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Ethnicity, deprivation and mortality due to 2009 pandemic influenza A(H1N1) in England during the 2009/2010 pandemic and the first post-pandemic season

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SUMMARY

The relationship between risk of death following influenza A(H1N1)pdm09 infection and ethnicity and deprivation during the 2009/2010 pandemic period and the first post-pandemic season of 2010/2011 in England was examined. Poisson regression models were used to estimate the mortality risk, adjusted for age, gender, and place of residence. Those of non-White ethnicity experienced an increased mortality risk compared to White populations during the 2009/2010 pandemic [10·5/1000 *vs.* 6·0/1000 general population; adjusted risk ratio (RR) 1·84, 95% confidence interval (CI) 1·39-2·54] with the highest risk in those of Pakistani ethnicity. However, no significant difference between ethnicities was observed during the following 2010/2011 season. Persons living in areas with the highest level of deprivation had a significantly higher risk of death (RR 2·08, 95% CI 1·49-2·91) compared to the lowest level for both periods. These results highlight the importance of rapid identification of groups at higher risk of severe disease in the early stages of future pandemics to enable the implementation of optimal prevention and control measures for vulnerable populations.

Key words: England, ethnicity, influenza A(H1N1)pdm09, mortality, pandemic.

INTRODUCTION

Evidence has been published since the 2009 influenza pandemic which indicates that ethnic minority populations experienced worse health outcomes following pandemic influenza infection compared to other groups [1, 2]. This has also been observed during previous influenza pandemics [3–5]. The higher impact of pandemic influenza in minority populations has been related to a range of potential factors including higher rates of underlying chronic disease (increasing risk of influenza-related complications [4], in particular secondary bacterial infections) and barriers to accessing healthcare. The explanations underlying the latter have been related to socioeconomic status and cultural, educational and language factors [3, 4].

The 2009 H1N1 influenza pandemic is now recognized to have been due to a relatively mild novel influenza virus compared to historical influenza pandemics in the 20th century, in particular the 1918 Spanish influenza pandemic [6, 7]. However, severe and fatal cases did still occur due to infection with the 2009 pandemic virus [influenza A(H1N1)pdm09]; including in previously healthy individuals [2, 8, 9]. Higher rates of influenza A(H1N1)pdm09-associated morbidity and mortality were noted particularly in various ethnic minority or indigenous populations

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including in the USA [10], New Zealand [5, 11], Australia [5], Canada [5, 12] and in children in England [2]. The latter study [2] highlighted that children in particular ethnic groups were at elevated risk of death. Deprivation has also been identified as a risk factor for fatal outcome during the 2009 influenza pandemic [13]. The first post-pandemic influenza season in England, 2010/2011, was dominated by circulation of A(H1N1)pdm09, with intense activity mainly in young adults with many deaths and ICU admissions reported [14–16]. The role of ethnicity and deprivation as risk factors for severe disease during this first post-pandemic season has not been fully explored.

The present study aims to provide a more detailed account of the relationship between ethnicity, deprivation and influenza mortality in England during the 2009/2010 pandemic period and to extend the analysis to the immediate post-pandemic season in 2010/2011, which was dominated by circulation of influenza A (H1N1)pdm09, and to determine if there were any differences between these two time periods.

METHODS

Study population and case definition

Individual-level mortality surveillance was established from the start of the 2009/2010 pandemic to ascertain, confirm and report fatal cases related to influenza A (H1N1)pdm09 infection in England [8]. A confirmed fatal case was defined as a resident in England who died between 27 April 2009 (the date of the first pandemic cases reported from the UK [17]) and 30 April 2010, and between 1 September 2010 and 31 August 2011, where influenza A(H1N1)pdm09 infection was laboratory-confirmed and/or recorded on the death certificate as described previously [8]. As ethnicity status was not recorded for fatal cases in this study, patients were classified as 'from an ethnic minority (i.e. non-White), or 'non-ethnic' (i.e. White) based on available surname and first name using Onomap software [18] (http://www.onomap.org/Index.aspx). The Onomap software assigned each study subject into one of the UK 2001 census ethnic groups as follows (*a*) White – British; (*b*) White – Irish; (*c*) White – any other White background; (*d*) Asian or Asian British – Indian; (*e*) Asian or Asian British – Pakistani; (*f*) Asian or Asian British – Bangladeshi; (*g*) Asian or Asian British – any other Asian background; (*h*) Black or Black British – African; (*i*) Chinese; (*j*) Other ethnic group – any other ethnic group and (*k*) Unclassified. The patients were then grouped into White for groups (*a*)–(*c*), non-White for groups (*d*)–(*j*) and Unknown for group (*k*) patients.

Using patients' home postcode, place of residence was classified into rural/suburban and urban areas as defined by the Office for National Statistics (ONS) [19]. The deprivation status of their geographical area of place of residence was defined using the Index of Multiple Deprivation (IMD 2007) [20]. The deprivation score can be obtained for each postcode by data linkage between IMD 2007 and postcodes. The IMD 2007 consists of seven dimensions of deprivation (income; employment; health deprivation and disability; education, skills and training; barriers to housing and services; crime and disorder; living environment). These are weighted and combined to create an overall index of multiple deprivation.

Additional information was available on a subset of cases on underlying medical condition, antiviral use and influenza vaccine uptake through surveys to patients' family physicians. Due to incomplete data reporting and missing data, these variables were only analysed using univariable analysis (χ^2 test).

Population data

Population denominator data were obtained from ONS (mid-2009 population) for England and used to calculate cumulative mortality rates. England was divided into the nine government office regions at that time. Population data by 362 local authorities (LAs), ethnicity, sex and age group (0–15, 16–64, $\geqslant$65 years) were used in conjunction with data for the whole of the England by ethnicity and age in 5-year age groups. These were combined on the assumption that the age distributions of the 5-year age groupings were constant within the broader groups across LAs. The resulting data were aggregated to regions to provide a region, age, gender and ethnicity-specific denominator for analysis. IMD and residence area type were available at a much finer geographical level, the lower super output area (LSOA), of which there are 32 378 areas in England to enable analysis of the relationship with deprivation.

Analysis

Poisson regression models were used to estimate the mortality risk in relation to ethnicity, age, sex, region,

socioeconomic status (using IMD 2007 in quartiles), and residence area type (rural/suburban *vs*. urban). As no data source exists that provides a full population breakdown according to all of the variables of interest, analyses were conducted using two models based on two sets of variables, for which denominator data were available. These were: model A (ethnicity, age, sex and region, with interaction terms between age and ethnicity); and model B (IMD, residence area type, and region). IMD must be analysed at a fine geographical level due to high local variability. Results are presented as adjusted incident rate ratios (IRRs) with 95% confidence intervals (CIs) from Poisson regression analysis.

RESULTS

A total of 389 fatal cases associated with influenza A (H1N1)pdm09 infection were reported between 1 June 2009 and 30 April 2010 in England. Two hundred and ninety-one (74·8%) were from both laboratory-confirmed cases and death certificates; 51 (13·1%) were from laboratory-confirmed cases only and 47 (12·1%) from death certificates only. Out of these 389 influenza deaths, 211 (54·2%) were male. For 348 (89·4%) of these deaths full name information was available. Of these, for 342 cases ethnicity information could be derived using Onomap software, with ethnicity status unclassified for six deaths, and for five cases other information was not available. Therefore 337 cases were included in the analysis. Sixty-seven (19·9%) of these 337 cases were classified as being from an ethnic minority (i.e. non-White), a significantly higher proportion than observed for the whole population in England (12·5%) ($P < 0·0001$).

Table 1*a* shows the crude and adjusted cumulative mortality rates by ethnicity from model A for the 2009/2010 pandemic period. We found a significant interaction effect between age group and ethnicity, but not for other covariates; the Pearson goodness-of-fit test for the final model provided a P value of 0·889 (no evidence of lack of fit). Within the White population, adjusting for gender and geography, there was only a significantly increased mortality risk in those aged 45–64 years compared to those aged 25–44 years in 2009/2010, with a significantly decreased mortality risk in those aged 5–14 years (Table 1*a*). The overall adjusted mortality risk of non-White *vs*. White ethnicity in 2009/2010 was significantly higher with an IRR of 1·84 (95% CI 1·39-2·45). Stratifying by age group, non-White populations had significantly increased risk for those aged 1–4, 5–14, 45–64 and ⩾65 years compared to those aged 25–44 years in the White population in 2009/2010. There was no evidence of significant differences in mortality by region or gender in 2009/2010 after adjusting for ethnicity and age.

During the 2010/2011 season there were 463 deaths associated with influenza A(H1N1)pdm09 infection in England; 291 (95·5%) were from both laboratory-confirmed cases and death certificates; one (0·2%) was from a laboratory-confirmed case only and 20 (4·3%) from death certificates only. For 15 cases information on name was incomplete. Therefore, in total, 448 cases were used in the analyses, giving a crude mortality rate of 8·7/million in 2010/2011 compared to 6·5/million in 2009/2010. Forty-nine (10·9%) of these fatal cases were classified as from an ethnic minority, non-statistically lower than the national average ($P = 0·195$).

The adjusted cumulative mortality risk changed significantly between the two periods, with significantly higher mortality in 2010/2011 compared to 2009/2010 (IRR 1·48, 95% CI 1·27-1·72, adjusted for age, region and gender) for White populations and a non-significant decrease in mortality risk for non-White populations (adjusted IRR 0·73, 95% CI 0·51-1·06).

Table 1*b* shows the results from model A for 2010/2011. The adjusted mortality risk in the White population was again significantly elevated in those aged 45–64 years compared to those aged 25–44 years. However, there were significant decreases in risk for those aged 1–4, 5–14 and 15–24 years compared to those aged 25–44 years in 2010–2011. The risk profile changed in 2010/2011 with no significantly increased risk found in non-White ethnic populations by age compared to those aged 25–44 years of White ethnicity, except a borderline significant increase in those aged 1–4 years. Unlike 2009/2010, there was evidence of significant regional differences in 2010/2011, with higher mortality rates in the northern and central parts of the country compared to South-East England. The Pearson goodness-of-fit test for the final model provided a P value of 0·998.

We also examined mortality risk by individual ethnic groups. Denominator data for these groups are only available from ONS using broader age groups of <15, 15–59 and ⩾60 years for females and <15, 15–64 and ⩾65 years for males. Results are shown in Table 2(*a, b*) for the two periods. Crude mortality rates were significantly higher for those of Pakistani

Table 1. *Rates of influenza A(H1N1)pdm09-associated mortality per million according to ethnicity and age, sex and region during (a) 2009/2010 and (b) 2010/2011 seasons in England*

	Deaths	Deaths/ million	Crude IRR	95% CI	*P*	Adjusted IRR	95% CI	*P*
(*a*) 2009/2010								
Age effect in White ethnicity								
0	3	5·42	0·92	0·29–2·94	0·894	0·93	0·29–2·94	0·897
1–4	10	4·74	0·81	0·42–1·57	0·530	0·81	0·42–1·57	0·533
5–14	18	3·32	0·57	0·34–0·95	0·032	0·57	0·34–0·95	0·032
15–24	23	4·10	0·70	0·44–1·12	0·138	0·70	0·44–1·12	0·135
25–44	70	5·86	1 (ref.)			1 (ref.)		
45–64	101	8·75	1·49	1·10–2·03	0·010	1·50	1·10–2·03	0·009
⩾65	45	5·54	0·95	0·65–1·38	0·772	0·97	0·67–1·41	0·868
Total	270	5·96						
Age-specific *change* in risk for non-White ethnicity (age × ethnicity interaction)								
0	2	14·71	2·72	0·45–16·25	0·274	2·62	0·44–15·71	0·292
1–4	7	14·90	3·15	1·20–8·26	0·020	3·03	1·15–7·99	0·025
5–14	12	11·92	3·59	1·73–7·45	0·001	3·47	1·66–7·22	0·001
15–24	6	5·10	1·24	0·51–3·05	0·636	1·20	0·49–2·96	0·690
25–44	14	6·04	1·03	0·58–1·83	0·919	1·00	0·56–1·77	0·986
45–64	19	18·65	2·13	1·31–3·48	0·002	2·06	1·26–3·39	0·004
⩾65	7	20·41	3·68	1·66–8·17	0·001	3·46	1·55–7·74	0·002
Total	67	10·35						
Gender								
Male	182	7·14	1 (ref.)			1 (ref.)		
Female	155	5·90	0·83	0·67–1·02	0·081	0·82	0·66–1·02	0·073
Region								
East Midlands	38	8·55	1·50	0·98–2·30	0·061	1·49	0·97–2·28	0·065
East of England	30	5·20	0·91	0·58–1·44	0·699	0·91	0·58–1·43	0·682
London	64	8·25	1·45	1·00–2·11	0·052	1·26	0·86–1·85	0·230
North East	19	7·36	1·29	0·76–2·20	0·343	1·33	0·78–2·26	0·298
North West	48	6·96	1·22	0·82–1·83	0·323	1·23	0·82–1·83	0·313
South East	48	5·69	1 (ref.)			1 (ref.)		
South West	26	4·97	0·87	0·54–1·41	0·580	0·89	0·55–1·44	0·639
West Midlands	34	6·26	1·10	0·71–1·71	0·670	1·06	0·68–1·64	0·797
Yorkshire & Humber	30	5·71	1·00	0·64–1·58	0·990	0·99	0·63–1·57	0·975
Total	337	6·52						
(*b*) 2010/2011								
Age effect in White ethnicity								
0	6	10·83	1·32	0·58–3·01	0·508	1·32	0·58–3·01	0·510
1–4	8	3·79	0·46	0·22–0·95	0·036	0·46	0·22–0·95	0·036
5–14	11	2·03	0·25	0·13–0·46	0·000	0·25	0·13–0·46	0·000
15–24	18	3·21	0·39	0·24–0·65	0·000	0·39	0·24–0·65	0·000
25–44	98	8·20	1 (ref.)			1 (ref.)		
45–64	182	15·76	1·92	1·50–2·46	0·000	1·92	1·51–2·46	0·000
⩾65	76	9·36	1·14	0·85–1·54	0·387	1·15	0·86–1·56	0·347
Total	399	8·81						
Age-specific *change* in risk for non-White ethnicity (age × ethnicity interaction)								
0	2	14·71	1·36	0·27–6·73	0·708	1·45	0·29–7·20	0·648
1–4	5	10·64	2·81	0·92–8·58	0·070	3·00	0·98–9·19	0·054
5–14	4	3·97	1·96	0·62–6·15	0·250	2·09	0·67–6·59	0·206
15–24	2	1·70	0·53	0·12–2·28	0·393	0·57	0·13–2·48	0·457
25–44	15	6·47	0·79	0·46–1·36	0·391	0·86	0·50–1·48	0·575
45–64	16	15·70	1·00	0·60–1·66	0·989	1·09	0·65–1·82	0·748
⩾65	5	14·58	1·56	0·63–3·85	0·337	1·75	0·70–4·34	0·230
Total	49	7·57						
Gender								
Male	236	9·26	1 (ref.)					

Table 1 (*cont.*)

	Deaths	Deaths/ million	Crude IRR	95% CI	*P*	Adjusted IRR	95% CI	*P*
Female	212	8·07	0·87	0·72–1·05	0·146	0·85	0·71–1·03	0·093
Region								
East Midlands	48	10·80	2·02	1·35–3·04	0·001	2·01	1·34–3·02	0·001
East of England	31	5·38	1·01	0·64–1·59	0·974	1·01	0·64–1·59	0·977
London	46	5·93	1·11	0·74–1·68	0·613	1·12	0·74–1·69	0·608
North East	27	10·46	1·96	1·22–3·16	0·006	1·95	1·21–3·14	0·006
North West	99	14·36	2·69	1·89–3·83	<0·001	2·69	1·89–3·82	0·000
South East	45	5·34	1 (ref.)			1 (ref.)		
South West	28	5·36	1·00	0·63–1·61	0·987	0·99	0·62–1·59	0·978
West Midlands	53	9·76	1·83	1·23–2·72	0·003	1·83	1·23–2·72	0·003
Yorkshire & Humber	71	13·51	2·53	1·74–3·68	<0·001	2·52	1·74–3·66	0·000
Total	448	8·67						

IRR, Incident rate ratio; CI, confidence interval; ref., reference category.

Table 2. *Univariable and multivariable results between influenza A(H1N1)pdm09-associated mortality risk and age, sex and ethnicity group during the (a) 2009/2010 and (b) 2010/2011 seasons in England*

	Deaths/pop. (thousands)	Deaths/ million	Crude IRR	95% CI	*P*	Adjusted IRR	95% CI	*P*
(*a*) 2009/2010								
Age <15	52/9647	5·39	0·75	0·56–1·02	0·067	0·74	0·55–1·00	0·048
Age 15–59/64	229/32 070	7·14	1 (ref.)			1 (ref.)		
Age >59/64	56/9939	5·63	0·79	0·59–1·06	0·112	0·88	0·66–1·19	0·411
White	270/45 304	5·96	1 (ref.)			1 (ref.)		
Bangladeshi	5/372	13·44	2·25	0·93–5·46	0·072	2·12	0·87–5·18	0·098
Black	4/1475	2·71	0·46	0·17–1·22	0·118	0·42	0·15–1·13	0·084
Indian	17/1408	12·07	2·03	1·24–3·31	0·005	1·87	1·14–3·08	0·013
Pakistani	20/983	20·35	3·41	2·17–5·38	<0·001	3·37	2·13–5·35	0·000
Other	21/2114	9·93	1.67	1·07–2·60	0·024	1·60	1·02–2.52	0·041
Male	182/25 441	7·15	1 (ref.)			1 (ref.)		
Female	155/26 215	5·91	0·83	0·67–1·02	<0·001	0·84	0·68–1·04	0·111
(*b*) 2010/2011								
Age <15	36/9647	3·73	0·38	0·27–0·54	0·000	0·38	0·27–0·54	0·000
Age 15–59/64	313/32 070	9·76	1 (ref.)			1 (ref.)		
Age > 59/64	99/9939	9·96	1·02	0·81–1·28	0·860	1·04	0·83–1·31	0·738
White	399/45 304	8·81	1 (ref.)			1 (ref.)		
Bangladeshi	4/372	10·75	1·22	0·46–3·27	0·692	1·45	0·54–3·91	0·458
Black	7/1475	4·75	0·54	0·26–1·14	0·105	0·66	0·31–1·41	0·282
Indian	13/1408	9·23	1·05	0·60–1·82	0·867	1·14	0·65–1·98	0·653
Pakistani	10/983	10·18	1·16	0·62–2·16	0·652	1·18	0·63–2·21	0·614
Other	15/2114	7·10	0·81	0·48–1·35	<0·001	0·96	0·57–1·62	0·877
Male	236/25 441	9·28	1 (ref.)			1 (ref.)		
Female	212/26 215	8·09	0·87	0·72–1·05	0·147	0·86	0·71–1·04	0·113

IRR, Incident rate ratio; CI, confidence interval; ref., reference category.
* Adjusted by region and gender.

and Indian ethnicity compared to the White population in 2009/2010, and after adjusting for region and gender, the mortality risk was still significantly higher in Pakistani and Indian groups with the greatest risk observed in those of Pakistani ethnicity (Table 2*a*). In general, there was little

Table 3. *Modelling results between influenza A(H1N1)pdm09-associated mortality risk and Index of Multiple Deprivation (IMD) quartiles and rural/suburban and urban area type during the (a) 2009/2010 and (b) 2010/2011 seasons in England*

	Deaths/pop. (thousands)	Deaths/ million	Crude IRR	95% CI	*P*	Adjusted IRR	95% CI	*P*
(*a*) 2009/2010								
IMD Q1	59/12 799	4·61	1 (ref.)			1 (ref.)		
IMD Q2	67/12 808	5·23	1·13	0·80–1·61	0·478	1·14	0·80–1·63	0·457
IMD Q3	83/12 749	6·51	1·41	1·01–1·97	0·043	1·38	0·98–1·94	0·067
IMD Q4	128/12 733	10·05	2·18	1·60–2·97	0·000	2·08	1·49–2·91	0·000
Urban	291/41 357	7·04	1 (ref.)			1 (ref.)		
Rural	46/9733	4·73	0·67	0·49–0·92	0·012	0·85	0·61–1·19	0·352
(*b*) 2010/2011								
IMD Q1	73/12 802	5·70	1 (ref.)			1 (ref.)		
IMD Q2	88/12 806	6·87	1·21	0·88–1·64	0·238	1·16	0·85–1·59	0·348
IMD Q3	111/12 751	8·71	1·53	1·14–2·05	0·005	1·46	1·08–1·97	0·014
IMD Q4	176/12 732	13·82	2·42	1·85–3·18	0·000	2·08	1·55–2·78	0·000
Urban	381/41 357	9·21	1 (ref.)			1 (ref.)		
Rural	67/9733	6·88	0·75	0·58–0·97	0·028	0·95	0·72–1·25	0·697

IRR, Incident rate ratio; CI, confidence interval; ref., reference category.
* Adjusted for age and gender.

power to test for age differences across individual ethnicities (interactions), but there were significantly higher rates in Pakistanis aged <15 years in 2009/2010 ($P = 0·001$) with 55% in this age group; and significantly higher rates in Indians aged >60 years in 2010/2011 ($P = 0·018$) with 31% in this age group; otherwise, the bulk of cases in non-White groups (~61%) was in the 15–59 years age group for non-White ethnicities. Overall, the adjusted mortality risk was not significantly different during the 2010/2011 season in different ethnic groups compared to those of White ethnicity (Table 2*b*). A non-significantly decreased mortality risk was seen in those of Black ethnicity compared to the White population in both periods.

Table 3(*a, b*) presents the results by IMD (quartiles) and area type (rural/suburban *vs.* urban). Mortality risk adjusted for age group and gender was significantly increased in patients living in areas with higher levels of deprivation in both 2009/2010 and 2010/2011, with a slightly higher risk in IMD quartile 3 and a significant, twofold increase for IMD quartile 4 (most deprived) compared to IMD quartile 1 (least deprived) for both periods. The effect of IMD was found to have a linear relationship with risk of death with an overall IRR of 1·29 (95% CI 1·20-1·38) per quartile increase in IMD. Patients living in rural or suburban areas had a lower risk of death than their urban counterparts for both study periods, although this difference was not significant after adjusting for deprivation levels.

During the 2009/2010 pandemic period, no difference was found in the proportion of fatal cases reported to have any underlying medical conditions between White and non-White patients (74·3%, 182/245 *vs.* 74·3%, 52/70, $P = 1·0$). Available data from the 2010/2011 period showed no significant difference in proportion of cases with any underlying medical conditions between White (64·9%, 237/365) and non-White cases (61·5%, 32/52, $P = 0·632$).

During the 2009/2010 pandemic period, data for reported influenza antiviral use between White and non-White cases did not show a significant difference (65·2%, 45/69 *vs.* 75·0%, 12/16, $P = 0·453$). In 2010/2011, there was no significant difference in the proportion of cases who received antivirals between White and non-White patients (33·6%, 37/110 *vs.* 30·8%, 4/13, $P = 0·836$). No influenza vaccine uptake data were available for analysis during the 2009/2010 period, and no significant difference between White and non-White cases was found in influenza vaccine uptake during 2010/2011 (20·5%, 45/220 *vs.* 25·0%, 6/24, $P = 0·603$).

DISCUSSION

Our study has shown that non-White ethnic minorities, in particular those of south Asian descent, experienced

a significantly increased risk of fatal outcome following influenza A(H1N1)pdm09 infection during the 2009/2010 influenza pandemic period in England compared to the White population. This excess risk appeared to be focused in the youngest and oldest age groups; but there were no significant differences according to gender or region in non-White ethnicities. This elevated risk of death in those from ethnic subgroups was no longer observed in the immediate post-pandemic 2010/2011 season, although the overall mortality risk in the general population was higher in 2010/2011 compared to that seen in 2009/2010. In addition, in both periods, patients living in areas with a higher level of deprivation had an increased risk of mortality associated with influenza A(H1N1)pdm09 infection compared to those living in the most affluent areas.

Our findings are of public health importance in that the novel pandemic influenza A(H1N1)pdm09 virus was associated with increased impact in non-White ethnic populations, particularly those of Asian heritage, during the 2009/2010 pandemic period compared to the White population. No such increased relative impact was found during the following non-pandemic influenza season, when the same virus strain continued to circulate with even higher population impact. Available historical data suggest that an increased risk of adverse health outcome including higher mortality is likely to occur in specific ethnic subgroups during an influenza pandemic [4]. Earlier published data indeed showed the disproportionately higher impact on ethnic minority populations during the 2009/2010 pandemic [2, 5, 10]. Previous studies on the impact of influenza have focused primarily on the impact during a pandemic period [1–5] but not during a post-pandemic period. The explanations for our findings of changes in risk over time are likely to be multifactorial and complex. The elevated impact of pandemic influenza in specific ethnic sub-populations could be due to either increased rates of infection and/or infection-severity ratios compared to the White population. Disentangling these two components is challenging. Importation and initial spread of a novel respiratory virus depends upon a range of factors including migration and mixing patterns. The roles of genetic factors on human susceptibility to influenza have long been suspected [21] but conclusive evidence is not available [22]. In terms of case severity, a recent study seemed to suggest that a certain gene might be linked to increased severity of influenza in Chinese populations during the 2009/2010 pandemic period when comparing severe with mild cases [23].

Other explanatory factors for elevated case severity may relate to poor underlying health status and access to preventive interventions. However, the proportion of cases with pre-existing underlying medical conditions during the 2009/2010 pandemic was similar for White and non-White patients. Available data did not show any significant differences in influenza vaccine uptake between the White and non-White cases. Although, data on influenza vaccination coverage among different ethnic populations have not been collected in England; an earlier study on pneumococcal vaccine uptake in England and Wales [24] did show that the uptake rates for pneumococcal vaccine for the total population are substantially higher in areas where >99% of the population are White, which might indicate a similar situation for other vaccines, such as that for influenza. This highlights the importance of gathering influenza vaccine uptake data by ethnicity, which is now being implemented into the UK.

Differences in healthcare-seeking behaviour might be a possible contributory factor. The disproportionately poorer health outcomes experienced by ethnic minority populations during the pandemic period may be associated with limited access to healthcare facilities, and greater cultural, educational and linguistic obstacles in adopting pandemic influenza control measures, unfavourable environmental factors (such as household crowding and poor quality housing), behavioural differences in response to influenza, [5, 25]. Available preliminary data, however, did not show any significant differences in influenza antiviral use between the White and non-White patients. Further work is required to better understand any differences.

Finally the strong association we found between higher deprivation and increased risk for severe disease might play a role in the higher mortality risk observed in the non-White ethnic minorities. An earlier study [13] showed that the mortality risk significantly increased in the deprived areas during the 2009/2010 influenza pandemic. Our results demonstrate that this increased risk was maintained into the post-pandemic period in 2010/2011 influenza season. This indicates that deprived areas are more vulnerable to the impact of influenza infection. Socioeconomic deprivation may result in poorer health outcomes because of a range of potential explanatory factors, such as poorer access to or use of healthcare services, lower vaccine uptake, overcrowding and poorer nutrition.

Some assumptions had to be made in these analyses. Population data were not available for the full

age, sex and ethnicity breakdown, and we have assumed that age distributions within the broad age groups available for LA data are constant. However, this should not impact adversely on our estimates of ethnicity and age-specific risks, as violation of this assumption would principally affect the estimation of regional differences. Our attempts to estimate all factors within one model were hampered by different levels of data granularity, and required us to examine risk factors within two different models. IMD in particular must be examined at a very local level as neighbourhoods with highly variable deprivation levels may be closely mixed, particularly in areas such as inner London [20]. In the absence of a fine-level breakdown of ethnicity and deprivation, it was not possible to disentangle the effects of inherent cultural differences between ethnic groups, and other factors such as deprivation and area type, which may well be correlated [26]. A potential limitation of this study is that the IMD is classified at a geographical level so there is the possibility of ecological bias, although IMD data is at the finest level (LSOA). Using the Onomap software package to classify patients into different ethnic groups may bring in some misclassification errors, although it is considered a standardized methodology for classification of ethnicity [18]. This ethnicity classification method is based on the patient's name, and if the maternal side is non-White but the paternal side is White, the name of the mixed-race status of the next generation may not be correctly classified using this method, hence a degree of caution should be exercised when interpreting the results.

Another limitation of this study is that a lack of denominator data on underlying medical conditions in the population means that we could not account for their contributions to mortality within our models; and underlying medical conditions would certainly play a major role. Nevertheless, the shift in patterns from 2009/2010 to 2010/2011 shows that there was increased risk for non-White ethnicities in the pandemic period compared to the post-pandemic period; regardless of potential differences in risk due to underlying conditions, which would have been unlikely to change in the general population. In conclusion, an increased mortality impact was associated with influenza A(H1N1)pdm09 during the 2009/2010 pandemic period in non-White ethnic minorities (especially in those of certain south Asian groups): an observation that disappeared in the first post-pandemic season. This suggests these groups were among the first affected by this novel virus, rather than necessarily inherently higher case-severity ratios and reflect initial transmission patterns. In addition elevated risk was also seen for those residing in areas with higher levels of deprivation in England in both the pandemic and first post-pandemic period. These results emphasize the importance of ensuring that influenza control and prevention measures are in place for at-risk and vulnerable populations both for seasonal influenza and when a novel influenza virus emerges. Further studies are warranted to further investigate the reasons for these differences in impact.

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DECLARATION OF INTEREST

None.

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