation.

*Material deprivation*

We tested the effect of material deprivation in multivariate models with mutual adjustment for sex, age, and ethnicity (Table 3, Model 4). There was a small, but statistically significant association between greater material deprivation and higher odds of COVID-19 positivity [OR 1.03 (1.01, 1.05)]. There remained strong and significant associations with male sex [OR 1.27 (1.11, 1.45)] and non-White ethnicity [OR 1.67 (1.34, 2.07)].

*Housing conditions*

We considered the effect of housing conditions in multivariate logistic regression models including sex, age, ethnicity, home type, and household size. In the whole sample, male sex, non-White ethnicity, and greater household size were associated with greater odds of COVID-19 positivity (Table 3, Model 5). Testing separately in men and women, non-White ethnicity was the only exposure which remained significantly associated with COVID-19 status. Attenuation of associations with household size is likely due to the small effect size and limited heterogeneity of the exposure in each of the sexes individually.

*Socialisation habits and attitudes to risk*

We undertook separate multivariate logistic regression models testing for associations between COVID-19 status, socialisation habits and risk-taking attitude (Supplementary Table 7) whilst adjusting for age, sex, and ethnicity. Statistically significant associations were observed with male sex and non-White ethnicity which were not attenuated from crude models by adjustment for socialisation or risk-taking attitude, which did not show significant associations.

**Final model**

We built a final multivariate logistic regression model, with covariates selected based on previous model permutations including sex, age, ethnicity, BMI, Townsend score, and household size (Table 3, Model 6). Male sex and non-White ethnicity were associated with greater odds of COVID-19 positivity: OR 1.23 (1.08, 1.41) and 1.59 (1.26, 1.99) respectively. Every 1kg/m2 increase in BMI was associated with 1.02 (1.01, 1.03) greater odds of COVID-19 positivity and for every additional person living in the same household the odds increased by 1.09 (1.03, 1.16).

**Discussion**

**Main finding of this study**

In 4,510 UKB participants tested for COVID-19 in a hospital setting, male sex, non-White ethnicity, higher BMI, and greater household size were associated with significantly greater odds of a positive result. Despite variation in burden of cardiometabolic morbidities, 25(OH)-vitamin D levels, and material deprivation by sex and ethnicity, these factors were not significantly associated with COVID-19 positivity and did not explain the strong association with ethnicity. The pattern of associations did not vary between men and women.

**What is already known on this topic**

Mounting evidence suggests disproportionate adverse effects of COVID-19 in non-White ethnicities2. UK national audit data demonstrates that up to one-third of COVID-19 patients requiring intensive care are from BAME backgrounds, a rate far greater than their representation in the general population17. An analysis of COVID-19 deaths amongst NHS staff, found that 64% of deaths were in BAME cohorts, markedly disproportionate to their 20% contribution to the NHS workforce18. The latest report from the Office of National Statistics (ONS) also demonstrates greater risk of COVID-19 mortality in BAME groups19; individuals of Black ethnicity had over 3.5 times greater risk of COVID-19 death compared to Whites, followed by Asian ethnicities19. Similarly, in the USA, there has been growing concern over the disproportionate number of COVID-19 deaths amongst African Americans20. These patterns are echoed across Europe, with Nordic countries reporting as much as ten times greater risk of COVID-19 in Somali populations21. We had previously documented this preponderance of cases amongst BAME individuals in our analysis of the initial UKB data release16; here we have confirmed the observation in the larger dataset, and importantly demonstrated a non-uniform impact across different BAME groups with highest rates amongst Black followed by Asian ethnicities.

The greater cardiometabolic burden in both BAME and male cohorts has been proposed as potentially important in driving adverse COVID-19 outcomes. In our analysis, cardiometabolic morbidities were not significantly associated with COVID-19 status in multivariate models and did not attenuate sex and ethnicity associations. This suggests that the greater cardiometabolic burden in non-White ethnicities does not account for the adverse COVID-19 outcomes in this group.

Consistent with our findings, data from the UK and USA highlight obesity as a marker of poor COVID-19 outcomes, such as requirement for intensive care22. There are suggestions of a possible pathophysiological link between adiposity and COVID-19 severity. Wide expression of ACE2 receptors within adipose tissue is thought to promote binding and cellular entry of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)23. It has been suggested that adipose tissue may act as a “viral reservoir” thereby contributing to a more prolonged and severe illness23. In addition, adipose tissue is a known source of inflammatory cytokines, such as Interleukin 624. This is hypothesised to be linked to the association of adiposity with greater likelihood of cytokine storms and the consequent risk of severe respiratory complications in COVID-19. Indeed, studies have demonstrated association of higher Interleukin 6 levels with respiratory failure and requirement for mechanical ventilation in COVID-19 patients25. Greater adiposity, as well as non-White ethnicity, is associated with lower 25(OH)-vitamin D status. Although the active 1,25(OH)2-vitamin D form has immune system functions26, evidence linking low 25(OH-vitamin D (the circulating storage form, and poorly correlated with 1,25(OH)2-vitamin D) with COVID-19 disease have been contradictory27. In our study, we found no independent associations between 25(OH)-vitamin D status and COVID-19 disease suggesting that the relationship is confounded by ethnicity and BMI. Interestingly, the BMI association was retained in multivariate models, suggesting a possible independent role for adiposity, which clearly deserves further investigation.

Socio-economic deprivation is associated with poorer global health outcomes28. It has been suggested that ethnic differences in COVID-19 severity may relate to clustering of material deprivation with BAME status29. In the UKB material deprivation is reported using the Townsend score, which is based on four factors- employment, car ownership, home ownership, and household overcrowding. Consistent with national reports, we found higher material deprivation in non-White participants. In multivariate models including age, sex, ethnicity, and Townsend score, there were significantly greater odds of COVID-19 with greater material deprivation whilst the association with ethnicity appeared strong and significant. Testing separately for the effect of household overcrowding, this exposure appeared significant independent of sex, ethnicity, age, and home type. This suggests that it is may not be global economic deprivation, but specific aspects relating to household overcrowding that has relevance to COVID-19. Consistent with these observations, a survey of COVID-19 cases from New York reports the highest number of cases occurring in areas with the largest average household size30. Furthermore, analysis of UK cases by the ONS also demonstrates that material deprivation does not adequately explain the ethnic disparities in COVID-19 outcomes19.

Behavioural factors, in particular attitudes that may compromise adherence to lockdown measures, have been proposed as potentially important in determining risk of exposure to SARS-CoV-231,32. In our analysis, we did not find socialisation habits and attitude to risk to be significantly important in conferring COVID-19 positive status.

**What this study adds**

This study is consistent with growing reports of higher risk of severe COVID-19 in men and non-White ethnicities, in particular Black populations. The augmented risk in BAME populations is non-uniform and disproportionately affects Black and Asian ethnicities. Higher BMI, greater material deprivation, and household overcrowding are independent risk factors for COVID-19. The sex and ethnicity differential pattern of COVID-19 is not adequately explained by variations in cardiometabolic factors, 25(OH)-vitamin D levels, socio-economic, or behavioural factors. Thus, investigation of alternative biological pathways and genetic susceptibilities is warranted.

**Limitations of this study**

Given the observational nature of the study, we cannot discern causal relationships, and although we controlled for a wide range of covariates, the possibility of residual confounding should be considered. The vitamin D levels used in this analysis are based on measurements taken at the UKB baseline visit; therefore, we cannot account for possible changes that may have occurred since this measurement was taken. However, there is evidence that vitamin D status tends to track with time, particularly after adjustment for season of blood draw33, 34 (as we present in the current paper) and there is no reason to expect population level shifts in vitamin D levels in this time period, therefore our analysis has validity. Studies with more recent vitamin D measures would be of interest. The limited age range in this dataset precludes widely generalisable conclusions about the effects of age. Occupational factors may have relevance in determining risk of exposure and viral transmission; this topic requires detailed dedicated study. Aggregating all BAME populations may overlook important differences between ethnicities; studies in samples with greater ethnic diversity are needed.

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**Table 1. Baseline demographics by COVID-19 status**

|  |  |  |
| --- | --- | --- |
|  | Test positive (*n*=1,326) | Test negative (*n*=3,184) |
| Men | 696 (52.5%) | 1,505 (47.3%) |
| Age | 68.11 (± 9.23) | 68.91 (± 8.72) |
| White ethnicity | 1,141 (86.0%) | 2,927 (91.9%) |
| Non-White ethnicity | 174 (13.1%) | 241 (7.6%) |
| Black ethnicity | 76 (5.7%) | 91 (2.9%) |
| Asian ethnicity | 60 (4.5%) | 78 (2.4%) |
| Chinese ethnicity | 6 (0.5%) | 3 (0.1%) |
| Mixed ethnicity | 9 (0.7%) | 24 (0.8%) |
| Other ethnicity\* | 34 (2.6%) | 61 (1.9%) |
| Smoking (current or previous) | 683 (51.5%) | 1,653 (51.9%) |
| Processed meat intake (g/day) | 17.08 (± 15.67) | 16.33 (± 15.00) |
| BMI (kg/m2) | 28.04 [± 6.47] | 27.41 [± 6.37] |
| Diabetes | 217 (16.4%) | 449 (14.1%) |
| Hypertension | 624 (47.1%) | 1,457 (45.8%) |
| High cholesterol | 437 (33.0%) | 1,034 (32.5%) |
| Prior myocardial infarction | 96 (7.2%) | 242 (7.6%) |
| Vitamin D (nmol/L)\*\* | 33.88 [± 27.01] | 35.45 [± 26.78] |
| Townsend deprivation score | -0.91 [± 5.34] | -1.55 [± 5.00] |
| Home type (flat/apartment) | 191 (14.4%) | 455 (14.3%) |
| Household size | 2.50 (± 1.31) | 2.32 (± 1.22) |
| Number of generations in household | 1.41 (± 0.52) | 1.35 (± 0.50) |
| Family/friend visits | 975 (73.5%) | 2,438 (76.6%) |
| Regular leisure activity | 897 (67.6%) | 2,124 (66.7%) |
| Tendency to take risks | 404 (30.5%) | 916 (28.8%) |

**Table 1 footnote:** Results are number (percentage) for categorical and mean (standard deviation) or median [interquartile range] for continuous variables. \*Ethnicity was missing for n=11 test positive and n=16 test negative participants, these participants are included as part of “other ethnicity” in this table but have been excluded from subsequent modelling. \*\*Vitamin D has been adjusted for seasonality.

**Table 2. Multivariate logistic regression models testing the role of cardiometabolic factors (Model 1), vitamin D (Model 2), and poor diet (Model 3) in determining risk of COVID-19**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | Exposures | Whole tested sample  *n=*4,510 | Men  *n=*2,201 | Women  *n=*2,309 |
| *Model 1: sex, age, ethnicity, smoking, BMI, diabetes, hypertension, high cholesterol, prior MI* | Male sex | 1.28\* [1.12, 1.46] | – | – |
|  | 4.0510-4 | – | – |
| Age | 0.99\* [0.98, 1.00] | 1.00 [0.98, 1.01] | 0.99\* [0.97, 1.00] |
|  | 0.0157 | 0.5128 | 0.0097 |
| Non-White ethnicity | 1.78\* [1.43, 2.20] | 2.07\* [1.50, 2.84] | 1.55\* [1.15, 2.09] |
|  | 1.8810-7 | 7.9010-6 | 0.0040 |
| Smoking (previous/current) | 1.02 [0.89, 1.16] | 1.12 [0.92, 1.36] | 0.91 [0.75, 1.10] |
|  | 0.7961 | 0.2533 | 0.3352 |
| BMI (kg/m2) | 1.02\* [1.01, 1.03] | 1.03\* [1.01, 1.05] | 1.02 [1.00, 1.03] |
|  | 0.0015 | 0.0051 | 0.0537 |
| Diabetes | 1.08 [0.88, 1.32] | 1.06 [0.82, 1.38] | 1.08 [0.77, 1.49] |
|  | 0.4781 | 0.6529 | 0.6665 |
| Hypertension | 1.01 [0.86, 1.18] | 0.93 [0.74, 1.16] | 1.11 [0.89, 1.40] |
|  | 0.8875 | 0.5004 | 0.3563 |
| High cholesterol | 0.97 [0.82, 1.15] | 1.04 [0.83, 1.31] | 0.89 [0.68, 1.15] |
|  | 0.7479 | 0.7108 | 0.3690 |
| Prior MI | 0.89 [0.68, 1.16] | 0.85 [0.62, 1.15] | 0.97 [0.55, 1.65] |
|  | 0.4041 | 0.2961 | 0.8990 |
| *Model 2: age, sex, ethnicity, vitamin D* | Male sex | 1.31\* [1.14, 1.50] | – | – |
|  | 1.8510-4 | – | – |
| Age | 0.99\* [0.98, 1.00] | 1.00 [0.99, 1.01] | 0.99\* [0.97, 1.00] |
|  | 0.0166 | 0.5500 | 0.0073 |
| Non-White ethnicity | 1.77\* [1.41, 2.22] | 2.02\* [1.45, 2.82] | 1.60\* [1.16, 2.18] |
|  | 9.2710-7 | 3.5110-5 | 0.0038 |
| Vitamin D | 1.00 [1.00, 1.00] | 1.00 [1.00, 1.01] | 1.00 [1.00, 1.01] |
|  | 0.7185 | 0.7464 | 0.9288 |
| *Model 3: age, sex, ethnicity, processed meat* | Male sex | 1.26\* [1.10, 1.44] |  |  |
|  | 8.5510-4 |  |  |
| Age | 0.99\* [0.98, 1.00] | 1.00 [0.99, 1.01] | 0.99\* [0.98, 1.00] |
|  | 0.0144 | 0.4993 | 0.0082 |
| Non-White ethnicity | 1.81\* [1.46, 2.24] | 2.08\* [1.52, 2.85] | 1.62\* [1.21, 2.17] |
|  | 4.1810-8 | 4.9510-6 | 0.0011 |
| Processed meat intake (100 grams/day) | 1.26 [0.81, 1.94] | 1.01 [0.57, 1.77] | 1.83 [0.91, 3.66] |
| 0.3032 | 0.9742 | 0.0871 |

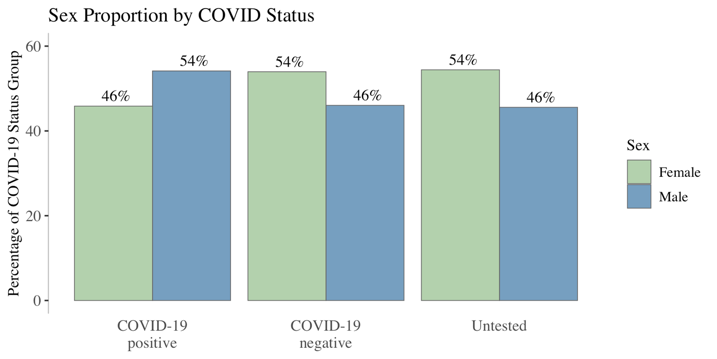
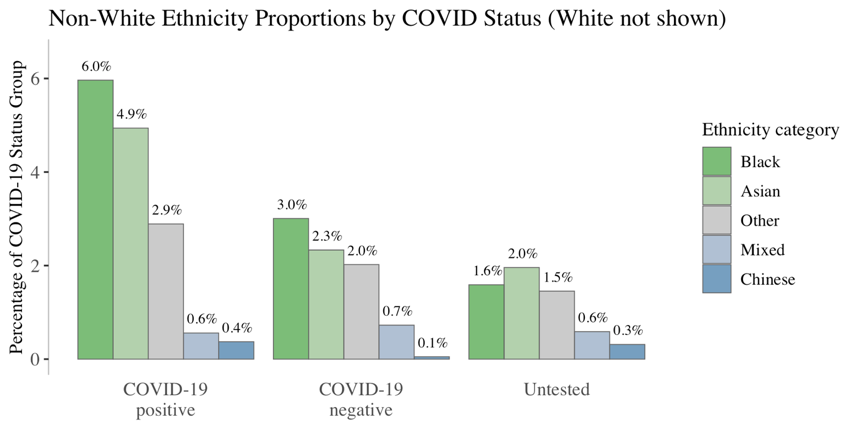
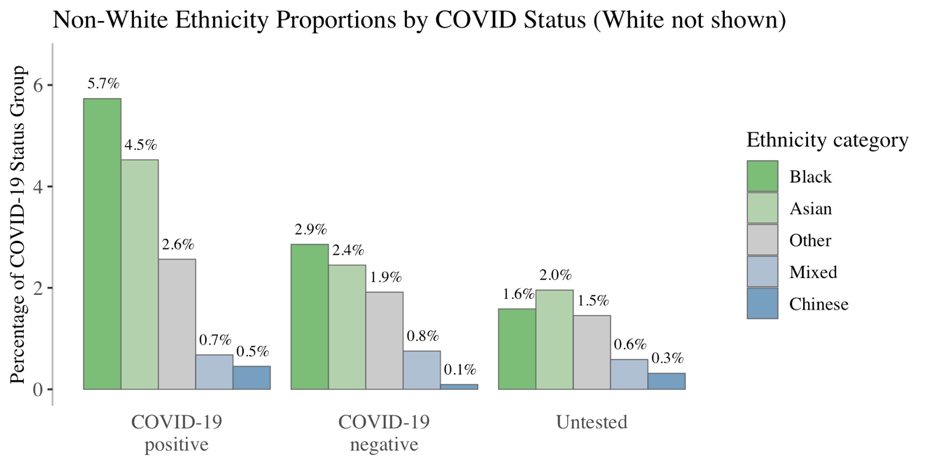
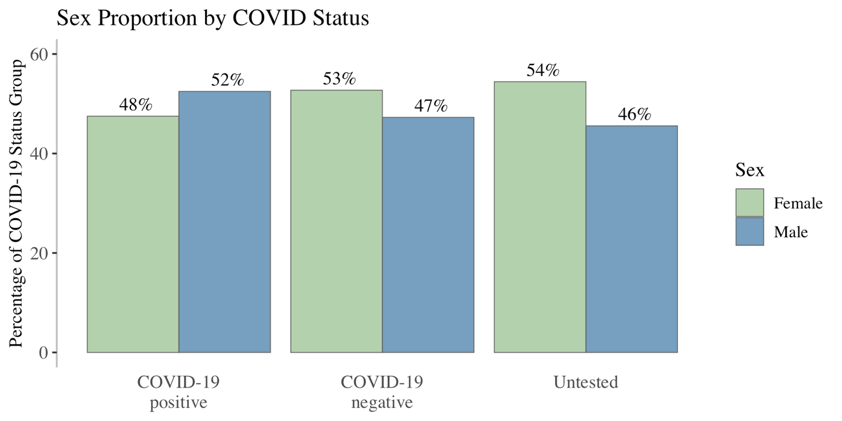
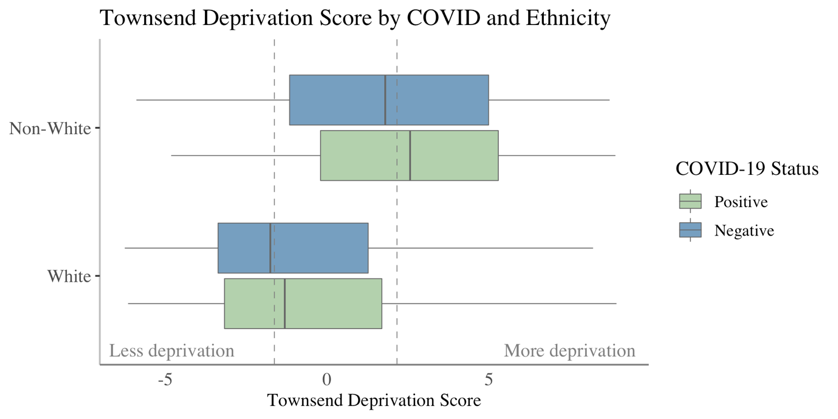
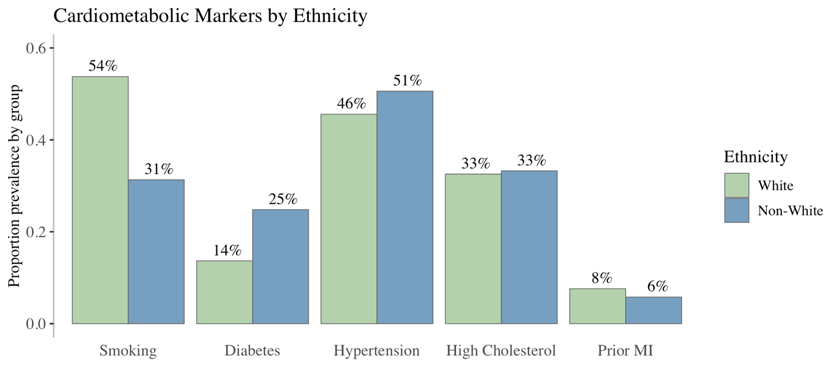
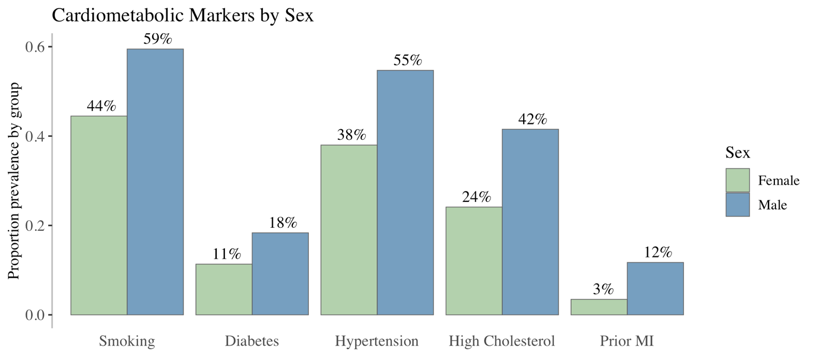
**Table 2 footnote:** Results are odds ratios, 95% confidence interval, and p-values for each exposure from three separate models (1, 2, and 3). Exposures are mutually adjusted.

**Table 3. Multivariate logistic regression models testing the role of material deprivation (Model 4), housing conditions (Model 5), and final model (Model 6) in determining risk of COVID-19**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | Exposures | Whole tested sample  *n=*4,510 | Men  *n=*2,201 | Women  *n=*2,309 |
| *Model 4: age, sex, ethnicity, Townsend deprivation score* | Male sex | 1.27\* [1.11, 1.45] | – | – |
|  | 3.8710-4 | – | – |
| Age | 0.99\* [0.98, 1.00] | 1.00 [0.99, 1.01] | 0.99\* [0.98, 1.00] |
|  | 0.0222 | 0.6323 | 0.0089 |
| Non- White ethnicity | 1.67\* [1.34, 2.07] | 1.92\* [1.39, 2.64] | 1.49\* [1.11, 2.01] |
|  | 3.9410-6 | 6.1510-5 | 0.0084 |
| Townsend deprivation score | 1.03\* [1.01, 1.05] | 1.03\* [1.00, 1.06] | 1.03\* [1.00, 1.06] |
|  | 0.0024 | 0.0402 | 0.0232 |
| *Model 5: age, sex, ethnicity, home type, household size\** | Male sex | 1.24\* [1.09, 1.42] | – | – |
|  | 0.0016 | – | – |
| Age | 1.00 [0.99, 1.01] | 1.00 [0.99, 1.01] | 0.99 [0.98, 1.00] |
|  | 0.3827 | 0.8207 | 0.1655 |
| Non- White ethnicity | 1.73\* [1.39, 2.17] | 1.86\* [1.33, 2.59] | 1.66\* [1.22, 2.24] |
|  | 1.3610-6 | 2.6010-4 | 0.0011 |
| Home type | 0.98 [0.80, 1.20] | 1.05 [0.80, 1.38] | 0.90 [0.66, 1.22] |
|  | 0.8650 | 0.7044 | 0.4918 |
| Household size | 1.08\* [1.02, 1.14] | 1.08 [0.99, 1.18] | 1.07 [0.99, 1.16] |
|  | 0.0140 | 0.0764 | 0.0941 |
| *Model 6 “final model”: age, sex, ethnicity, BMI, Townsend deprivation score, Household size* | Male sex | 1.23\* [1.08, 1.41] | – | – |
|  | 0.0021 | – | – |
| Age | 1.00 [0.99, 1.00] | 1.00 [0.99, 1.01] | 0.99 [0.98, 1.00] |
|  | 0.3648 | 0.8297 | 0.1674 |
| Non-White ethnicity | 1.59\* [1.26, 1.99] | 1.74\* [1.24, 2.45] | 1.50\* [1.10, 2.04] |
|  | 7.8510-5 | 0.0015 | 0.0105 |
| BMI (kg/m2) | 1.02\* [1.01, 1.03] | 1.03\* [1.01, 1.05] | 1.02\* [1.00, 1.03] |
|  | 9.7110-4 | 0.0036 | 0.0476 |
| Townsend deprivation score | 1.03\* [1.01, 1.06] | 1.04\* [1.01, 1.07] | 1.03\* [1.00, 1.06] |
|  | 0.0011 | 0.0133 | 0.0319 |
| Household size | 1.09\* [1.03, 1.16] | 1.09 [1.00, 1.18] | 1.10\* [1.01, 1.19] |
|  | 0.0022 | 0.0529 | 0.0203 |

**Table 3 footnote:** Results are odds ratios, 95% confidence interval, and p-values for each exposure from three separate models (4, 5, and 6). BMI: body mass index; COVID-19: coronavirus disease 2019. Exposures are mutually adjusted. \* Initial analyses additionally included number of generations in household, however, we observed significant multicollinearity between this variable and household size with higher VIF in the latter, hence it was removed from the final model. Exposures are mutually adjusted.

**Figure 1. Baseline participant characteristics**



A

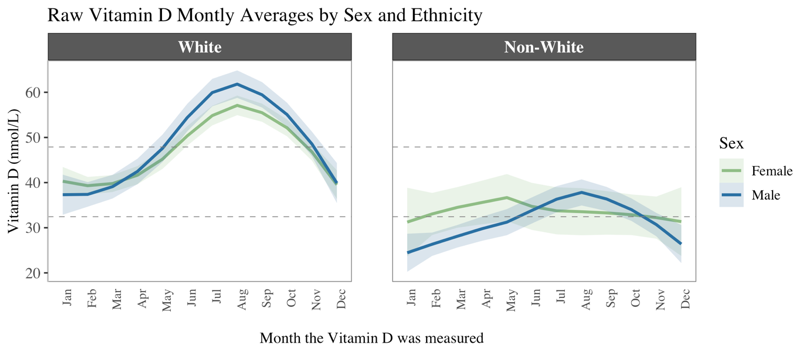
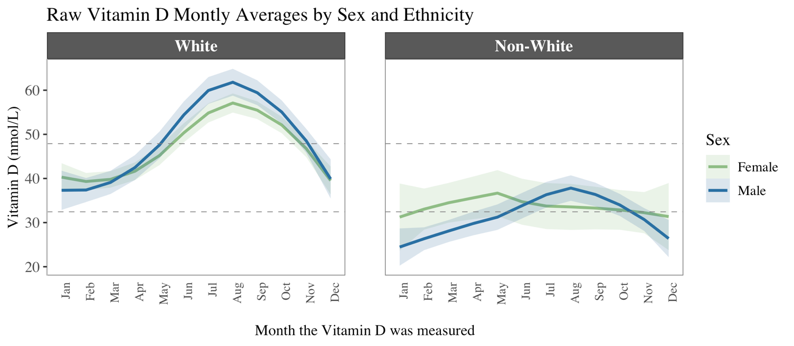
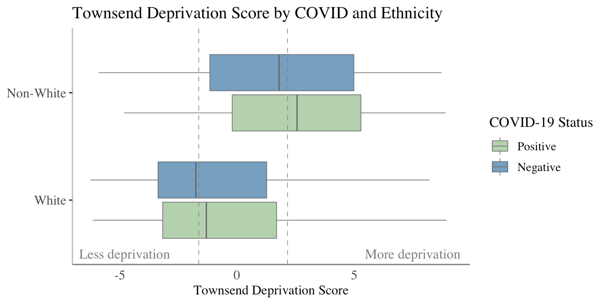
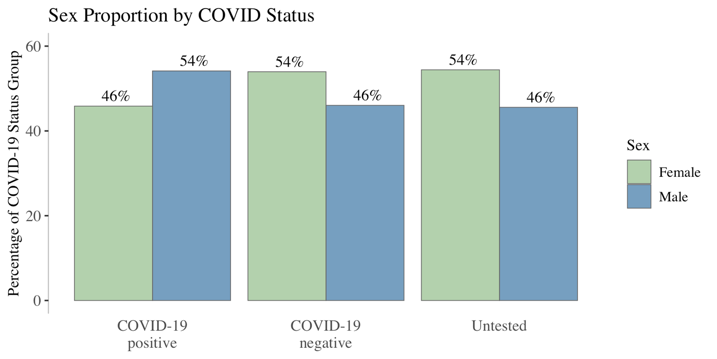
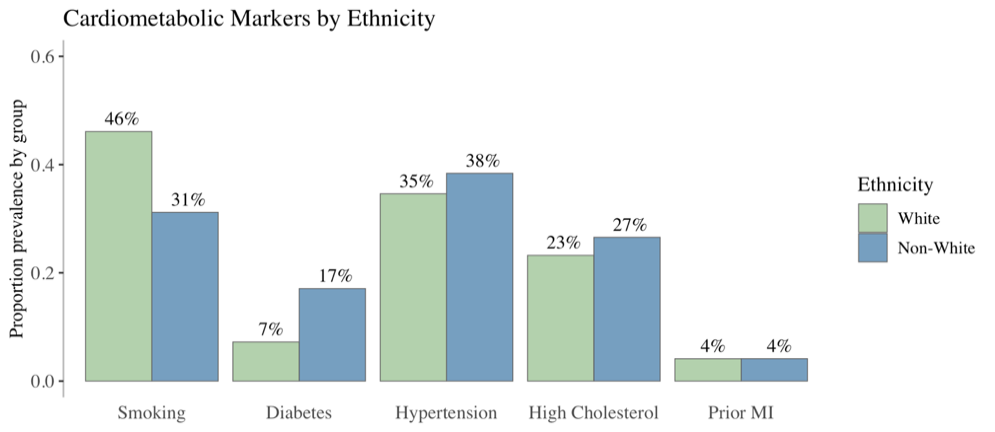
B

C

D

E

F



**Figure 1 footnote: Panel A:** Male: Female split by COVID-19 status; **Panel B:** Percentage of participants from different non-Caucasian ethnicities by COVID-19 status; **Panel C:** Townsend deprivation score by ethnicity and COVID-19 status; **Panel D:** Vitamin D levels by month of measurement stratified by sex and ethnicity; **Panel E:** Cardiometabolic profile stratified by ethnicity; **Panel F:** Cardiometabolic profile stratified by sex. COVID-19: coronavirus disease 2019; MI: myocardial infarction.

**Supplementary Table 1. Exposures considered and their definitions in the present study**

|  |  |  |
| --- | --- | --- |
| Exposure | Description | Data type |
| Sex | Sex recorded at baseline | Binary categorical |
| Age | Age calculated as of 1st April 2020 | Numerical continuous |
| Ethnicity | As recorded at baseline visit, per existing UKB categories: White, Black, Asian, Chinese, Mixed, Other | Categorical |
| Smoking | Self-report of current, previous, never- considered as: current/previous vs never | Binary categorical |
| BMI | Calculated from height and weight at baseline:  BMI = weight (Kg) /height(m)2 | Numerical continuous |
| Vitamin D | Serum levels measured at baseline visit; corrected for seasonality | Numerical continuous |
| Processed meat | Self-report of weekly intake frequency, converted to grams/day intake based on 1 portion=75g | Numerical continuous |
| Diabetes | Self-report and HES (Supplementary Table 2) | Binary categorical |
| Hypertension | Self-report and HES (Supplementary Table 2) | Binary categorical |
| High cholesterol | Self-report and HES (Supplementary Table 2) | Binary categorical |
| Prior MI | Algorithmically defined outcomes | Binary categorical |
| Townsend deprivation score | Score of relative material deprivation from baseline visit | Numerical continuous |
| Home type | Self-report of flat/apartment, sheltered accommodation, house, bungalow; considered as: communal living space vs non-communal (house/bungalow) | Binary categorical |
| Household size | Self-report of number of people in household at baseline | Numerical discrete |
| Generations in household | Self-report of relationship to people in household. | Numerical discrete |
| Family/friends visit | Self-reported answer to the question “"How often do you visit friends or family or have them visit you?" Coded as 1 if once a week or more, 0 if less than once per week. | Binary categorical |
| Regular leisure activity | Self-report of at least weekly leisure activity outside the home including sports, pub, religious group, adult education classes; considered as: regular leisure activity vs no regular leisure activity | Binary categorical |
| Risk taking | Self-report answer to “tendency to take risks” at baseline visit: Yes/No. | Binary categorical |

**Supplementary Table 1 footnote:** BMI: body mass index; HES: Hospital Episode Statistics; MI: myocardial infarction; UKB: UK Biobank

**Supplementary Table 2. International Classification of Disease (ICD) codes used to define comorbidities from Hospital Episode Statistic data**

|  |  |  |
| --- | --- | --- |
| Condition | ICD code | Code description |
| Diabetes | E100 | Type 1 diabetes mellitus: With coma |
| Diabetes | E101 | Type 1 diabetes mellitus: With ketoacidosis |
| Diabetes | E102 | Type 1 diabetes mellitus: With renal complications |
| Diabetes | E103 | Type 1 diabetes mellitus: With ophthalmic complications |
| Diabetes | E104 | Type 1 diabetes mellitus: With neurological complications |
| Diabetes | E105 | Type 1 diabetes mellitus: With peripheral circulatory complications |
| Diabetes | E106 | Type 1 diabetes mellitus: With other specified complications |
| Diabetes | E107 | Type 1 diabetes mellitus: With multiple complications |
| Diabetes | E108 | Type 1 diabetes mellitus: With unspecified complications |
| Diabetes | E109 | Type 1 diabetes mellitus: Without complications |
| Diabetes | E110 | Type 2 diabetes mellitus: With coma |
| Diabetes | E111 | Type 2 diabetes mellitus: With ketoacidosis |
| Diabetes | E112 | Type 2 diabetes mellitus: With renal complications |
| Diabetes | E113 | Type 2 diabetes mellitus: With ophthalmic complications |
| Diabetes | E114 | Type 2 diabetes mellitus: With neurological complications |
| Diabetes | E115 | Type 2 diabetes mellitus: With peripheral circulatory complications |
| Diabetes | E116 | Type 2 diabetes mellitus: With other specified complications |
| Diabetes | E117 | Type 2 diabetes mellitus: With multiple complications |
| Diabetes | E118 | Type 2 diabetes mellitus: With unspecified complications |
| Diabetes | E119 | Type 2 diabetes mellitus: Without complications |
| Diabetes | E130 | Other specified diabetes mellitus: With coma |
| Diabetes | E131 | Other specified diabetes mellitus: With ketoacidosis |
| Diabetes | E132 | Other specified diabetes mellitus: With renal complications |
| Diabetes | E133 | Other specified diabetes mellitus: With ophthalmic complications |
| Diabetes | E134 | Other specified diabetes mellitus: With neurological complications |
| Diabetes | E135 | Other specified diabetes mellitus: With peripheral circulatory complications |
| Diabetes | E136 | Other specified diabetes mellitus: With other specified complications |
| Diabetes | E137 | Other specified diabetes mellitus: With multiple complications |
| Diabetes | E138 | Other specified diabetes mellitus: With unspecified complications |
| Diabetes | E139 | Other specified diabetes mellitus: Without complications |
| Diabetes | E140 | Unspecified diabetes mellitus: With coma |
| Diabetes | E141 | Unspecified diabetes mellitus: With ketoacidosis |
| Diabetes | E142 | Unspecified diabetes mellitus: With renal complications |
| Diabetes | E143 | Unspecified diabetes mellitus: With ophthalmic complications |
| Diabetes | E144 | Unspecified diabetes mellitus: With neurological complications |
| Diabetes | E145 | Unspecified diabetes mellitus: With peripheral circulatory complications |
| Diabetes | E146 | Unspecified diabetes mellitus: With other specified complications |
| Diabetes | E147 | Unspecified diabetes mellitus: With multiple complications |
| Diabetes | E148 | Unspecified diabetes mellitus: With unspecified complications |
| Diabetes | E149 | Unspecified diabetes mellitus: Without complications |
| Diabetes | G590 | Diabetic mononeuropathy |
| Diabetes | G632 | Diabetic polyneuropathy |
| Diabetes | H280 | Diabetic cataract |
| Diabetes | H360 | Diabetic retinopathy |
| Diabetes | M142 | Diabetic arthropathy |
| Diabetes | N083 | Glomerular disorders in diabetes mellitus |
| Diabetes | O240 | Diabetes mellitus in pregnancy: Pre-existing type 1 diabetes mellitus |
| Diabetes | O241 | Diabetes mellitus in pregnancy: Pre-existing type 2 diabetes mellitus |
| Diabetes | O243 | Diabetes mellitus in pregnancy: Pre-existing diabetes mellitus, unspecified |
| Diabetes | O244 | Diabetes mellitus arising in pregnancy |
| Diabetes | O249 | Diabetes mellitus in pregnancy, unspecified |
| Diabetes | Y423 | Insulin and oral hypoglycaemic [antidiabetic] drugs |
| Hypertension | I10X | Essential (primary) hypertension |
| Hypertension | I110 | Hypertensive heart disease with (congestive) heart failure |
| Hypertension | I119 | Hypertensive heart disease without (congestive) heart failure |
| Hypertension | I120 | Hypertensive renal disease with renal failure |
| Hypertension | I129 | Hypertensive renal disease without renal failure |
| Hypertension | I130 | Hypertensive heart and renal disease with (congestive) heart failure |
| Hypertension | I131 | Hypertensive heart and renal disease with renal failure |
| Hypertension | I132 | Hypertensive heart and renal disease with both (congestive) heart failure and renal failure |
| Hypertension | I139 | Hypertensive heart and renal disease, unspecified |
| High cholesterol | E780 | Pure hypercholesterolaemia |
| High cholesterol | E782 | Mixed hyperlipidaemia |
| High cholesterol | E783 | Hyperchylomicronaemia |
| High cholesterol | E784 | Other hyperlipidaemia |
| High cholesterol | E785 | Hyperlipidaemia, unspecified |

**Supplementary Table 3. Baseline demographics by COVID-19 status**

|  |  |  |  |
| --- | --- | --- | --- |
|  | Test positive (*n*=1,326) | Test negative (*n*=3,184) | Untested (*n*=497,996) |
| Men | 696 (52.5%) | 1,505 (47.3%) | 226,921 (45.6%) |
| Age | 68.11 (± 9.23) | 68.91 (± 8.72) | 68.25 (± 8.10) |
| White ethnicity | 1,141 (86.0%) | 2,927 (91.9%) | 468,629 (94.1%) |
| Non-White ethnicity | 174 (13.1%) | 241 (7.6%) | 26,618 (5.3%) |
| Black ethnicity | 76 (5.7%) | 91 (2.9%) | 7,894 (1.6%) |
| Asian ethnicity | 60 (4.5%) | 78 (2.4%) | 9,744 (2.0%) |
| Chinese ethnicity | 6 (0.5%) | 3 (0.1%) | 1,565 (0.3%) |
| Mixed ethnicity | 9 (0.7%) | 24 (0.8%) | 2,925 (0.6%) |
| Other ethnicity\* | 34 (2.6%) | 61 (1.9%) | 7,239 (1.5%) |
| Smoking (current or previous) | 683 (51.5%) | 1,653 (51.9%) | 225,902 (45.4%) |
| Processed meat intake (g/day) | 17.08 (± 15.67) | 16.33 (± 15.00) | 15.91 (± 14.94) |
| BMI (kg/m2) | 28.04 [± 6.47] | 27.41 [± 6.37] | 26.74 [± 5.77] |
| Diabetes | 217 (16.4%) | 449 (14.1%) | 38,472 (7.7%) |
| Hypertension | 624 (47.1%) | 1,457 (45.8%) | 172,913 (34.7%) |
| High cholesterol | 437 (33.0%) | 1,034 (32.5%) | 116,225 (23.3%) |
| Prior myocardial infarction | 96 (7.2%) | 242 (7.6%) | 20,477 (4.1%) |
| Vitamin D (nmol/L)\*\* | 33.88 [± 27.01] | 35.45 [± 26.78] | 37.55 [± 26.49] |
| Townsend deprivation score | -0.91 [± 5.34] | -1.55 [± 5.00] | -2.14 [± 4.19] |
| Home type (flat/apartment) | 191 (14.4%) | 455 (14.3%) | 51,087 (10.3%) |
| Household size | 2.50 (± 1.31) | 2.32 (± 1.22) | 2.39 (± 1.15) |
| Number of generations in household | 1.41 (± 0.52) | 1.35 (± 0.50) | 1.37 (± 0.50) |
| Family/friend visits | 975 (73.5%) | 2,438 (76.6%) | 384,280 (77.2%) |
| Regular leisure activity | 897 (67.6%) | 2,124 (66.7%) | 344,518 (69.2%) |
| Tendency to take risks | 404 (30.5%) | 916 (28.8%) | 127,913 (25.7%) |

**Supplementary Table 3 footnote:** Results are number (percentage) for categorical and mean (standard deviation) or median [interquartile range] for continuous variables. \*\*Ethnicity was missing for <1% of participants across all categories; they are displayed as part of “other ethnicity” in this table but have been excluded from subsequent modelling. \*Vitamin D has been adjusted for seasonality.

**Supplementary Table 4. Baseline characteristics stratified by sex and COVID-19 status**

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
|  | Test positive (*n*=1,326) | | Test negative (*n*=3,184) | | Untested (*n*=497,996) | | |
|  | Men (n=696) | Women (n=630) | Men (n=1,505) | Women (n=1,679) | Men (n=226,921) | Women (n=271,075) |
| Age | 69.65 (± 8.83) | 66.41 (± 9.36) | 70.24 (± 8.30) | 67.72 (± 8.92) | 68.46 (± 8.20) | 68.08 (± 8.01) |
| White ethnicity | 600 (86.2%) | 541 (85.9%) | 1,397 (92.8%) | 1,530 (91.1%) | 213,262 (94.0%) | 255,367 (94.2%) |
| Non-White ethnicity | 89 (12.8%) | 85 (13.5%) | 99 (6.6%) | 142 (8.5%) | 12,164 (5.4%) | 14,454 (5.3%) |
| Black ethnicity | 38 (5.5%) | 38 (6.0%) | 28 (1.9%) | 63 (3.8%) | 3,341 (1.5%) | 4,553 (1.7%) |
| Asian ethnicity | 32 (4.6%) | 28 (4.4%) | 46 (3.1%) | 32 (1.9%) | 5,216 (2.3%) | 4,528 (1.7%) |
| Chinese ethnicity | 1 (0.1%) | 5 (0.8%) | 0 (0.0%) | 3 (0.2%) | 583 (0.3%) | 982 (0.4%) |
| Mixed ethnicity | 5 (0.7%) | 4 (0.6%) | 7 (0.5%) | 17 (1.0%) | 1,093 (0.5%) | 1,832 (0.7%) |
| Other ethnicity\* | 20 (2.9%) | 14 (2.2%) | 27 (1.8%) | 34 (2.0%) | 3,426 (1.5%) | 3,813 (1.4%) |
| Smoking (current, previous) | 422 (60.6%) | 261 (41.4%) | 887 (58.9%) | 766 (45.6%) | 115,977 (51.1%) | 109,925 (40.6%) |
| Processed meat intake (g/day) | 20.43 (± 16.46) | 13.38 (± 13.86) | 20.74 (± 16.21) | 12.39 (± 12.60) | 20.24 (± 16.32) | 12.29 (± 12.58) |
| BMI (kg/m2) | 28.31 [± 5.55] | 27.61 [± 7.43] | 27.77 [± 5.60] | 27.04 [± 7.12] | 27.30 [± 5.08] | 26.12 [± 6.27] |
| Diabetes | 140 (20.1%) | 77 (12.2%) | 264 (17.5%) | 185 (11.0%) | 22,829 (10.1%) | 15,643 (5.8%) |
| Hypertension | 380 (54.6%) | 244 (38.7%) | 824 (54.8%) | 633 (37.7%) | 90,879 (40.0%) | 82,034 (30.3%) |
| High cholesterol | 293 (42.1%) | 144 (22.9%) | 621 (41.3%) | 413 (24.6%) | 68,010 (30.0%) | 48,215 (17.8%) |
| Prior MI | 76 (10.9%) | 20 (3.2%) | 182 (12.1%) | 60 (3.6%) | 15,521 (6.8%) | 4,956 (1.8%) |
| Vitamin D\*\* | 34.80 [± 28.32] | 33.07 [± 25.63] | 36.19 [± 26.80] | 34.58 [± 26.54] | 37.62 [± 25.81] | 37.49 [± 27.10] |
| Townsend deprivation score | -0.98 [± 5.51] | -0.90 [± 5.15] | -1.64 [± 5.12] | -1.41 [± 4.84] | -2.13 [± 4.27] | -2.15 [± 4.11] |
| House (Flat/Apartment) | 119 (17.1%) | 72 (11.4%) | 244 (16.2%) | 211 (12.6%) | 24,842 (10.9%) | 26,245 (9.7%) |
| Household size | 2.46 (± 1.34) | 2.54 (± 1.28) | 2.30 (± 1.17) | 2.34 (± 1.26) | 2.45 (± 1.17) | 2.34 (± 1.13) |
| Generations in household | 1.36 (± 0.51) | 1.47 (± 0.53) | 1.31 (± 0.48) | 1.39 (± 0.51) | 1.36 (± 0.50) | 1.37 (± 0.51) |
| Family/friend visits | 483 (69.4%) | 492 (78.1%) | 1,092 (72.6%) | 1,346 (80.2%) | 164,568 (72.5%) | 219,712 (81.1%) |
| Leisure activity | 485 (69.7%) | 412 (65.4%) | 1,006 (66.8%) | 1,118 (66.6%) | 157,461 (69.4%) | 187,057 (69.0%) |
| Tendency to take risks | 245 (35.2%) | 159 (25.2%) | 547 (36.3%) | 369 (22.0%) | 75,866 (33.4%) | 52,047 (19.2%) |

**Supplementary Table 4 footnote:** BMI: body mass index; COVID-19: coronavirus disease 2019; MI: myocardial infarction\*\*Ethnicity was missing for <1% of participants across all categories; they are displayed as part of “other ethnicity” in this table but have been excluded from subsequent modelling. \*Vitamin D has been adjusted for seasonality.

**Supplementary Table 5. Baseline characteristics stratified by ethnicity and COVID-19 status**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  | Test positive (*n*=1,326) | | Test negative (*n*=3,184) | | Untested (*n*=497,996) | |
|  | White (n=1,141) | Non-White (n=174) | White (n=2,927) | Non-White (n=241) | White (n=468,629) | Non-White (n=26,618) |
| Men | 600 (52.6%) | 89 (51.1%) | 1,397 (47.7%) | 99 (41.1%) | 213,262 (45.5%) | 12,164 (45.7%) |
| Age | 68.75 (± 9.21) | 63.80 (± 8.16) | 69.27 (± 8.62) | 64.45 (± 8.86) | 68.50 (± 8.03) | 63.95 (± 8.18) |
| Black ethnicity |  | 76 (43.7%) |  | 91 (37.8%) |  | 7,894 (29.7%) |
| Asian ethnicity |  | 60 (34.5%) |  | 78 (32.4%) |  | 9,744 (36.6%) |
| Chinese ethnicity |  | 6 (3.4%) |  | 3 (1.2%) |  | 1,565 (5.9%) |
| Mixed ethnicity |  | 9 (5.2%) |  | 24 (10.0%) |  | 2,925 (11.0%) |
| Other ethnicity |  | 23 (13.2%) |  | 45 (18.7%) |  | 4,490 (16.9%) |
| Smoking (current, previous) | 621 (54.4%) | 56 (32.2%) | 1,566 (53.5%) | 74 (30.7%) | 215,755 (46.0%) | 8,298 (31.2%) |
| Processed meat intake (g/day) | 17.56 (± 15.47) | 14.04 (± 16.79) | 16.67 (± 14.97) | 12.35 (± 14.91) | 16.11 (± 14.91) | 12.38 (± 15.00) |
| BMI (kg/m2) | 27.88 [± 6.33] | 29.03 [± 7.99] | 27.38 [± 6.33] | 27.85 [± 7.11] | 26.71 [± 5.74] | 27.08 [± 6.01] |
| Diabetes | 176 (15.4%) | 37 (21.3%) | 380 (13.0%) | 66 (27.4%) | 33,587 (7.2%) | 4,518 (17.0%) |
| Hypertension | 523 (45.8%) | 94 (54.0%) | 1,331 (45.5%) | 116 (48.1%) | 161,763 (34.5%) | 10,170 (38.2%) |
| High cholesterol | 377 (33.0%) | 56 (32.2%) | 947 (32.4%) | 82 (34.0%) | 108,513 (23.2%) | 7,044 (26.5%) |
| Prior MI | 83 (7.3%) | 11 (6.3%) | 226 (7.7%) | 13 (5.4%) | 19,217 (4.1%) | 1,095 (4.1%) |
| Vitamin D | 35.60 [± 26.88] | 22.53 [± 18.70] | 36.29 [± 27.19] | 25.77 [± 20.58] | 38.28 [± 26.35] | 25.18 [± 21.14] |
| Townsend deprivation score | -1.31 [± 4.86] | 2.56 [± 5.49] | -1.76 [± 4.64] | 1.79 [± 6.15] | -2.26 [± 3.96] | 1.03 [± 5.61] |
| House (Flat/Apartment) | 140 (12.3%) | 49 (28.2%) | 379 (12.9%) | 73 (30.3%) | 44,241 (9.4%) | 6,505 (24.4%) |
| Household size | 2.39 (± 1.24) | 3.20 (± 1.57) | 2.26 (± 1.16) | 3.07 (± 1.66) | 2.35 (± 1.10) | 3.08 (± 1.63) |
| Generations in household | 1.37 (± 0.51) | 1.68 (± 0.54) | 1.33 (± 0.48) | 1.67 (± 0.58) | 1.35 (± 0.50) | 1.62 (± 0.57) |
| Family/friend visits | 868 (76.1%) | 101 (58.0%) | 2,276 (77.8%) | 156 (64.7%) | 366,439 (78.2%) | 16,684 (62.7%) |
| Leisure activity | 772 (67.7%) | 123 (70.7%) | 1,950 (66.6%) | 168 (69.7%) | 325,635 (69.5%) | 17,761 (66.7%) |
| Tendency to take risks | 334 (29.3%) | 69 (39.7%) | 822 (28.1%) | 91 (37.8%) | 117,718 (25.1%) | 9,635 (36.2%) |

**Supplementary Table 5 footnote:** BMI: body mass index; COVID-19: coronavirus disease 2019; MI: myocardial infarction

**Supplementary Table 6. Univariate logistic regression models exposures associations with COVID-19 status in whole cohort, men, and women within the tested sample (n=4,510)**

|  |  |  |  |
| --- | --- | --- | --- |
| **Exposures** | **Whole sample** | **Men** | **Women** |
|  | Test positive, *n*=1,326  Test negative, *n*=3,184 | Test positive, *n*=696  Test negative, *n*=1,505 | Test positive, *n*=630  Test negative, *n*=1,679 |
| Sex (Male) | 1.23\* [1.08, 1.40] | – | – |
|  | 0.0014 | – | – |
| Age | 0.99\* [0.98, 1.00] | 0.99 [0.98, 1.00] | 0.98\* [0.97, 0.99] |
|  | 0.0059 | 0.1290 | 0.0020 |
| Ethnicity (Non-white) | 1.85\* [1.51, 2.28] | 2.09\* [1.55, 2.83] | 1.69\* [1.27, 2.25] |
|  | 5.0310-9 | 1.6210-06 | 3.1110-04 |
| Townsend deprivation score | 1.04\* [1.02, 1.06] | 1.04\* [1.02, 1.07] | 1.05\* [1.02, 1.07] |
|  | 6.9210-6 | 0.0015 | 0.0017 |
| Home Type (flat/apartment) | 1.01 [0.84, 1.21] | 1.07 [0.84, 1.36] | 0.892 [0.667, 1.180] |
|  | 0.9439 | 0.5936 | 0.4294 |
| Household Size | 1.12\* [1.06, 1.17] | 1.11\* [1.03, 1.20] | 1.12\* [1.05, 1.21] |
|  | 1.8010-5 | 0.0040 | 0.0011 |
| Generations in household | 1.26\* [1.11, 1.43] | 1.21\* [1.01, 1.45] | 1.35\* [1.14, 1.61] |
|  | 3.3210-4 | 0.0374 | 7.4610-04 |
| Family/friend visits | 0.84\* [0.72, 0.98] | 0.85 [0.70, 1.04] | 0.87 [0.69, 1.11] |
|  | 0.0264 | 0.1184 | 0.2584 |
| Socialisation habits | 1.04 [0.91, 1.19] | 1.14 [0.94, 1.39] | 0.94 [0.77, 1.14] |
|  | 0.5848 | 0.1864 | 0.5269 |
| Processed meat intake | 1.38 [0.90, 2.09] | 0.89 [0.51, 1.55] | 1.78 [0.88, 3.53] |
|  | 0.1354 | 0.6767 | 0.1036 |
| Diabetes | 1.19 [1.00, 1.42] | 1.18 [0.94, 1.49] | 1.12 [0.84, 1.49] |
|  | 0.0512 | 0.1473 | 0.4168 |
| Hypertension | 1.05 [0.93, 1.20] | 0.99 [0.83, 1.19] | 1.05 [0.87, 1.26] |
|  | 0.4254 | 0.9465 | 0.6499 |
| High Cholesterol | 1.02 [0.89, 1.17] | 1.04 [0.86, 1.24] | 0.91 [0.73, 1.13] |
|  | 0.7534 | 0.7116 | 0.3839 |
| Body mass index (kg/m2) | 1.02\* [1.01, 1.04] | 1.03\* [1.01, 1.05] | 1.02\* [1.00, 1.03] |
|  | 8.0710-05 | 0.0010 | 0.0221 |
| Smoking (current/previous) | 0.98 [0.87, 1.12] | 1.07 [0.89, 1.29] | 0.84 [0.70, 1.01] |
|  | 0.8029 | 0.4513 | 0.0710 |
| Prior myocardial infarction | 0.95 [0.74, 1.21] | 0.89 [0.67, 1.18] | 0.89 [0.52, 1.45] |
|  | 0.6752 | 0.4263 | 0.6408 |
| Vitamin D | 1.00 [0.99, 1.00] | 1.00 [0.99, 1.00] | 1.00 [0.99, 1.00] |
|  | 0.6386 | 0.6256 | 0.7117 |
| Risk Taking | 1.09 [0.94, 1.25] | 0.95 [0.79, 1.15] | 1.20 [0.97, 1.48] |
|  | 0.2534 | 0.6030 | 0.0968 |

**Supplementary Table 6 footnote:** COVID-19: coronavirus disease 2019

**Supplementary Table 7. Multivariate logistic regression models testing the role of socialisation habits (Model A), and attitude to risk (Model B) in determining risk of COVID-19**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | Exposures | Whole tested sample  *n=*4,510 | Men  *n=*2,201 | Women  *n=*2,309 |
| *Model A: age, sex, ethnicity, socialisation habits* | Male sex | 1.27\* [1.11, 1.46] | – | – |
|  | 4.7810-4 | – | – |
| Age | 0.99\* [0.98, 1.00] | 1.00 [0.99, 1.01] | 0.99\* [0.98, 1.00] |
|  | 0.0129 | 0.4313 | 0.0099 |
| Non-White ethnicity | 1.77\* [1.43, 2.20] | 2.12\* [1.54, 2.91] | 1.53\* [1.13, 2.06] |
|  | 2.0310-7 | 3.3110-6 | 0.0050 |
| Family/friend visits | 0.91 [0.78, 1.06] | 0.88 [0.71, 1.08] | 0.95 [0.75, 1.21] |
|  | 0.2340 | 0.2073 | 0.6886 |
| Regular leisure activity | 1.05 [0.91, 1.21] | 1.15 [0.94, 1.41] | 0.95 [0.78, 1.16] |
|  | 0.5104 | 0.1633 | 0.6149 |
| *Model B: age, sex, ethnicity, risk taking* | Male sex | 1.27\* [1.11, 1.45] | – | – |
|  | 4.0710-4 | – | – |
| Age | 0.99\* [0.98, 1.00] | 1.00 [0.99, 1.01] | 0.99\* [0.98, 1.00] |
|  | 0.0191 | 0.5423 | 0.0114 |
| Non-White ethnicity | 1.79\* [1.45, 2.21] | 2.06\* [1.51, 2.82] | 1.60\* [1.19, 2.13] |
|  | 5.9910-8 | 4.7110-6 | 0.0016 |
| Risk taking | 1.02 [0.88, 1.17] | 0.93 [0.77, 1.12] | 1.14 [0.92, 1.42] |
|  | 0.8377 | 0.4340 | 0.2221 |

**Supplementary Table 7 footnote:** Results are odds ratios, 95% confidence interval, and p-values for each exposure from two separate models (A, B). Exposures are mutually adjusted.