Ethnic inequalities in COVID-19 infection, hospitalisation, intensive care admission, and death: a global systematic review and meta-analysis of over 200 million study participants

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Summary

Background COVID-19 has exacerbated existing ethnic inequalities in health. Little is known about whether inequalities in severe disease and deaths, observed globally among minoritised ethnic groups, relates to greater infection risk, poorer prognosis, or both. We analysed global data on COVID-19 clinical outcomes examining inequalities between people from minoritised ethnic groups compared to the ethnic majority group.

Methods Databases (MEDLINE, EMBASE, EMCARE, CINAHL, Cochrane Library) were searched from 1st December 2019 to 3rd October 2022, for studies reporting original clinical data for COVID-19 outcomes disaggregated by ethnicity: infection, hospitalisation, intensive care unit (ICU) admission, and mortality. We assessed inequalities in incidence and prognosis using random-effects meta-analyses, with Grading of Recommendations Assessment, Development, and Evaluation (GRADE) use to assess certainty of findings. Meta-regressions explored the impact of region and time-frame (vaccine roll-out) on heterogeneity. PROSPERO: CRD42021284981.

Findings 77 studies comprising over 200,000,000 participants were included. Compared with White majority populations, we observed an increased risk of testing positive for infection for people from Black (adjusted Risk Ratio [aRR]:1.78, 95% CI:1.59–1.99, I^2 = 99.1), South Asian (aRR:3.00, 95% CI:1.59–5.66, I^2 = 99.1), Mixed (aRR:1.64, 95% CI:1.02–1.67, I^2 = 93.2) and Other ethnic groups (aRR:1.36, 95% CI:1.01–1.82, I^2 = 85.6). Black, Hispanic, and South Asian people were more likely to be seropositive. Among population-based studies, Black and Hispanic ethnic groups and Indigenous peoples had an increased risk of hospitalisation; Black, Hispanic, South Asian, East Asian and Mixed ethnic groups and Indigenous peoples had an increased risk of ICU admission. Mortality risk was increased for Hispanic, Mixed, and Indigenous groups. Smaller differences were seen for prognosis following infection. Following hospitalisation, South Asian, East Asian, Black and Mixed ethnic groups had an increased risk of ICU admission, and mortality risk was greater in Mixed ethnic groups. Certainty of evidence ranged from very low to moderate.

Interpretation Our study suggests that systematic ethnic inequalities in COVID-19 health outcomes exist, with large differences in exposure risk and some differences in prognosis following hospitalisation. Response and recovery interventions must focus on tackling drivers of ethnic inequalities which increase exposure risk and vulnerabilities to severe disease, including structural racism and racial discrimination.

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Research in context

Evidence before this study

We searched PROSPERO for existing systematic reviews with 'ethnic' or 'race' in the title, from 1st December 2019 to 30th August 2022. Previous systematic reviews, conducted early in the pandemic, synthesised emerging evidence primarily from the United Kingdom (UK) and United States of America (USA), identifying an increased risk of infection, severe disease, and death amongst minoritised ethnic groups. However, it is not known whether ethnic inequalities in severe disease and deaths, seen in many countries, relates to greater infection risk, poorer prognosis, or both. Broad ethnic groups described in previous meta-analyses may have also obscured heterogeneity in risks of infection, severe disease and death between different minoritised ethnic groups.

Added value of this study

We searched MEDLINE, EMBASE, EMCARE, CINAHL, and the Cochrane Library from 1st December 2019 to 3rd October 2022. We assessed inequalities in both incidence and prognosis by separating population-based studies from studies including only COVID-19 cases. Over 200 million participants were included from 77 studies. Compared with White majority groups, we identified an increased risk of testing positive for infection for Black, South Asian, Mixed, and Other ethnic groups; an increased risk of infection for Hispanic people was observed only in seroprevalence studies. In population-based studies, Black and Hispanic ethnic groups and Indigenous peoples had an increased risk of hospitalisation; most minoritised ethnic groups and Indigenous peoples had an increased risk of ICU admission; and Hispanic, Mixed, and Indigenous groups had an increased risk of mortality. Following hospitalisation, South Asian, East Asian, Black and Mixed ethnic groups had an increased risk of ICU admission, and the risk of mortality was greater in Mixed ethnic groups.

Implications of all the available evidence

We present the most comprehensive summary of ethnic inequalities in a range of outcomes relating to COVID-19, during the first few years of the pandemic, before widespread immunity. We demonstrate the presence of systematic ethnic inequalities relating to both COVID-19 infection and severe disease, but to varying extents across minoritised ethnic groups. In particular, we observed large differences in infection risk for minoritised ethnic groups. Our findings highlight the need for policy interventions to address ethnic inequalities in exposure to the virus. Ethnic inequalities in prognosis following hospitalisation were also noted, which may reflect poorer healthcare quality for minoritised ethnic groups or differential vulnerability to severe disease. Our findings are of vital public health importance and should inform strategic response and recovery policies.

Introduction

Minoritised ethnic groups have suffered disproportionately from the COVID-19 pandemic, with higher rates of SARS-CoV-2 infection, hospitalisation, intensive care unit (ICU) admission, and death, compared to the ethnic majority group in a given population.[1–7] It is not known whether the inequalities in severe disease and deaths, seen in many countries among minoritised groups, relates to greater infection risk, poorer prognosis, or both.[8–12]

Previous systematic reviews conducted early in the pandemic have demonstrated and quantified some of these risks.[13–17] However, a new review is warranted for several reasons: first, seroprevalence studies were lacking despite providing the best estimates of infection; second, broad ethnic categorisations were analysed and may hide important heterogeneity in risks between different ethnic groups; and third, now that COVID-19 has spread across the world and remains endemic in many areas, a much larger body of literature is available to address these issues.[13–17]

We therefore sought to perform a systematic review and meta-analysis examining clinical outcomes in COVID-19, comparing people from minoritised ethnic groups to the ethnic majority group, incorporating data from lower- and middle-income countries that were not available in previous meta-analyses.

Methods

This systematic review and meta-analysis is reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines,[18] and was registered with PROSPERO (CRD420212824981). Ethical approval and informed consent of participants were not required for this meta-analysis as no new data were collected.

Search strategy

The search strategy was developed and conducted by an academic librarian (PD), using the databases MEDLINE, EMBASE, EMCARE, CINAHL, and the Cochrane Library (supplementary materials: search strategy). Peer-reviewed publications were searched for between 1st December 2019 and 3rd October 2022.

Eligibility criteria

The relevant outcomes were SARS-CoV-2 infection (laboratory confirmed infection through polymerase chain reaction [PCR] or evidence of previous infection through laboratory confirmed IgG/IgM for SARS-CoV-2 [seroprevalence]), hospitalisation, ICU admission, and mortality. Studies were eligible if they reported original clinical data (cross-sectional, case–control, cohort studies) on the relevant outcomes, disaggregated by ethnicity or closely related indicators of ethnicity (i.e., Indigenous status, race, country of birth, migrant status) (see Table S1 for detailed inclusion and exclusion criteria). Although we did not set out to make comparisons by country of birth or migrant status, these indicators were included as ethnicity data are not collected in some countries.[19] Population-based studies (individuals with and without confirmed SARS-CoV-2 infection) were eligible, as well as studies which examined prognosis (individuals with confirmed SARS-CoV-2 infection only). Ecological studies, prognostic modelling studies, animal studies, qualitative studies, and pre-prints, were excluded. Studies were also excluded if participants were recruited based on a specific physical or mental health condition or healthcare utilisation (other than for COVID-19), as were studies specifically in children (under the age of 16) and religious groups.

Study selection

Two reviewers independently screened titles, abstracts, and full texts (PI assessing all articles, with DK, HT, SVK, LB, DP and SS each assessing a proportion). Where there were disagreements in title and abstract screening, articles identified as potentially relevant by one reviewer were included for full text screening. Disagreements in full texts were resolved by discussion or consultation with the review team. The software, Covidence,[20] was used for screening following automatic de-duplication. To minimise the inclusion of duplicate data (i.e., participants from the same population assessing the same outcome), predefined criteria were used to determine which dataset to include (supplementary materials: criteria to minimise inclusion of duplicate data).

Data extraction

One reviewer (PI) completed 100% of the data extraction from each eligible article, all of which were independently checked by additional reviewers (DK, HT, DP, SS). Data on study and participant characteristics, ethnicity, outcomes, and covariates, were extracted to an Excel file. Ethnicity measures were extracted and evaluated to determine the comparison group for meta-analyses. When describing ethnic groups, we have used terminology from the included studies. Where US studies reported both race and ethnicity, we chose the categories that were most amenable to meta-analysis (i.e., comparable to other studies).

Risk of bias and conceptualisation of ethnicity

Two reviewers (PI completing 100% and DK, HT, LB, DP and SS a proportion) independently assessed the risk of bias of each included study, using an adapted Joanna Briggs Institute (JBI) tool[21] (supplementary materials: adapted JBI critical appraisal tool). Scores were calculated as a total out of the maximum number of applicable questions, then standardised. Studies with a score of 80–100% were considered low risk of bias, 60–79% medium risk of bias, and 0–59% high risk of bias. One item from the JBI, regarding the description of study participants, was adapted to critically evaluate how ethnicity was conceptualised and measured. Studies which measured ethnicity through self-report or another reliable indicator (e.g., country of birth registered at birth or migration) and used disaggregated descriptions of ethnicity scored positively on this item of the risk of bias tool. No studies were excluded based on critical appraisal scores.

Data synthesis

Meta-analysis

To determine ethnic inequalities in COVID-19 health outcomes, we prioritised extracting age- and sex-adjusted results which are important confounders of ethnic inequalities,[22] but considered other variables to be likely mediators and therefore should not be adjusted for.[8] We contacted authors to request data, if unavailable (supplementary material: data manipulation methods). Unadjusted or over-adjusted models were extracted if age- and sex-adjusted models were not available (highlighted in forest plots and reported in Table 1).

We conducted meta-analyses on age- and sex adjusted data to determine the risk of each outcome across disaggregated minoritised ethnic groups (guided by the critical appraisal) compared to the White majority. The Grading of Recommendations Assessment, Development, and Evaluation (GRADE) approach was used to evaluate the certainty of the overall evidence for adjusted analyses.[23] The overall certainty estimates were categorised as high, moderate, low, or very low. In keeping with GRADE guidance for prognostic studies, observational studies started as high certainty evidence and were rated down for risk of bias, imprecision, inconsistency, indirectness, and publication bias (supplementary materials: GRADE criteria).[23]

First author	Outcomes	Country	Period of data collection	Comorbidities considered	Covariates adjusted for	Study quality %	Majority N	Minority N
High-income countries								
Song	Hospital admission; ICU admission	USA	1st Mar–10th Aug 20	Respiratory failure, myocardial infarction, stroke, pulmonary hypertension, embolism, thrombosis, acute renal failure	N/A	50%	465,565 (White)	182,637
Acosta	Mortality	USA	1st Mar 20–28th Feb 21	NR	Age	88%	63,981 (White)	79,361
Adeji	Mortality	USA	1st Jul 21–30th Jun 22	NR	N/A	81%	207,926 (White)	80,218
Lindsay	Seropositivity	USA	20th Feb–10th Jul 20	Charlson Comorbidity Index	N/A	75%	1,092,652 (White)	269,941
Luo	Mortality	USA	4th Mar–23rd Jun 20	Cardiovascular disease, obesity, diabetes	N/A	25%	7,300,959 (White)	3,321,251
Metra	ICU admission	USA	20th Jan–30th Sep 20	Hypertension, diabetes, obesity, COPD, cerebral infarction, systemic connective tissue disorders, reduced mobility, pregnancy, neoplasm, nicotine dependence	Age, sex, diabetes, hypertension, obesity, homelessness, neoplasms	50%	157,049 (White)	50,376
Zerbo	Hospital admission; ICU admission; Mortality	USA	1st Nov 20–27th Jul 21	BMI, diabetes, hypertension, renal disease, asthma, heart disease, COPD, pneumonia, cerebral infarction, Alzheimers, Parkinsons, movement disorders, demyelinating disorders, epilepsy	Age, comorbidities	75%	1,891,899 (White)	1,300,923
Bennett	Hospital admission; Mortality	USA	1st Jan 20–7th Dec 20	Diabetes, renal disease, heart failure, chronic pulmonary disease, peripheral vascular disease, stroke, cancer, dementia, myocardial infarction, liver disease, rheumatologic disease, hemiplegia, peptic ulcer disase	N/A	69%	1,251,401 (White)	675,298
Chang	Infection; Hospital admission	USA	1st Jan 20–31st Dec 20	27 conditions	N/A	88%	23,769,184 (White)	7,859,910
Egede	ICU admission	USA	1st Mar 20–10th Jul 20	Comorbidity count	N/A	81%	23,788 (White)	7761
Feldman	Mortality	USA	1st Jan 20–24th Feb 21	NR	Age	69%	227,532 (White)	148,593
Ioannou	Infection	USA	1st Feb 20–31st Mar 21	Charlson Comorbidity Index	Age, sex, region, location, BMI, comorbidities	93%	5,887,349 (White)	3,240,324
Jones	Seroprevalence	USA	1st Jul 20–31st May 21	NR	N/A	63%	1,226,745 (White)	216,774
Young	Hospital admission	USA	1st Jan 20–31st Dec 20	Presence of comorbidities	Age, sex, service, rank, education, occupation, region, comorbidities	93%	374,525 (White	622,726
Thomas 1	ICU admission; Mortality	UK	1st Mar 20–31st May 20	NR	N/A	81%	13,092 (White)	697
Gray	Mortality	UK	1st Mar 20–31st Mar 21	Charlson Comorbidity Index	Age, sex, deprivation, comorbidities	93%	359,160 (White)	15,084
Knight	Hospital admission	UK	1st Jan 20–7th Dec 20	Number of diagnoses	N/A	93%	36,131,134 (White)	8,833,352
Thomas 2	Infection	UK	21st Nov 20–22nd Dec 20	NR	Age, deprivation, catchment area, smoking status, place of work, key worker	88%	2729 (White British or Irish)	88

(Table 1 continues on next page)

First author	Outcomes	Country	Period of data collection	Comorbidities considered	Covariates adjusted for	Study quality %	Majority N	Minority N
(Continued from previous page)								
Martin	Infection	UK	Dec 20–Mar 21	Diabetes, immunosuppressed	Age, sex, migration, religiosity, deprivation, household factors	93%	7583 (White)	8276
Talaei	Seroprevalence	UK	1st May 20–2nd Nov 20	Arterial disease, asthma, autoimmune disease, cancer, COPD, diabetes, heart disease, hypertension, immunodeficiency, kidney disease, major neurological conditions	Age, sex	93%	10,651 (White)	479
Mathur	Infection; Hospital admission; ICU admission; Mortality	UK	1st Feb 20 to 31st Dec 20	Hypertension, asthma, respiratory disease, heart disease, diabetes, cancer, liver disease, stroke, dementia, neurological disease, kidney disease, immunosuppression	Age, sex	100%	10,877,978 (White)	6,410,554
Hippisley Cox	Hospital admission; Mortality	UK	8th Dec 20–15th Jun 21	Kidney disease, chemotherapy, diabetes, other health conditions	Age, sex, BMI, vaccination dose, background infection rate	75%	4,781,050 (White)	903,979
Ward	Seropositivity	UK	20th Jun 20–13th July 20	NR	Age, sex, region	94%	92,737 (White)	6667
Farrell	ICU admission; Mortality	Ireland	13th Mar 20–1st May 20	Charlson Comorbidity Index	Age	69%	208 (White Irish and White Other)	49
Allen	Seroprevalence	Ireland	19th Apr 21–28th Apr 21	NR	N/A	81%	3798 (White Irish)	1287
Chu	Hospital admission	Canada	1st Jen 20–30th Sep 20	Charlson Comorbidity Index	N/A	75%	42,547 (General)	4645
Saeed	Seropositivity	Canada	9th May 20–21st Jul 20	NR	Age, sex, region, deprivation, blood type	81%	52,852 (White)	10,695
Passos-Castilho	Hospital admission	Canada	1st Jan 20–30th Sep 20	Charlson Comorbidity Index	N/A	88%	42,547 (Canadian born)	4645
Islamoska	Hospital admission	Denmark	1st Feb 20–30th Jun 20	Charlson Comorbidity Index	Age, sex	88%	433,539 (Danish)	60,114
Guijarro	Infection	Spain	1st Jan 20–14th Mar 20	NR	Age, sex	100%	131,599 (Spain)	20,419
Ramos-Rincon	ICU admission; Mortality	Spain	1st Mar 20–31st Dec 21	Charlson Comorbidity Index	N/A	81%	20,599 (European)	3354
Rostila	Mortality	Sweden	31st Jan 20–4th May 20	NR	Age, sex	94%	11,232,511 (Sweden)	541,119
Nwaru	Hospital admission; ICU admission	Sweden	1st Jan 20–28th Feb 21	Hypertension, diabetes, obesity, stroke, pneumonia, COPD, asthma, psychiatric conditions	N/A	88%	266,848 (Swedish born)	59,204
Stralin	ICU admission; Mortality	Sweden	1st Mar 20–30th Sep 20	Hypertension, diabetes, chronic lung disease, cancer, ischaemic disease, neuromuscular disease, stroke, obesity, dementia	Age, sex, comorbidities, care dependency, healthcare region	88%	9973 (Sweden)	6243
Gustafsson	Hospital admission; Mortality	Sweden	4th Apr 20–14th Sep 20	Charlson Comorbidity Index	Age, sex, comorbidities, housing, family structure, civil status, region, education, income	100%	52,201 (Sweden)	20,527
Consolazio	Infection	Italy	20th Feb 20–3rd May 20	NR	Age, sex, comorbidities	88%	2,856,202 (Italy)	469,473
Lombardi	Seropositivity	Italy	27th Apr 20–12th Jun 20	Hypertension, diabetes, cardiac, respiratory, renal chronic diseases	Age, sex, occupation, frontline area, BMI, smoking	81%	3869 (Italian)	186

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First author	Outcomes	Country	Period of data collection	Comorbidities considered	Covariates adjusted for	Study quality %	Majority N	Minority N
(Continued from previous page)								
Fabiani	Hospital admission; ICU admission; Mortality	Italy	20th Feb 20–19th Jul 20	Oncologic, cardiovascular, respiratory, diabetes/ metabolic diseases	Age, sex, geographical macro-area of diagnosis, level of urbanisation of place of residence, comorbidities, calendar period of diagnosis, random effect due to regional contextual differences	88%	197,206 (Italian)	15,974
Cacciani	Hospital admission; ICU admission	Italy	22nd Feb 21–2nd Jul 21	NR	N/A	88%	45,580 (Italian)	2513
DiGirolamo	Mortality	Italy	22nd Feb 21–16th Jul 21	NR	N/A	81%	34,370,041 (Italian)	4,006,808
Pagani	Seroprevalence	Italy	23rd Dec 20–19th Feb 21	NR	N/A	88%	1572 (Italian)	472
Coyer 1	Hospital admission	Netherlands	29th Feb 20–31st May 20	NR	Age	88%	386,521 (Ethnic Dutch)	486,534
Collard	Mortality	Netherlands	18th Feb 20–30th Jan 21	Hypertension, asthma/ COPD, chronic kidney disease, diabetes, malignancy, chronic cardiac disase, obesity, BMI	N/A	69%	763 (Dutch)	415
Coyer 2	Seropositivity	Netherlands	24th Jun 20–9th Oct 20	Diabetes, high blood pressure	N/A	88%	503 (Dutch)	1994
Vos	Seropositivity	Netherlands	31st Mar 20–11th May 20	NR	Weighted to population	88%	2306 (Dutch)	331
Indseth	Infection; Hospital admission; Mortality	Norway	1st Mar 20–18th Oct 20	NR	N/A	81%	450,801 (Non-Immigrants)	869,442
Labberton	Infection; Hospital admission	Norway	15th Jun 20–21st Mar 21	14 medical conditions	N/A	100%	53,890 (Norwegian)	28,642
Jefferies	Infection	New Zealand	2nd Feb 20–13th May 20	NR	N/A	69%	1091 (European)	412
Ishii	Infection	Japan	16th Jul 20 – NR	NR	N/A	75%	3242 (Japanese)	298
Saidel Odes	Infection	Israel	8th Sep 20–31st Dec 20	NR	Age, sex	81%	6381 (Jewish)	2137
Al Awaidy	Hospital admission; Mortality	Oman	14th Feb 20–23rd Jul 20	Hypertension, cardiovascular disease, lung disease	N/A	75%	40,859 (Omani)	28,523
Al Abri	Seropositivity	Oman	1st Jul 20–30th Nov 20	Presence of comorbidities	Selection bias	81%	11,582 (Omani)	5875
Abu Ruz	Mortality	United Arab Emirates	1st Mar 20–30th May 20	Diabetes, hypertension, anaemia, vitamin D deficiency, dyslipidaemia, chronic kidney disease, asthma, cancer, COPD	N/A	75%	486 (Middle Eastern)	2810
Al Zahmi	ICU admission	United Arab Emirates	26th Feb 20–31st May 20	Cardiac disease, hypertension, chronic lung disease, asthma, chronic kidney disease, diabetes, cancer, immunosuppression, HIV, medication	N/A	75%	73 (Emirati)	4,87
Hamadah	ICU admission; Mortality	Kuwait	24th Feb 20–20th Apr 20	Diabetes, hypertension, cardiovascular, asthma, cerebrovascular, hepatitis, cancer, hypothyroidism, renal disease, immunodeficiency, recent surgery	Age, smoking, comorbidities	88%	294 (Kuwait)	829

(Table 1 continues on next page)

First author	Outcomes	Country	Period of data collection	Comorbidities considered	Covariates adjusted for	Study quality %	Majority N	Minority N
(Continued from previous page)								
Al Kuwari	Infection	Qatar	18th Feb 20–18th Apr 20	Hypertension, diabetes, cardiovascular disease, lung disease, kidney disease, malignancy, tuberculosis, liver disease, immunodeficiency	N/A	88%	497 (Qatari National)	5188
Shaikh	ICU admission; Mortality	Saudi Arabia	1st May 20–1st Aug 20	Hypertension, obesity, diabetes, cardiovascular disease, respiratory diseases, malignancies, other chronic conditions	Age, BMI, comorbidities, ICU admission (for mortality only)	69%	131 (Saudi National)	434
Nasif	Mortality	Saudi Arabia	Nov 20–Jun 21	NR	N/A	50%	953 (Saudi National)	1664
Low- and middle-income countries								
Horta	Seropositivity	Brazil	14th May 21–24th Jun 20	NR	Age, sex	100%	2064 (White)	32,383
Da Silva	Infection	Brazil	27th Oct 20–11th Dec 20	NR	N/A	56%	2945 (White)	3039
Rodrigues	Mortality	Brazil	22nd Feb 20–10th May 21	Cardiovascular disease, diabetes, haematological disease, down syndrome, obesity, pulmonary disease, liver disease, renal disease, neurological disease	N/A	50%	435,144 (White)	405,057
Sansone	Mortality	Brazil	22nd Feb 20–4th Apr 21	Cardiopathy, haematological disorder, down syndrome, Hepatic disorder, asthma, diabetes, neurological disorder, chronic respiratory disease, immunosuppressive disorder, renal disease, obesity, other comorbidities	N/A	88%	309,646 (White)	276,009
Silva	ICU admission	Brazil	26th Feb 20–9th Oct 20	Obesity, cardiovascular disease	Age	50%	73,464 (White)	86,399
Ibarra-Nava	ICU admission	Mexico	28th Feb 20–3rd Aug 20	Diabetes, COPD, high blood pressure, chronic kidney disease	N/A	94%	412,368 (Non-Indigenous)	3487
Servan-Mori	Hospital admission; Mortality	Mexico	1st Mar 20–28th Feb 21	Diabetes, obesity, hypertension, COPD, asthma, health problems during pregnancy	Age, sex, comorbidities, timing of presentation to care, social deprivation	88%	787,856 (Non-Indigenous)	8022
Bojorquez-Chapela	Seroprevalence	Mexico	1st Nov 20–30th Apr 21	NR	N/A	75%	121 (Mexican-born)	227
Dahal	Mortality	Mexico	19th Feb 20–25th Mar 22	Pneumonia, diabetes, COPD, asthma, other disease, cardiovascular disease, obesity, chronic kidney failure	Age, sex	100%	2,151,140 (Non-Indigenous)	21,896
Ramli	Infection	Malaysia	1st Jan 21–31st Oct 21	Presence of comorbidities	N/A	56%	110 (Malay)	10
Utulu	Infection	Nigeria	Nov 20–Dec 20	Hypertension, diabetes	N/A	63%	157 (Other)	1337
Cifuentes	Mortality	Colombia	2nd Mar 20–26th Oct 20	NR	Age, sex, area of residence, health insurance, socioeconomic status	81%	971,078 (White)	62,140
Concha	Infection	Colombia	28th Mar 21–26th Apr 21	NR	Sex, village	81%	72 (Colombian)	380
Sultanoglu	Mortality	Cyprus	9th Mar 20–4th May 20	NR	N/A	13%	76 (Native)	32
Sacoto	Mortality	Ecuador	21st Feb 20–31st Dec 20	Presence of comorbidities	N/A	50%	205,718 (Mestizo)	46,047

(Table 1 continues on next page)

First author	Outcomes	Country	Period of data collection	Comorbidities considered	Covariates adjusted for	Study quality %	Majority N	Minority N
(Continued from previous page)								
Kadyrova	Infection; Seroprevalence	Kazakhstan	1st Apr 21–30th May 21	Presence of comorbidities	N/A	38%	55 (Kazakh)	45
Ikram	Infection	South Africa	NR	NR	N/A	69%	8 (White)	228
Jugwanth	Infection	South Africa	Nov 20–Dec 20	Diabetes, hypertension, HIV, tuberculosis, chronic kidney disease, heart disease, asthma/COPD, liver disease, cancer, pregnancy	N/A	44%	275 (White)	255
Stead	Infection	South Africa	1st Jan 20–14th Mar 20	Diabetes, hypertension, HIV	Sex, education, smoking status, profession, COVID-19 exposure, PPE training, public transport use, BMI, diabetes, hypertension, HIV	93%	114 (White)	1181

Table 1: **Description of studies by outcome, period of data collection, comorbidities considered, covariates adjusted for, study quality, and sample sizes for the ethnic majority group and minoritised ethnic groups.**

To disentangle ethnic inequalities in risk of testing positive for infection from SARS-CoV-2, from ethnic inequalities in prognosis once infected (i.e., hospital admission, ICU admission, mortality), we separated studies by their denominator in the following way: (1) general population studies including individuals with and without SARS-CoV-2 infection; (2) individuals with confirmed SARS-CoV-2 infection; and (3) individuals hospitalised with COVID-19.

As some studies used country of birth or migrant status as an indicator of ethnicity, and the majority ethnic group was not White in some studies, we conducted unadjusted meta-analyses to identify the risk of each outcome for minoritised ethnic groups (combined within each study) compared with the ethnic majority group, which varied depending on which ethnic group was the majority in the study country. Crude numbers were used to calculate unadjusted risk ratios (RR) for minoritised ethnic groups *versus* the ethnic majority group. We conceptualised majority groups in terms of power and privilege, and minority if the ethnic or Indigenous group meets one or more of the following criteria: the group is numerically smaller than the rest of the population; it is not in a social, economic, or politically dominant position; and it has a culture, language, religion or ethnicity that is distinct from that of the majority. This guided our decisions when selecting the ethnic majority group (for the unadjusted analyses), as we did not always use the reference group of the study or the statistical majority. This analysis is reported in supplementary materials.

We performed meta-analyses using the DerSimonian and Laird random effects model[24] to determine the pooled effect sizes with corresponding 95% Confidence Intervals (CIs). Levels of statistical heterogeneity were determined using the I^2 statistic.[25] Where there were sufficient data ($n > 10$), meta-regressions were used to explore whether region (low- and middle-income countries [LMIC]) *versus* high-income countries [HIC]) and time frame (before widespread vaccine roll-out *versus* after widespread vaccine roll-out) were drivers of heterogeneity. Publication bias was explored through visual funnel plots, Egger's test of asymmetry for meta-analyses with 10 or more studies.[26] All analyses were conducted using Stata SE 15.[27]

Synthesis without meta-analysis (SWiM)

Where data were not amenable to meta-analyses, for example, where it was not possible to extract or calculate effect sizes, we provide synthesis without meta-analysis (SWiM) in supplementary materials. Effect direction plots and sign tests were conducted to assess evidence of associations. The quantitative synthesis is reported in line with the SWiM guidelines.[28]

Sensitivity analyses

As several studies reported country of birth, nationality, or migrant status, as an indicator of ethnicity, sensitivity analyses were conducted, replicating the unadjusted analyses with these studies excluded. To further explore differences across regions (LMIC *versus* HIC), the adjusted meta-analyses were stratified by region, where there were sufficient data ($n > 10$). Additional sensitivity analyses were conducted, excluding studies with a high risk of bias (as determined using the JBI), to explore the impact on the main findings. The findings of the sensitivity analyses are presented in supplementary materials.

Role of the funding source

The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript. All authors had access to the data and critically reviewed and approved the manuscript as submitted.

Results

After removing 15,189 duplicates, 17,993 records were screened against eligibility criteria, resulting in 579 eligible studies (Fig. 1). After excluding studies which contained duplicate patient data, 77 studies comprising over 200,000,000 confirmed COVID-19 cases were included.[4,29–135] Thirty studies were conducted in Europe (11 in the UK and Ireland), 21 in North America (14 in USA, four in Mexico, three in Canada), eight in South America, 13 in Asia, four in Africa, and one in Oceania (Aotearoa/New Zealand) (Fig. S1). Most study designs were cohort (N = 50, 65%) or cross-sectional (N = 23, 30%) (Table S2). Details of the outcomes, comorbidities, and covariates included in each study are reported in Table 1.

The summary of scores for the JBI risk of bias items are presented in supplementary materials (Fig. S2) and the standardised scores for each study are presented in Table S2. Whist an indicator for ethnicity was reported in all 77 studies, only 29 (38%) studies measured ethnicity (or closely related indicators) in a valid and reliable way, and it was unclear how ethnicity was measured in 17 (22%) studies. These 29 studies also disaggregated broad ethnic groups, which we considered important to determine whether there was heterogeneity in COVID-19 outcomes when using a more granular categorisation of minoritised ethnic groups (e.g., South Asian and East Asian, as opposed to an aggregated Asian ethnic group).

Ten studies reported the risk of testing positive for infection across disaggregated ethnic groups. Compared to White majority people, Black people were more likely to be infected (K = 8, adjusted risk ratio [aRR] = 1.78, 95% CI = 1.59 to 1.99, I^2 = 99.1), as were South Asian

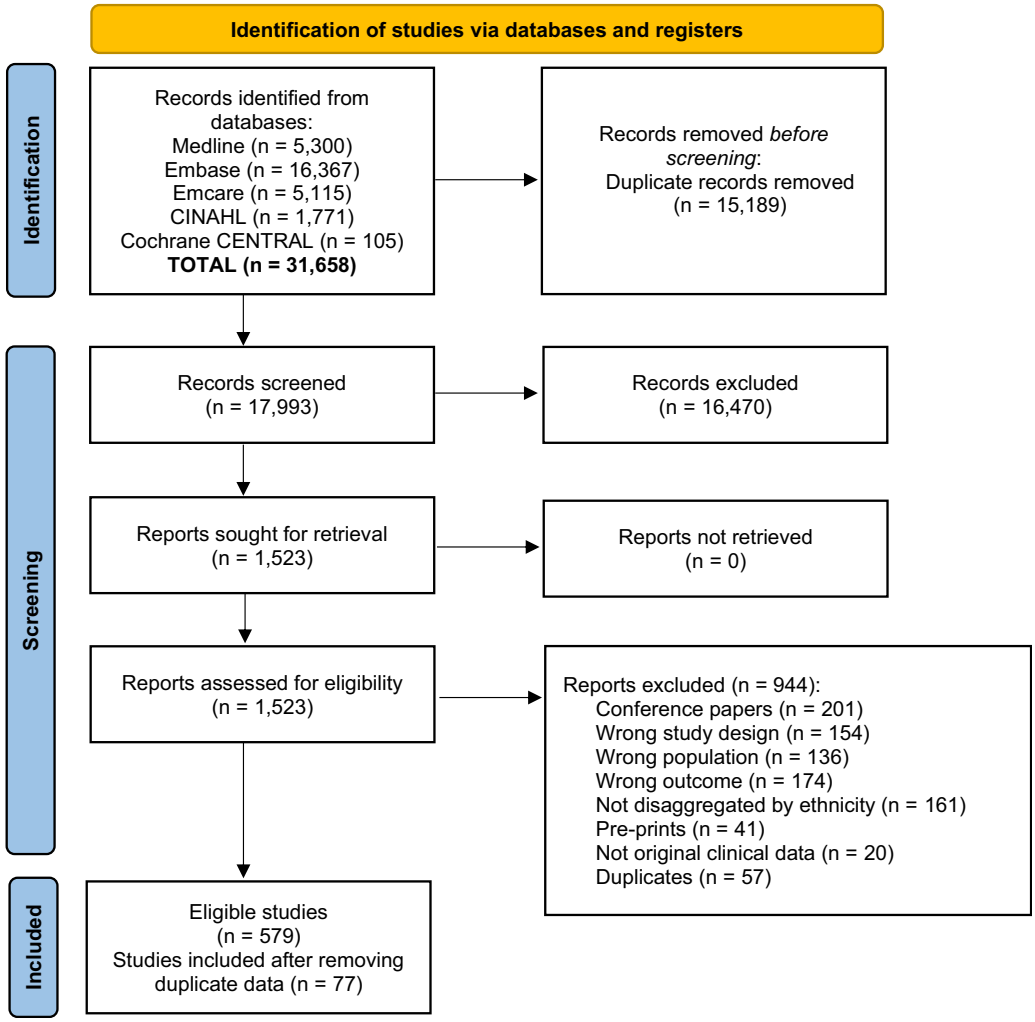


Fig. 1: **PRISMA flow diagram indicating the identification of studies via databases and registers.** (*) unadjusted risk ratio used (adjOR) adjusted odds ratio used (unadjOR) unadjusted odds ratio used. R: Risk Ratio. ROB: Risk of Bias.

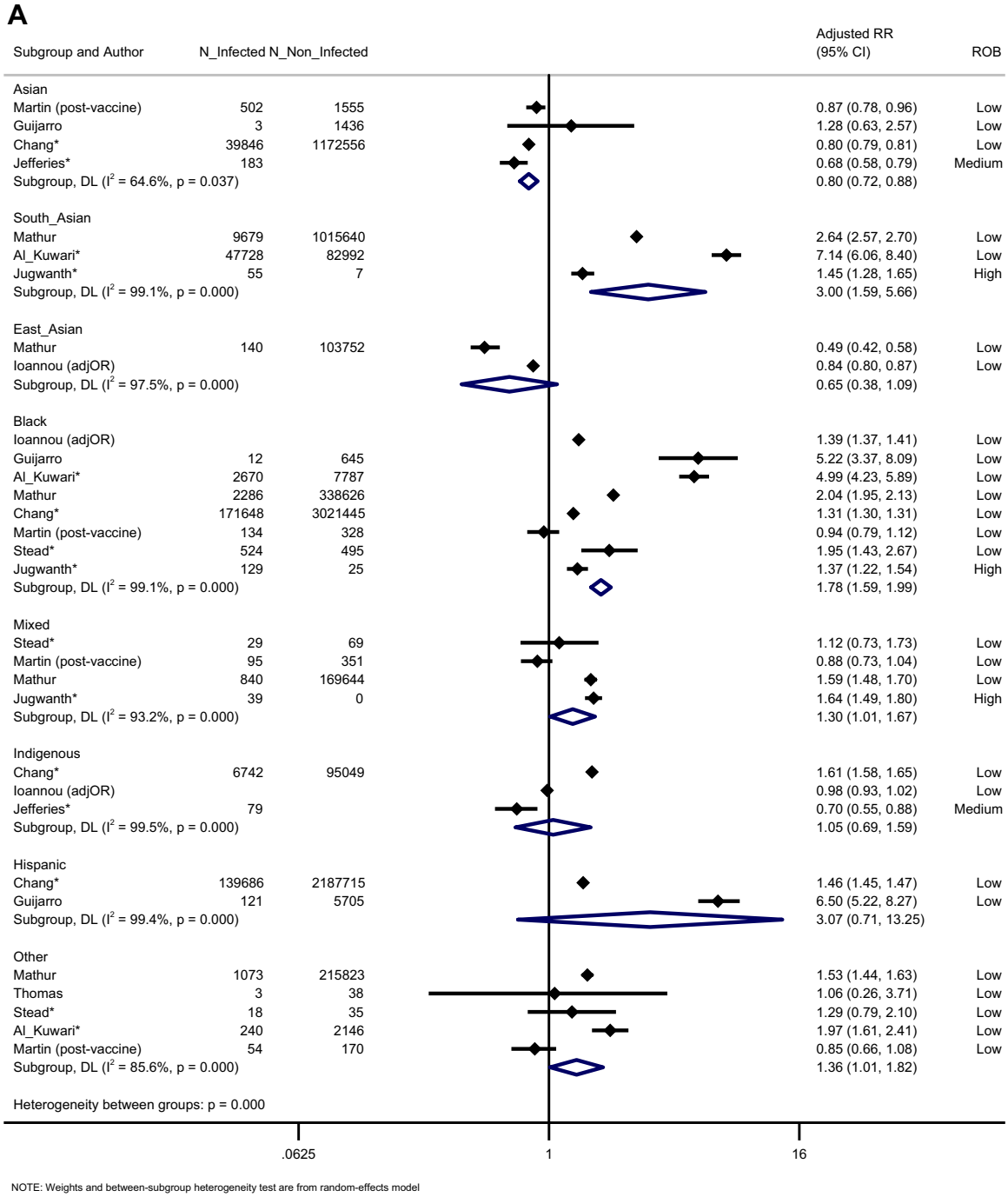


Fig. 2: **Forest plot showing the pooled effect sizes for the risk of infection (compared to White ethnicity) for each ethnic group (A) and the pooled effect sizes for the risk of seropositivity (compared to majority White ethnic group) for each minoritised ethnic group (B).** (*) unadjusted risk ratio used (adjOR) adjusted odds ratio used (unadjOR) unadjusted odds ratio used. R: Risk Ratio. ROB: Risk of Bias.

people (K = 3, aRR = 3.00, 95% CI:1.59 to 5.66, I^2 = 99.1), Mixed people (K = 4, aRR = 1.64, 95% CI:1.02 to 1.67, I^2 = 93.2), and those from Other ethnic backgrounds (K = 5, aRR = 1.36, 95% CI:1.01 to 1.82, I^2 = 85.6). Studies using an aggregated Asian ethnic group showed a reduced likelihood of testing positive. There was no difference in the risk of infection for East Asian, Indigenous, and Hispanic people, compared to

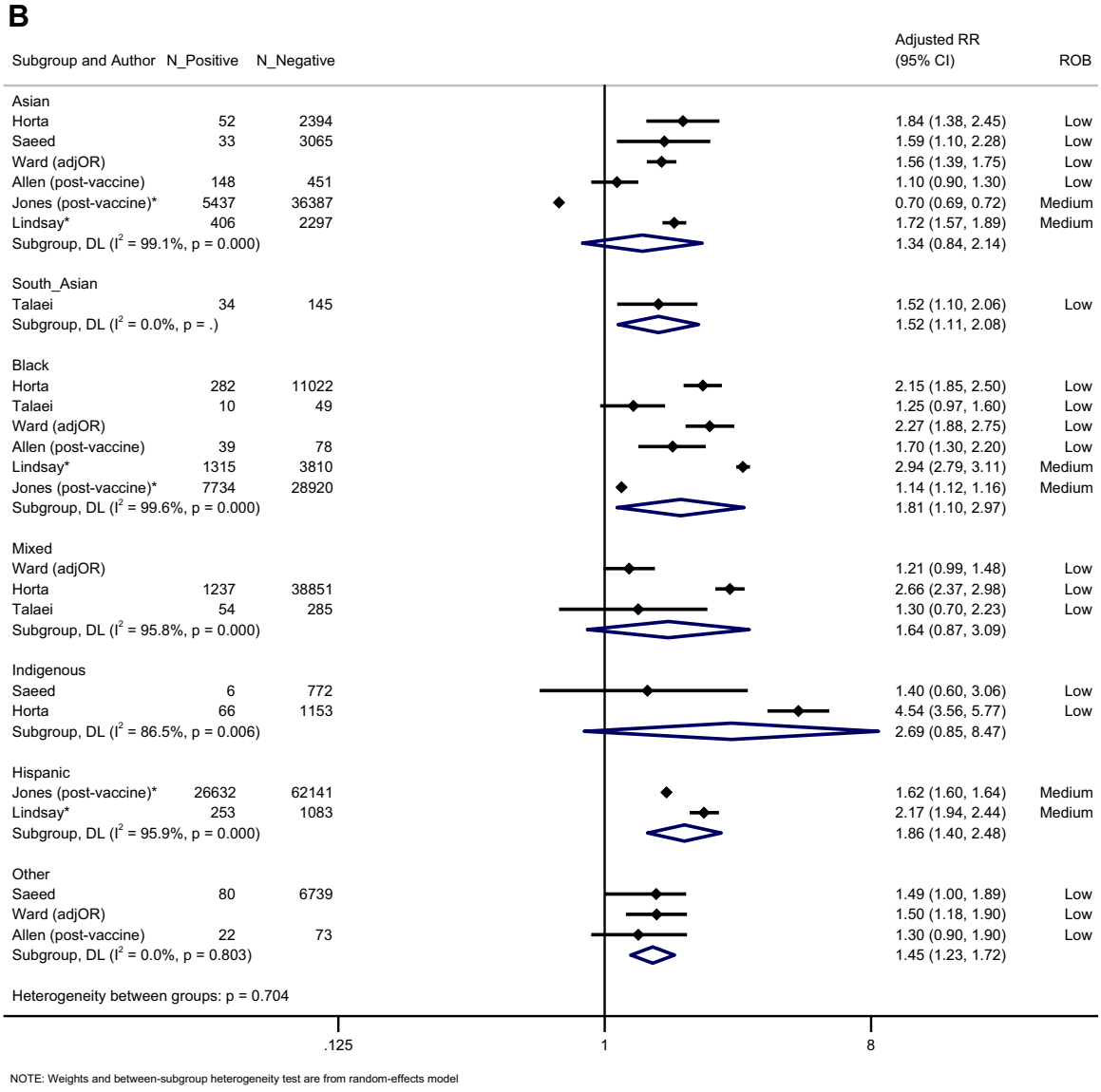


Fig. 2: Continued

the White majority (Fig. 2A). Evidence was rated as moderate certainty (Table S3). Egger's test showed no evidence of publication bias (p = 0.706, Fig. S18).

Seven studies investigated seroprevalence (due to infection rather than vaccination). Compared to White majority participants, Black and Hispanic ethnic groups were more likely to be seropositive (Black: K = 6, aRR = 1.81, 95% CI: 1.10–2.97, I^2 = 99.6; Hispanic: K = 2, aRR = 1.86, 95% CI: 1.40–2.48, I^2 = 95.9). South Asian people were more likely to be seropositive (K = 1, aRR = 1.52, 95% CI: 1.11–2.08), though this was not seen for studies which only reported outcomes for aggregated Asian ethnic groups (including both South and East Asian people) (K = 6, aRR = 1.34, 95% CI: 0.84–2.14, I^2 = 99.1). People from Other ethnic groups were also more likely to be seropositive (K = 3, aRR = 1.46, 95% CI: 1.23–1.72, I^2 = 0.00). There was no difference in the risk of seropositivity for Mixed ethnic groups or Indigenous people, compared to White majority participants (Fig. 2B). The certainty of evidence was moderate (Table S3).

Five population-based studies investigated the risk of hospitalisation (Fig. 3A). Black, Hispanic, and Indigenous people had an increased risk of hospitalisation (Black: K = 4, aRR = 1.71, 95% CI: 1.40–2.08, I^2 = 99.1; Hispanic: K = 3, aRR = 1.64, 95% CI: 1.32–2.04, I^2 = 93.4; Indigenous: K = 2, aRR = 1.91, 95% CI: 1.06–1.64, I^2 = 78.8) (moderate certainty). Six studies reported the risk of ICU admission in the general population (Fig. 3B). We identified an increased risk of ICU

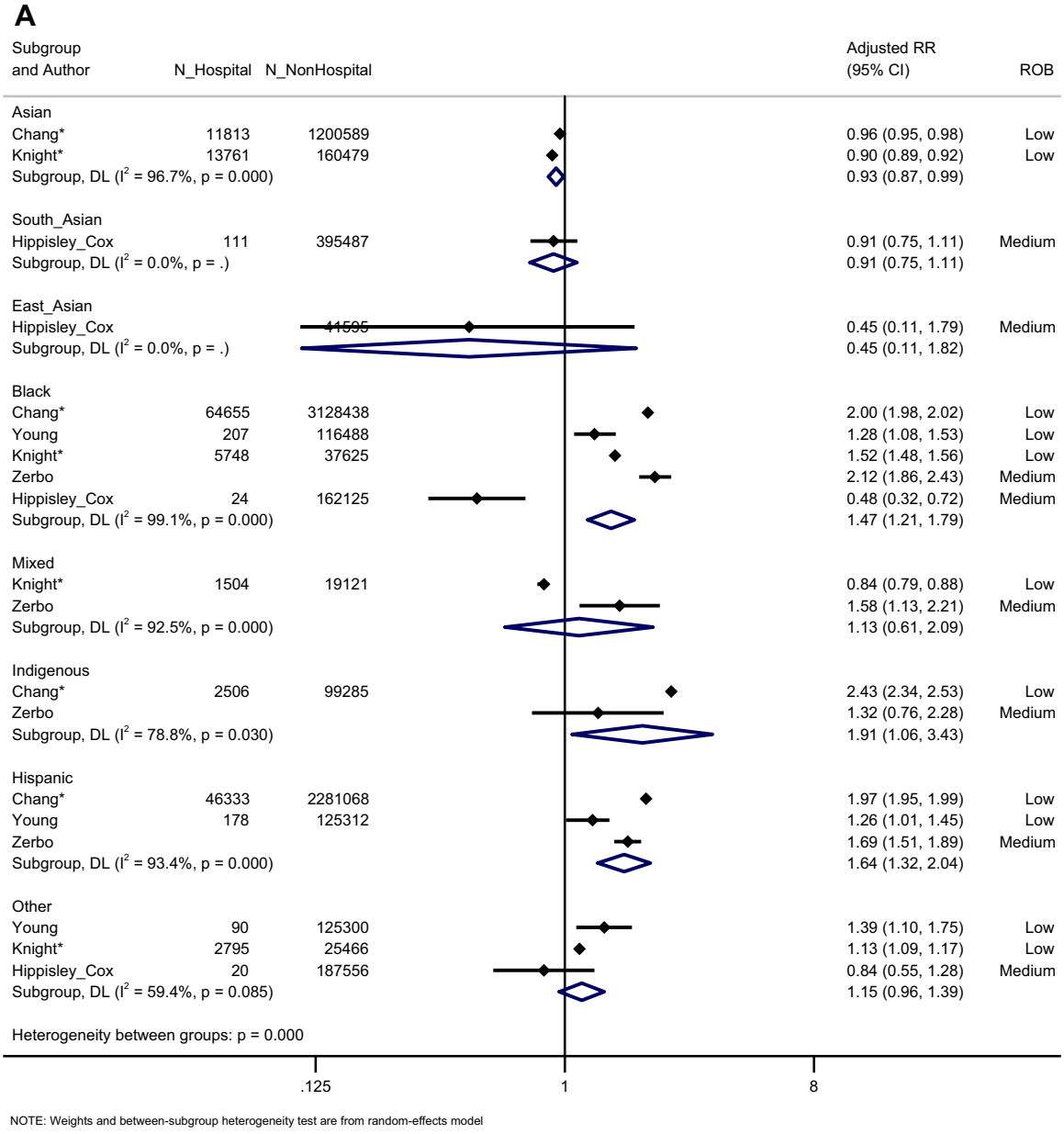


***Fig. 3*: Forest plot showing the pooled effect sizes for the risk of hospital admission (compared to White majority ethnic group) in the general population (A); the pooled effect sizes for the risk of ICU admission (compared to White majority ethnic group) in the general population (B) and the pooled effect sizes for the risk of mortality (compared to White majority ethnic group) in the general population (C).** (*) unadjusted risk ratio used (adjOR) adjusted odds ratio used (unadjOR) unadjusted odds ratio used. R: Risk Ratio. ROB: Risk of Bias.

admission for South Asian, East Asian, Black, Mixed, Indigenous, and Hispanic people, but not other ethnic groups, compared to the White majority (very low certainty). Five studies investigated the risk of mortality in the general population (Fig. 3C). We observed an increased risk of mortality for Mixed, Hispanic, and Indigenous people, compared to the White majority (Mixed: K = 2, aRR = 1.43, 95% CI: 1.13–1.82, I^2 = 0.0; Hispanic: K = 2, aRR = 1.30, 95% CI: 1.12–1.52, I^2 = 17.0; Indigenous: K = 2, aRR = 2.14, 95% CI: 1.99–2.31, I^2 = 0.0), but not Asian, Black or Other ethnic groups (very low certainty).

We assessed prognosis following infection. Four studies reported the risk of hospitalisation among confirmed COVID-19 cases (Fig. 4A). Hospitalisation risk was increased for Asian and Indigenous people,

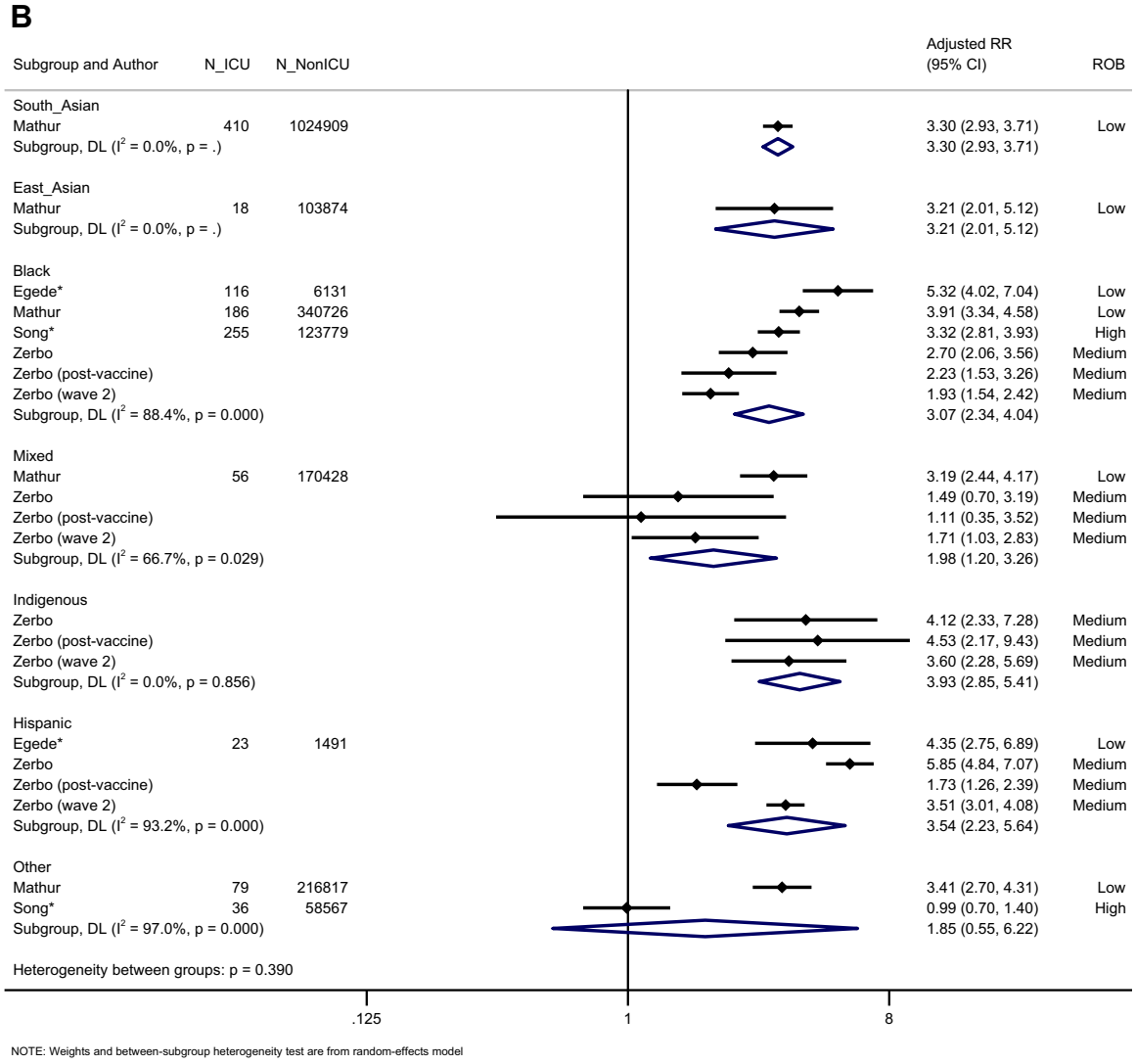
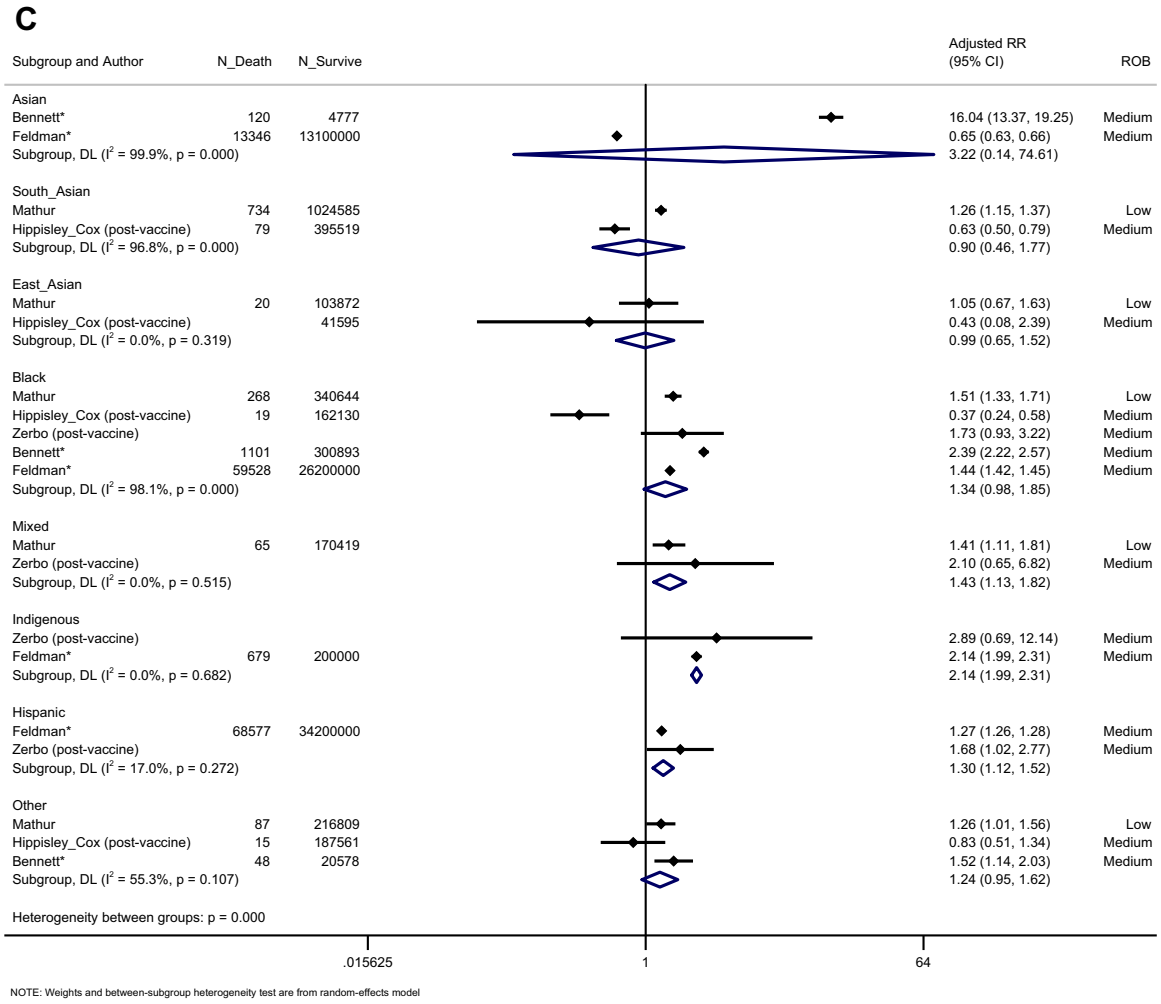


Fig. 3: Continued

compared to White majority participants (Asian: K = 1, aRR = 17.06, 95% CI: 15.91–18.30; Indigenous: K = 1, aRR = 2.53, 95% CI: 1.99–3.21), and reduced for South Asian and Mixed ethnicity people. People from East Asian and Other ethnic groups showed no difference in the risk of hospitalisation, following infection, compared to White ethnic groups. Four studies assessed the risk of ICU admission among confirmed COVID-19 cases, demonstrating a trend towards an increased risk for Black people (aRR = 1.53, 95% CI: 0.99–2.35, I^2 = 99.0), with no other differences across ethnic groups (Fig. 4B). However, three of the four studies had a high risk of bias. The risk of death among confirmed COVID-19 cases was reported in four studies, identifying no increased risk for any minoritised ethnic group, compared to the White majority, observing reduced risks for South Asian, East Asian, Mixed, and Other ethnic groups (Fig. 4C).

When assessing prognosis following hospitalisation, we noted that South Asian, East Asian, Asian (aggregated in studies, including South and East Asian people), Black, and Mixed groups were all more likely to be admitted to ICU compared to White majority participants. Those from Hispanic and Other ethnic groups were not at an increased risk, across four studies (Fig. 5A). In a synthesis of 11 studies, we observed an increased risk of mortality for Mixed ethnic groups (K = 6, aRR = 1.06, 95% CI: 1.02–1.09, I^2 = 97.0), compared to the White majority, with trends towards an increased risk for Indigenous peoples (K = 3, aRR = 2.03, 95% CI: 0.99–4.14, I^2 = 99.8) (Fig. 5B). Egger's test indicated no evidence of publication bias (p = 0.626).



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The findings of the unadjusted analyses are reported in supplementary materials. Minoritised ethnic groups were found to have an increased risk of infection, seropositivity, hospital admission (population-based studies only), ICU admission (population-based studies and studies of hospitalised patients with COVID-19 only), but not mortality (Figs. S3-S7). Sensitivity analyses replicated the unadjusted analyses, excluding studies which reported country of birth, nationality, and migrant status. In these analyses, minoritised ethnic groups were not at an increased risk of infection or hospital admission, though all other original findings remained the same (Figs. S8–S12).

Meta-regressions explored whether region (LMIC *versus* HIC) was associated with heterogeneity, where there were sufficient data ($n > 10$). Region did not explain heterogeneity for the pooled unadjusted or adjusted risk of infection (unadjusted: β = 0.91, 95% CI: 0.38–2.22, p = 0.838, I^2 = 99.95; adjusted: β = 0.98, 95% CI: 0.52–1.84, p = 0.950, I^2 = 99.77), unadjusted risk of seropositivity (β = 1.70, 95% CI: 0.57–5.06, p = 0.291, I^2 = 99.02), or the unadjusted risk of hospital admission (among studies of confirmed COVID-19 cases only, β = 2.22, 95% CI: 0.48–10.33, p = 0.250, I^2 = 99.93). Region was significantly associated with variance in the unadjusted risk of ICU admission, for studies of hospitalised COVID-19 cases (β = 3.40, 95% CI: 1.98–5.84, p = 0.001, I^2 = 92.97), whereby the unadjusted risk of ICU admission was greater in LMIC than in HIC (Fig. S20). In the unadjusted analyses, for mortality, region was significantly associated with heterogeneity among studies of confirmed COVID-19 cases (β = 3.57, 95% CI: 1.73 to 7.35, p = 0.004, I^2 = 98.75, Fig. S21), and among studies of hospitalised COVID-19 cases (β = 1.75, 95% CI: 1.02–3.02, p = 0.044, I^2 = 98.74, Fig. S22), whereby the risk of

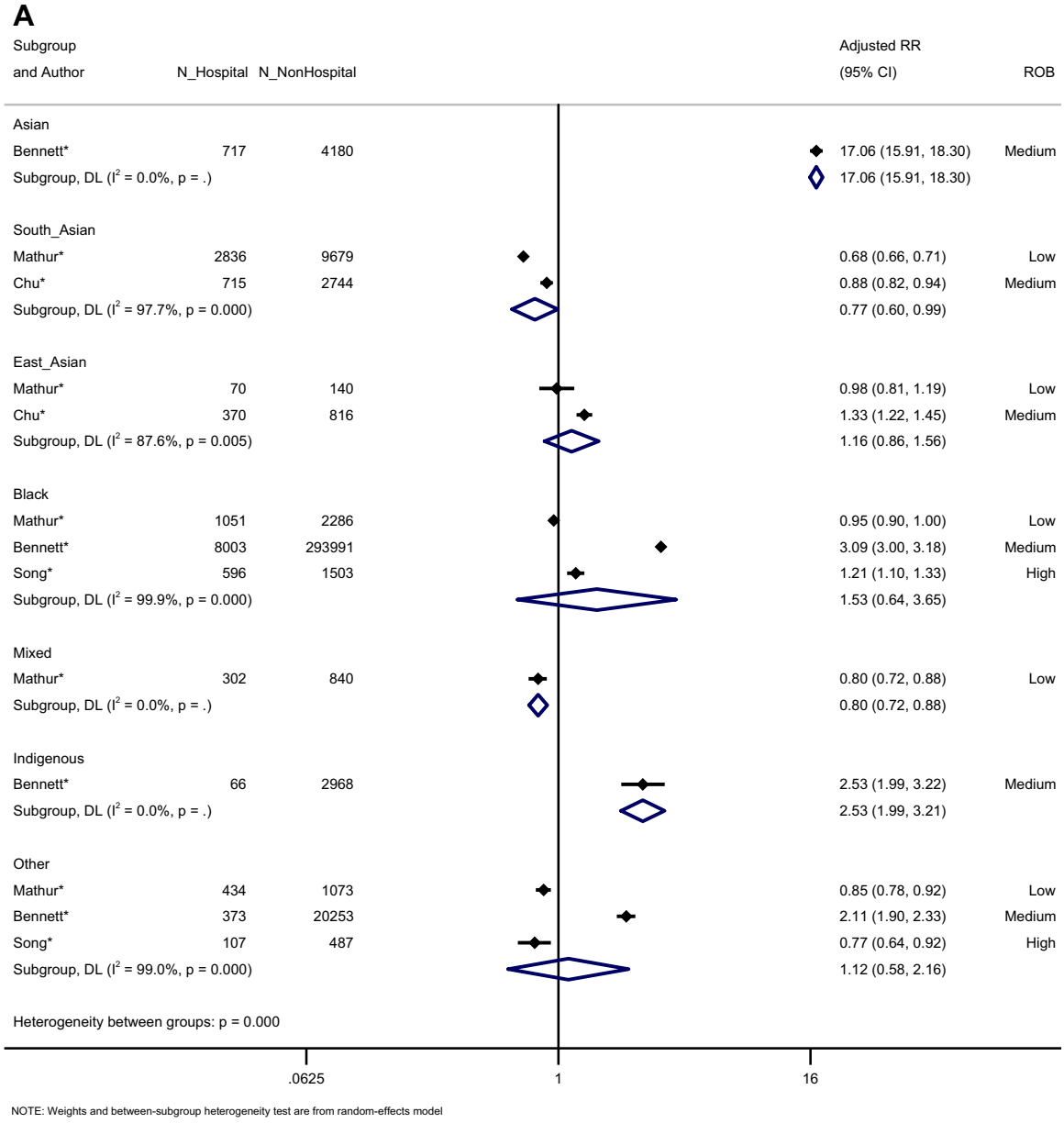
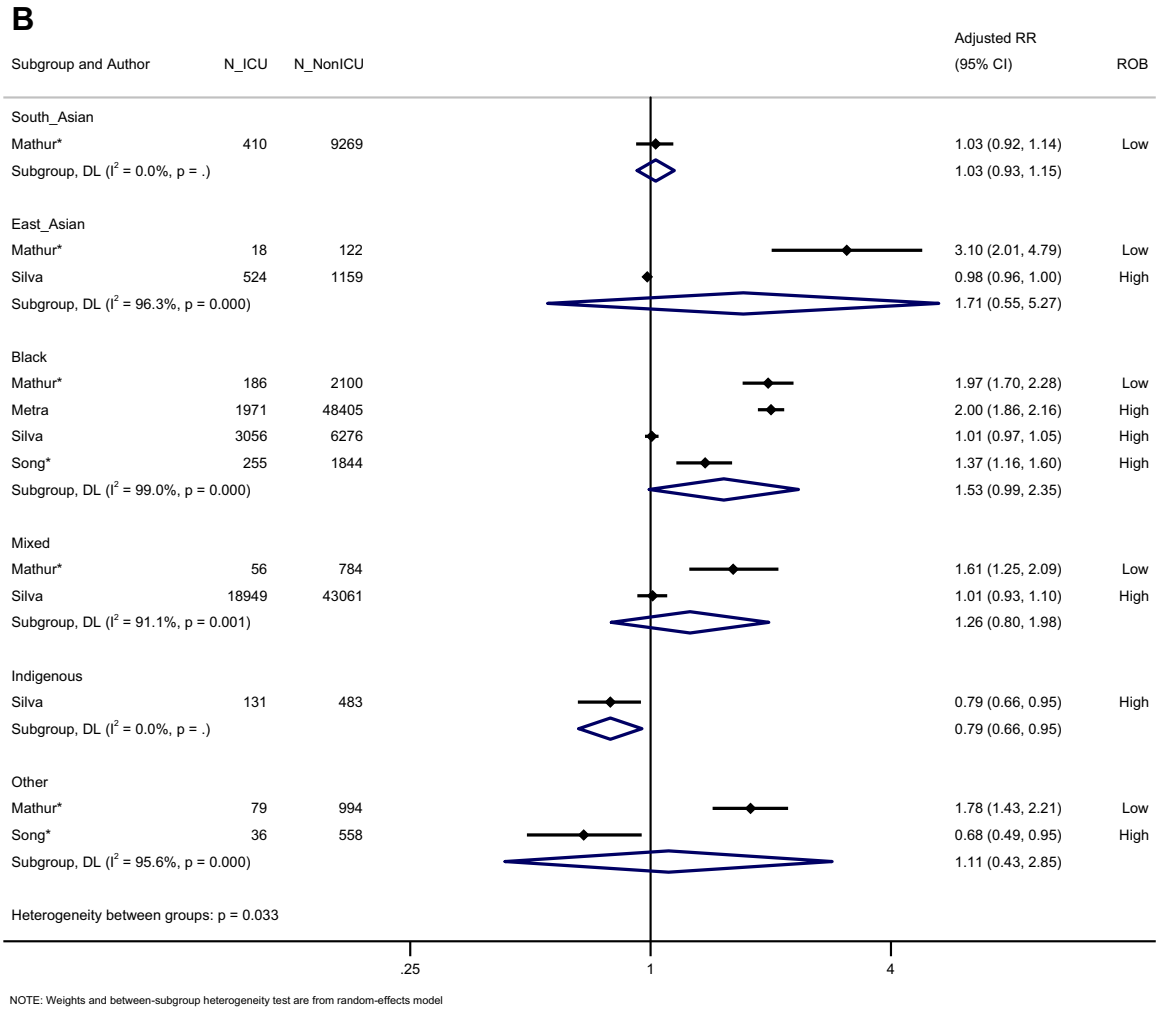


Fig. 4: **Forest plot showing the pooled effect sizes for the risk of hospitalisation (compared to White majority ethnic group) in confirmed COVID-19 cases (A); the pooled effect sizes for the risk of ICU admission (compared to White majority ethnic group) in confirmed COVID-19 cases (B); and the pooled effect sizes for the risk of mortality (compared to White majority ethnic group) in confirmed COVID-19 cases (C).** (*) unadjusted risk ratio used (adjOR) adjusted odds ratio used (unadjOR) unadjusted odds ratio used. R: Risk Ratio. ROB: Risk of Bias.

mortality was greater in LMIC than in HIC. However, region did not explain heterogeneity for the pooled adjusted risk of mortality among studies of hospitalised COVID-19 cases (β = 0.94, 95% CI: 0.63–1.40, p = 0.745, I^2 = 99.59). The findings of the meta-analyses, stratified by region, are presented in supplementary materials (Figs. S23–S26).

Meta-regressions were also used to explore whether time-frame (before vaccine roll-out *versus* after vaccine roll-out) was associated with heterogeneity, where there was sufficient data. Time-frame did not explain heterogeneity for the pooled unadjusted or adjusted risk of infection (unadjusted: β = 0.63, 95% CI: 0.21–1.92, p = 0.384, I^2 = 99.95; adjusted: β = 0.56, 95% CI: 0.28–1.12, p = 0.096, I^2 = 99.77). However, when examining Fig. 2A, the study conducted post-vaccine roll-out shows no difference in the adjusted risk of infection for each ethnic group, compared to the White



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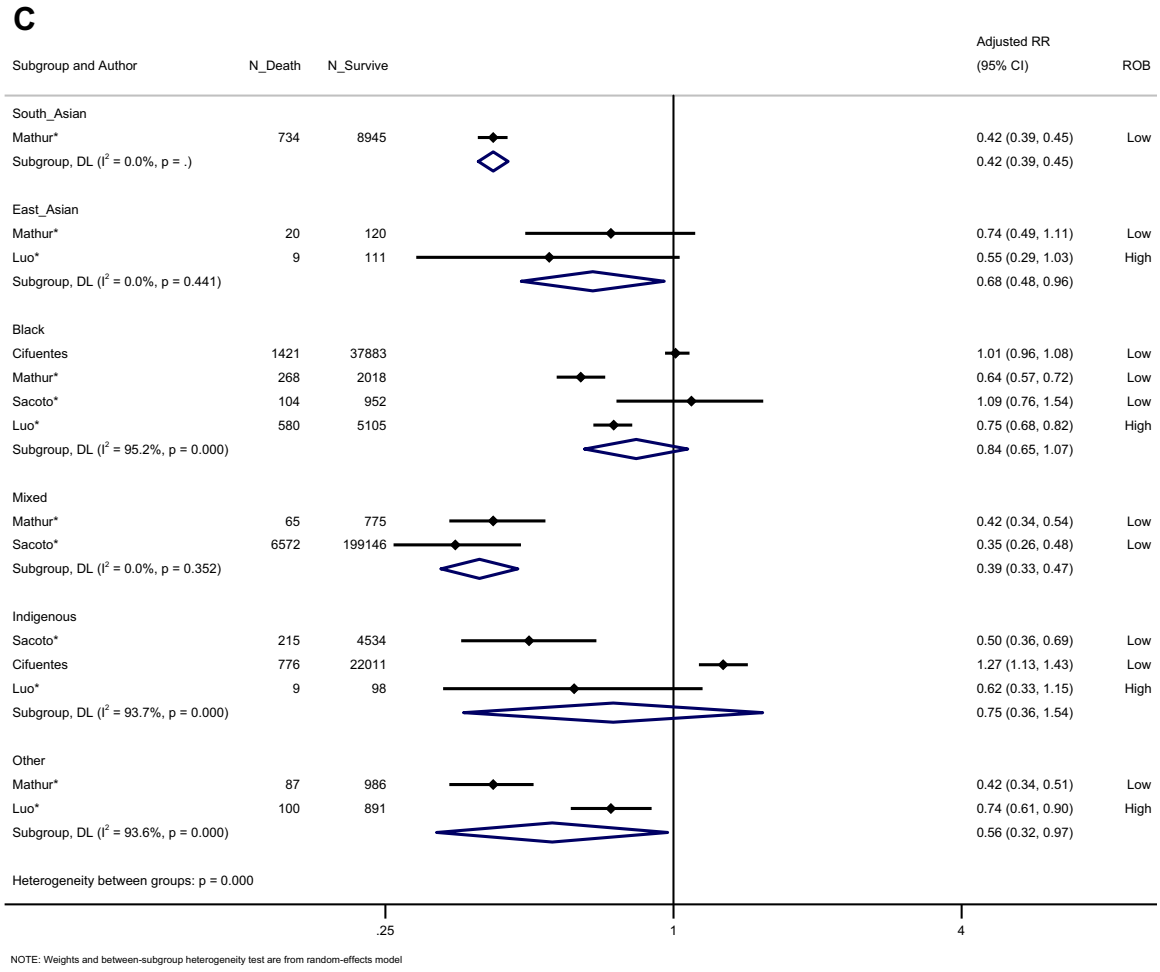
majority, contrasting most other effect sizes. Time-frame was not associated with heterogeneity in the unadjusted risk of seropositivity (β = 1.09, 95% CI: 0.49–2.36, p = 0.827, I^2 = 96.80). Time-frame did not explain heterogeneity for the unadjusted or adjusted risk of mortality among hospitalised cases (unadjusted: β = 1.23, 95% CI: 0.63–2.43, p = 0.522, I^2 = 99.37; adjusted: β = 0.79, 95% CI: 0.54–1.18, p = 0.245, I^2 = 99.55).

We provide SWiM for the findings of studies that were not amenable to meta-analysis. Across all outcomes, 16 studies were excluded from the unadjusted analyses, and 37 studies that either contained certain ethnic groups that were not included (i.e., if only one study reported an effect for that ethnic group), or could not be included at all (i.e., if the reference group was not White). The SWiM reports mixed findings, which may reflect the heterogeneity of the studies (supplementary materials: Tables S4–S6).

Discussion

We identified systematic inequalities experienced by minoritised ethnic groups, but to a varying extent across ethnic and Indigenous groups. We found that Black, South Asian, Mixed, and Other ethnic groups had a greater risk of testing positive for infection. The findings demonstrate large differences in exposure risk, which may be driving ethnic inequalities in severe outcomes. Almost all minoritised ethnic groups were at an increased risk of hospital admission and ICU admission, in population-based studies, yet these findings attenuated when examining outcomes among confirmed COVID-19 cases only. Additionally, Hispanic people were more likely to be seropositive, compared to the White majority. Seropositivity to any SARS-CoV-2 protein within populations that have not yet been vaccinated highlights a history of past infection; therefore, it may be that Hispanic groups had reduced access to testing early in the pandemic.



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We also observed differences in prognosis following hospitalisation, with South and East Asian, Black and Mixed ethnic groups being more at risk of ICU admission, and Mixed ethnic groups being more likely to die from COVID-19. Finally, we found that the unadjusted risk of severe disease (ICU admission and mortality) among hospitalised COVID-19 cases was greater for minoritised ethnic groups (*versus* the majority ethnic group) in LMIC compared to HIC. This could be due to HIC having more universal health care systems (excluding the USA). Our work is the most comprehensive summary of risk for minoritised ethnic groups globally, by using multiple markers for key clinical outcomes (molecular testing and serology for infection, and hospitalisation, ICU admission, and death for severe disease). Future work will likely include cohorts with differing levels of immunity (from previous infections, or heterogenous vaccine regimens) to different SARS-CoV-2 variants.

In agreement with previous meta-analyses, our data clearly demonstrates that the COVID-19 pandemic has exacerbated existing socioeconomic inequalities that disproportionately affect the health of minoritised ethnic and Indigenous groups.[7,13,14,16,17,136] Structural racism (discrimination embedded within systems) drives socioeconomic inequalities that increase the risk of exposure to COVID-19 infection.[137] During the pandemic, when multiple countries implemented strict lockdown measures, minoritised ethnic groups were more likely to be employed in sectors with increased exposure and were less likely to be able to self-isolate or work from home, due to economic precarity.[8,138,139] Minoritised ethnic groups were also more likely to live in overcrowded households with reduced access to open spaces (a consequence of racism and socioeconomic inequality), which could lead to frequent and prolonged exposure to airborne pathogens.[140] Among population-based studies, we observed an increased risk of severe disease for Black, Hispanic, South Asian, East Asian,

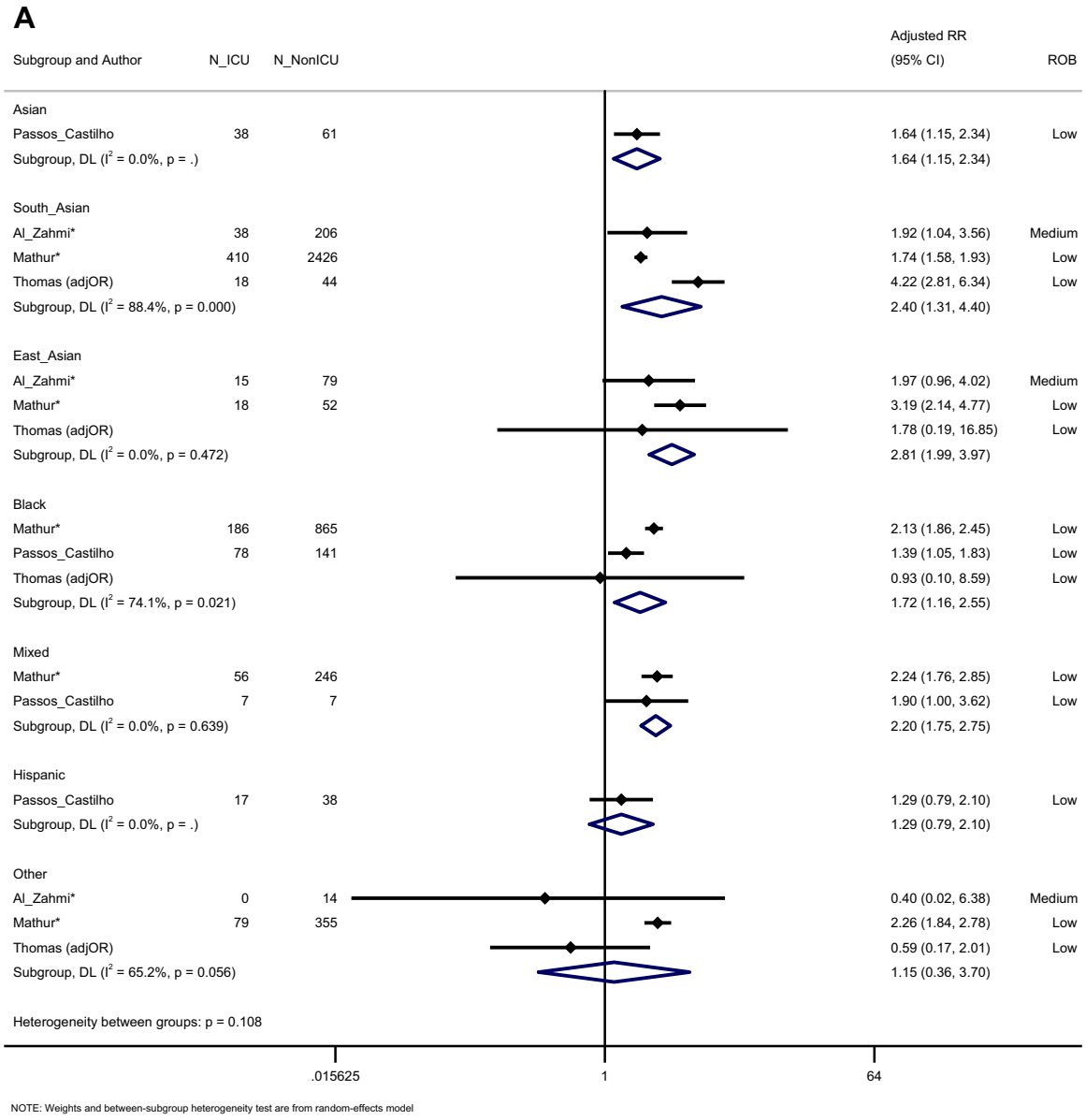
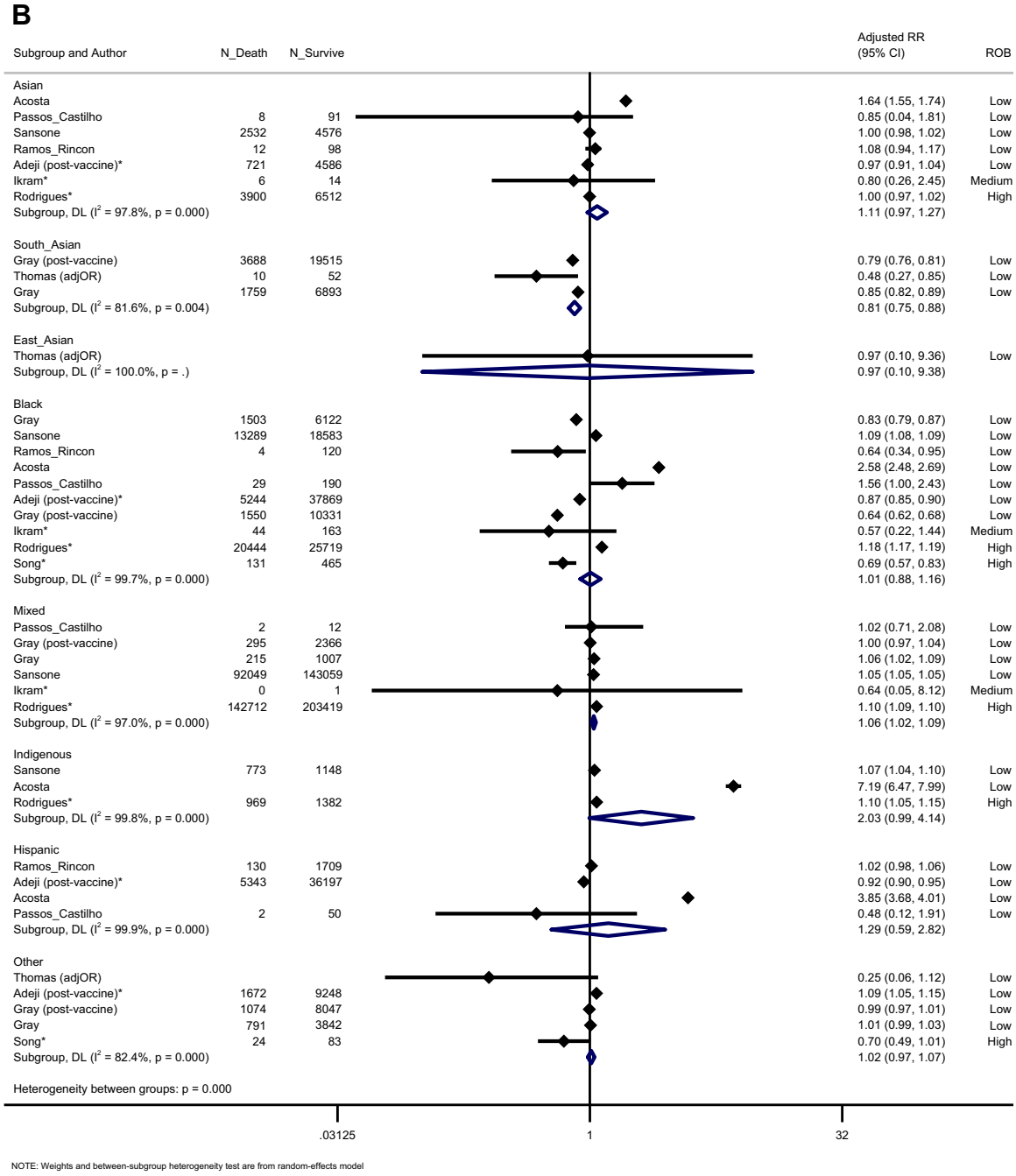


Fig. 5: **Forest plot showing the pooled effect sizes for the risk of ICU admission (compared to White majority ethnic group) in hospitalised patients with COVID-19 (A); the pooled effect sizes for the risk of mortality (compared to White majority ethnic group) in hospitalised patients with COVID-19 (B).** (*) unadjusted risk ratio used (adjOR) adjusted odds ratio used (unadjOR) unadjusted odds ratio used. R: Risk Ratio. ROB: Risk of Bias.

Mixed and Indigenous ethnic groups, but this attenuated once we examined prognosis following infection, demonstrating that differences in exposure risk may have driven the greater number of people experiencing severe disease. When assessing prognosis following hospitalisation, we observed ethnic inequalities in ICU admission and mortality, which potentially reflect poorer healthcare quality, or barriers to adequate healthcare (e.g., language barriers, migrant status, mistrust, disparities resulting from highly marketised healthcare in the US),[141] resulting from institutional racism.[136] Going forwards, unless significant effort is made to address these inequalities, it is likely that minoritised ethnic groups will continue to have increased exposure to respiratory viruses as the world learns to live with COVID-19.

Despite the importance of racism in relation to clinical outcomes, as captured by ethnicity, we found that the quality of ethnicity recording in studies to be suboptimal. Approximately a quarter of studies did not



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describe how they recorded ethnicity, despite investigating its relation to clinical outcomes. The most common method was using routinely recorded data, yet evidence shows that ethnicity is often miscoded, and this affects minoritised ethnic groups disproportionately.[142] Poor data may obscure the true extent of ethnic inequalities in COVID-19 health outcomes. In contrast to previous systematic reviews,[13,14,16,17] when we further disaggregated Asian groups, we found that South Asian people were at increased risk of infection, whereas East Asian people were not, and studies using an aggregated Asian group observed a decreased risk of infection. Our analysis demonstrates the importance of granularity in collecting ethnic categories to describe the

extent and drivers of ethnic inequalities in COVID-19 outcomes. If we only examined 'Asian' as one ethnic group, we may have missed the increased risk of infection among South Asian people, since the decreased risk of infection in all Asian people may have nullified this effect. We call for health systems to make a concerted effort to record self-identified ethnicity and for research to use disaggregated ethnic groups.[143]

Our systematic review has limitations. There was a large decrease in the number of studies and participants when we excluded studies with duplicate data, which was necessary to ensure rigour. This meant that in some analyses, estimates for certain ethnic groups were obtained from a small number of studies. Furthermore, although we set out to conduct a global synthesis, there were regions with no or limited data (e.g., Australia). Although there was some indication of publication bias in the unadjusted analyses, there was no evidence of publication bias in the adjusted analyses. Relatedly, the certainty of evidence for each outcome ranged from moderate to very low, mainly due to inconsistency within ethnic groups across studies. Additionally, heterogeneity between studies was high. However, we note that this is common with observational studies as I^2 is calculated as the proportion of total variation which is attributable to between-study variation, meaning studies with large sample sizes (i.e., small within-study variation), are likely to show inflated heterogeneity.[144] These limitations may result from the inclusion of observational studies from a range of regions. Nevertheless, we sought to present the most comprehensive work illustrating the disproportionate impact COVID-19 has had on minoritised ethnic groups. Clearly, this will have implications for future pandemics, especially if future pathogens have similar transmission dynamics and structural determinants.

In conclusion, we found clear evidence of systematic inequalities in COVID-19 health outcomes, experienced by minoritised ethnic groups, but to varying extents across ethnic groups during the first two years of the pandemic. We highlight the need to recognise and determine that pathways that lead to differing risks to COVID-19, before and after vaccine rollout periods. We observed large differences in exposure risk, particularly for Black, South Asian, Mixed, and Other ethnic groups. Hispanic groups may have had limited access to molecular testing early on in the pandemic, as reflected by the greater risk of seropositivity. Almost all minoritised ethnic groups being at an increased risk of severe outcomes among population-based studies (which attenuated when assessing outcomes only in confirmed cases), demonstrating the need for policy interventions to reduce exposure to infection. The differences in prognosis following hospitalisation may reflect poorer healthcare quality, illustrating the need for services and clinicians to ensure equitable care.[145] The COVID-19 pandemic has exposed and exacerbated ethnic inequalities in health, therefore response and recovery should focus on tackling the drivers of inequalities, including structural racism and racial discrimination.[146]

Contributors

PI, DK, LB, HT, SA, and SVK drafted the study protocol. DP, SS, PD, LJG, LBN, and MP provided critical feedback on the protocol. PD conducted the literature searches. PI screened all records, and DK, DP, SS, SK, LB, HT, and SVK contributed to the screening process and selection of included studies. PI initially extracted data, which were subsequently verified by a second reviewer (DK, DP, SS, SVK, HT) who also completed independent risk of bias scores. PI completed the data analysis, and all authors had access to the data. EK created the visual map and both EK and LJG supported the analyses. All authors critically reviewed and approved the manuscript as submitted.

Data sharing statement

The study protocol is published on PROSPERO: CRD42021284981. All extracted data and analytical codes are available from the corresponding author are available upon request.

Declaration of interests

SVK was co-chair of the Scottish Government's Expert Reference Group on Ethnicity and COVID-19 and a member of the Scientific Advisory Group on Emergencies (SAGE) subgroup on ethnicity. MP reports grants from Sanofi and Gilead Sciences and personal fees from QIAGEN, outside the submitted work.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.eclinm.2023.101877.

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