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1 **Blood pressure change across pregnancy in white British and Pakistani women: analysis of**
2 **data from the Born in Bradford cohort**

3
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19

20 **Short title:** Maternal blood pressure change in white British and Pakistani women

21 **Abstract**

22 The incidence of gestational hypertension (GH) and pre-eclampsia (PE) is increasing. Use of
23 blood pressure (BP) change patterns may improve early detection of BP abnormalities. We
24 used Linear spline random-effects models to estimate BP patterns across pregnancy for
25 white British and Pakistani women. Pakistani women compared to white British women had
26 lower BP during the first two trimesters of pregnancy, irrespective of the development of
27 GH or PE or presence of a risk factor. Pakistani compared to white British women with GH
28 and PE showed steeper BP increases towards the end of pregnancy. Pakistani women were
29 half as likely to develop GH, but as likely to develop PE than white British women. To
30 conclude; BP trajectories differ by ethnicity. Because GH developed evenly from 20 weeks
31 gestation, and PE occurred more commonly after 36 weeks in both ethnic groups, the lower
32 BP up to the third trimester in Pakistani women resulted in a lower GH rate, whereas PE
33 rates, influenced by the steep third trimester BP increase were similar. Criteria for
34 diagnosing GH and PE may benefit from considering ethnic differences in BP change across
35 pregnancy.

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43 Introduction

44 Ten percent of pregnant women will develop gestational hypertension (GH) or pre-
45 eclampsia (PE)¹⁻⁴. The incidence of these conditions is increasing and influenced by
46 population characteristics, in particular the rise in overweight and obesity^{5,6}. GH and PE are
47 major causes of maternal and infant perinatal morbidity and mortality⁷⁻⁹. There is also
48 growing evidence that the longer-term cardiovascular health of the mother and offspring
49 exposed to GH or PE may be adversely affected^{10,11}. Consequently the burden to the
50 individual and to health services is growing¹²⁻¹⁴. Early detection and treatment of pregnancy
51 hypertension can reduce risks¹⁵⁻¹⁷, but this early detection is somewhat hampered by the
52 diagnosis of GH or PE being defined as hypertension after 20 weeks of gestation (i.e. after
53 the nadir of early pregnancy decline in BP reported in many previous studies)¹⁸⁻²⁰.

54 Hypertensive disorders of pregnancy (HDP) which includes GH and PE, represents a
55 spectrum of disorders, all of which are characterised by high diastolic (DBP) or systolic (SBP)
56 blood pressure (BP)²¹. Elevated BP after 20 weeks in women without a history of
57 hypertension is defined as GH; when it occurs with clinically significant levels of proteinuria
58 it is PE^{21,22}. The offspring of women who develop PE, more so than those developing GH,
59 are at greater risk of adverse outcomes including preterm birth and small for gestational
60 age²³. Normal pregnancy is characterised by decreasing levels of BP from conception to 20
61 weeks then a slight and steady increase thereafter to term²⁰. Elevated BP after 20 weeks in
62 all cases of HDP can be reached by different patterns of gestational BP change, including
63 higher than average levels from conception, a smaller decline in BP in early pregnancy
64 and/or a more rapid or pronounced rise following this early pregnancy decline. Whether
65 different patterns of reaching the criteria for GH and PE are helpful in predicting more

66 adverse outcomes, or what risk factors influence them is unclear and this information may
67 be important when considering diagnostic thresholds and treatment of GH and PE in
68 different ethnic groups.

69 We have shown previously that in white European women without GH or PE, mean BP at
70 the start of pregnancy, the rate of decline in early pregnancy and the steepness of the
71 increase in late pregnancy, differs by established risk factors (age, parity, BMI, multiple
72 pregnancy) in the same direction that those risk factors are associated with HDP^{18,24}. We
73 have further shown that early BP change can be used to predict which women are likely to
74 experience a HDP, and proposed gestational BP centile charts for use in white European
75 women^{25,26}.

76 South Asians, in comparison to white Europeans, have higher levels of adiposity for a given
77 body mass from birth, and are at increased risk of insulin resistance, diabetes and coronary
78 heart disease²⁷⁻³⁰. In pregnancy this is reflected in higher rates of gestational diabetes and
79 mean levels of fasting and post-load glucose^{29,31}. Greater adiposity and gestational diabetes
80 are both associated with an increased risk of a HDP^{23,32,33}, therefore we might expect HDP to
81 be more common in south Asian women. However, the small number of studies that have
82 compared the incidence of HDP between south Asian and white Europeans have found
83 either a lower or similar incidence³⁴⁻³⁷. To our knowledge few previous studies have
84 examined ethnic differences in SBP and DBP change across pregnancy. Those that have
85 include few women from ethnic groups other than white European^{38,39}.

86 The aims of this study were to explore whether: (i) patterns of BP change in pregnancy
87 differed between white British and Pakistani women; (ii) any differences in BP change
88 between these two ethnic groups were related to differences in GH and PE incidence and

89 whether (iii) established risk factors (maternal age, BMI and parity) for GH and PE differed
90 between the ethnic groups in how they relate to BP trajectories and GH and PE incidence.
91 By addressing these aims we hope to better understand the mechanisms that might lead to
92 ethnic differences in HDP incidence and to provide the foundations for exploring whether
93 different thresholds for diagnosing GH and PE between these different ethnic groups would
94 improve their detection, management and outcomes.

95 **Methods**

96 *Participants*

97 Our analyses were undertaken in two ethnic groups – white British and Pakistani women,
98 because these represent the largest ethnic groups in our cohort study. Data from women
99 participating in the Born in Bradford (BiB) cohort were used. BiB is a prospective birth
100 cohort study including 12,450 women who booked to deliver at the Bradford Royal
101 Infirmary, Bradford, UK, between 2007 and 2011. The cohort is broadly representative of
102 the obstetric population in Bradford and includes equal numbers of Pakistani and white
103 British women. The study methods have been described in full elsewhere⁴⁰. At recruitment,
104 participants gave informed written consent, height and weight was measured, a
105 questionnaire was administered by trained research assistants and information was
106 abstracted from medical records, informed written consent was also given by participants
107 for the future abstraction of information from medical records. Interviews were conducted
108 in English or in south Asian languages (including Urdu and Mirpuri). Research Ethics
109 Committee approval was obtained (07/H1302/112), all research was performed in
110 accordance with relevant guidelines and regulations. Women were excluded if they did not
111 complete a baseline questionnaire, had one or less BP measurement recorded antenatally

112 or did not have their birth outcomes recorded. Following further exclusions (women with
113 pre-existing hypertension, multiple pregnancies or 'other' ethnicities (than white British or
114 Pakistani)), 8212 women were included in these analyses. Figure 1 shows the full inclusion
115 and exclusion criteria of women from BiB for this study.

116 *Outcomes*

117 All BP and proteinuria measurements that were recorded as part of routine antenatal care
118 were abstracted from each participant's medical records (i.e. each woman would have
119 several repeat measurements of SBP, DBP and proteinuria).

120 These repeat measurements were conducted by midwives, doctors or health care assistants
121 working in the community or hospital clinics, who were trained to measure BP in the seated
122 position using the appropriate cuff size, diastolic BP (DBP) was measured using Korotkoff
123 phase V. Most measurements were scheduled according to UK clinical guideline
124 recommendations (at 10, 16, 28, 34, 36, 38, 41 weeks gestation and additionally at 25, 31
125 and 40 weeks gestation for women during their first pregnancy)⁴¹. Women who were
126 deemed to require additional monitoring by their clinical team or who self-referred for
127 various reasons (for example abdominal pain or reduced fetal movements) had
128 measurements recorded at additional times. A variety of devices were used to measure BP
129 and proteinuria, all devices were calibrated according to standard UK health service
130 practice²¹. The gestational age on the date of each measurement was derived from the
131 infant's gestational age at birth and date of measurement, gestational age at birth was
132 derived from the 10 week ultrasound scan and date of birth.

133 The extraction of BP and proteinuria measurements from paper medical records was
134 undertaken by four trained research assistants. Accuracy of abstractions was checked on 5%

135 of records for all abstracted data; all error rates were less than 1%. The BP and proteinuria
136 repeat measurements were used to derive gestational BP change trajectories across
137 pregnancy and also to define GH and PE in a systematic way for all participants. GH was
138 defined as new-onset hypertension after 20 weeks gestation; DBP equal to or exceeding
139 90mmHg and/or SBP equal to or exceeding 140mmHg on at least two separate occasions. PE
140 was defined as for GH, but also with concurrent (with the elevated blood pressure) clinically
141 significant proteinuria; greater or equal to 1 '+' on the reagent strip reading (the equivalent
142 of ~30mg/mmol [0.3mg/mg] on spot urine protein/creatinine ratio)^{21,22}.

143 *Ethnicity, HDP risk factors and other measurements*

144 The woman's ethnicity, age, education, parity and smoking during pregnancy were obtained
145 from interviewer administered questionnaires at recruitment. Ethnicity was categorised as
146 white British and Pakistani according to UK Office of National Statistics criteria⁴². Education
147 was equivalized to UK standard attainments and participants were included in one of five
148 mutually exclusive categories (<5 GCSE equivalent, +5 GCSE equivalent, A level equivalent,
149 higher, other)²⁹. Family history of diabetes and hypertension was abstracted from antenatal
150 clinical paper records as described above for the repeat BP and proteinuria measures.

151 Maternal weight as recorded at the first antenatal clinic visit, glucose levels (at the 26-28
152 week gestation oral glucose tolerance test) and infant sex⁴³ were obtained from hospital
153 electronic records. Gestational diabetes was defined according to modified World Health
154 Organization (WHO) criteria operating at the time of recruitment (either fasting glucose ≥ 6.1
155 mmol/L, or 2 h post-load glucose ≥ 7.8 mmol/L)^{29,44}.

156 Body mass index (BMI kg/m²) was calculated using height from the recruitment assessment
157 and the first antenatal clinic visit weight, which was generally before 12 weeks gestation, as

158 that would be little influenced by pregnancy or fetal size in comparison to the measure at
159 recruitment (around 28 weeks). BMI was categorised according to WHO definitions of
160 underweight or normal (≤ 24.9 kg/m²; there being too few women to categorize
161 underweight separately), overweight (≥ 25.0 – 29.9 kg/m²) and obese (≥ 30.0 kg/m²)⁴⁵.
162 Smoking during pregnancy which was self-reported and categorized as yes or no.

163 *Statistical analysis*

164 Linear spline random-effects models were used to derive SBP and DBP change trajectories
165 across gestation, as previously described^{18,24}. Multilevel modelling (using antenatal visit as
166 level 1 and woman as level 2) was conducted to account for multiple antenatal
167 measurements and clustering (non-independence) of these within each woman. The
168 median, interquartile range, and full range of the number of BP measurements per woman
169 were 10, 8-14 and 1-142 respectively. In order to prevent the small number of women with
170 many repeat measurements overly influencing results we split gestation into two week
171 periods and for any woman who had more than one measure in any of these two-week
172 periods, one measurement was selected at random to remain in the analyses. This was
173 achieved by creating a random number for each record using the `invnorm(uniform)`
174 command in Stata, sorting data by participant identifier, gestation period and random
175 number, then choosing the first record within each gestation period. Following this, there
176 was a median of 9 BP measurements per woman (interquartile range, 8-10 and full range 2-
177 16).

178 The best fitting model was derived using fractional polynomials; we examined these in the
179 whole cohort and for the two ethnic groups, with the appearance of the average cohort
180 trajectory from this modelling used to explore potential knot points. Knot points are the

181 gestational ages at which a change in the direction or magnitude of BP trajectory slopes
182 occurs. We examined the five highest log-likelihoods for SBP and DBP models separately, in
183 the whole cohort and also separately for white British and Pakistani women. Our aim was to
184 identify knot points that were a good reflection (fit) of the observed data and if possible
185 have similar knot points for both SBP and DBP and for both ethnic groups in order to aid
186 comparisons. We set eight weeks gestation as the baseline (intercept) as there were too few
187 measurements before that time to produce meaningful results. The modelling created five
188 measurements of SBP and DBP change for each woman: baseline level at 8 weeks
189 (intercept) and change between 8 and 24, 24 and 30, 30 and 36 weeks and 36 weeks to
190 delivery.

191 Women with PE have a high risk of preterm delivery, particularly if maternal BP rises rapidly,
192 and earlier delivery may lead to fewer measurements contributing to the models and thus a
193 lower influence from pregnancies with a steeper increase in BP, so underestimating the
194 slope. In order to adjust for this, we combined the random-effects spline models for BP
195 change with length of gestation to produce a joint model¹⁸. Individual trajectories and
196 trajectories by categorical covariates were obtained from random-intercept and -slope
197 models, which provide a measure of each individual's deviation from the whole cohort
198 mean BP at 8-weeks and from the average slope in each period of gestation. The following
199 covariates were explored in these models: HDP (normotensive (reference group), GH, PE);
200 age (< 25 (reference) 25-29, 30-35, >35); BMI (underweight/normal (reference), overweight,
201 obese); ethnicity (white British (reference), Pakistani); parity (nulliparous (reference),
202 multiparous); smoking in pregnancy (no (reference), yes); gestational diabetes (no

203 (reference), yes); education (<5 GCSE equivalent (reference), 5+ GCSE equivalent, A level
204 equivalent, higher, other) and offspring sex (male (reference), female).

205 The associations between BP changes and time to delivery were derived from the random
206 effects for BP outcomes and the residual error from the length of gestation. Our initial
207 model (model 1) adjusted for all covariates (described above). In model 2, we additionally
208 adjusted each slope (mean change in BP per gestational week) for the mean BP at 8 weeks,
209 and each preceding slope(s), so that results were not confounded by the initial BP measure
210 and earlier slope(s) which correlate with subsequent slopes^{18,24}.

211 **Results**

212 Table 1 shows the distribution of characteristics of all women and by ethnic group; 4345
213 were Pakistani and 3867 white British. Compared to white British women, Pakistani women
214 were older, were more likely to have attained a higher educational level, to be multiparous,
215 less likely to be obese or to have smoked in pregnancy and more likely to have developed
216 gestational diabetes in the current pregnancy and to have a family history of diabetes or
217 hypertension.

218 Approximately twice as many white British than Pakistani women developed GH (11 % vs
219 5%), whereas similar proportions of white British and Pakistani women developed PE (3% vs
220 2%). Of those who developed GH in both ethnic groups approximately half developed it
221 between 20 to 36 weeks of gestation and half at or after 36 weeks, whereas most cases of
222 PE in both groups developed at or after 36 weeks (see Table 2).

223 Table 3 shows the unadjusted associations between risk factors and GH and PE by ethnicity.
224 Generally, point estimates tended to be closer to the null for Pakistani women than for
225 white British women.

226 Figures 2a to 2f show the time to delivery adjusted trajectories (i.e. joint model) of SBP and
227 DBP across pregnancy for Pakistani and white British women who were normotensive (see
228 Fig. 2a and 2b), and who developed GH (see Fig. 2c and 2d) or PE (see Fig. 2e and 2f). Tables
229 4 and 5 show the corresponding mean differences in SBP and DBP respectively at 8 weeks
230 and the change in BP at each subsequent period. Pakistani women who remained
231 normotensive started pregnancy with lower SBP (-4.00, 95% CI: -4.71 to -3.29) and DBP (-
232 1.92, 95% CI: -2.44 to -1.40) and demonstrated lower BP trajectories across pregnancy
233 compared to white British women.

234 There was no evidence of an early pregnancy decline in SBP or DBP in normotensive women
235 for either ethnic group, with a very slight increase in BP in both groups across pregnancy up
236 to 24 weeks, after which the magnitude of rate of change increased (see Fig. 2a and 2b and
237 Tables 4 and 5).

238 As with normotensive Pakistani women, Pakistani women who developed GH had lower SBP
239 (-4.93, 95% CI: -7.74 to -2.12) and DBP (-1.58, 95% CI: -3.60 to 0.44) at the start of pregnancy
240 than white British women who developed this condition, and continued with lower levels
241 until 36 weeks. From 36 weeks onwards, a steeper increase in SBP occurred than the
242 increase demonstrated by white British women, such that by delivery Pakistani women with
243 GH had similar SBP and DBP to white British women with GH (see Fig. 2c and 2d and Tables
244 4 and 5).

245 Pakistani women who developed PE began pregnancy with lower SBP (-2.58, 95% CI: -7.16
246 to 2.00) but similar DBP (0.48, 95% CI: -2.93 to 3.88) than white British women who
247 developed this condition. Pakistani women demonstrated a steeper increase in BP from 30
248 weeks gestation than white British women. (see Fig. 2e and 2f). The steepness of the
249 increase continued to term, which meant that by delivery, Pakistani women's SBP and DBP
250 were similar to those of white British women. White British women who developed PE
251 showed a decline in BP from 24 weeks, their BP reached a nadir at 30 weeks, after which
252 time both SBP and DBP increased until term. Pakistani women with PE did not show a
253 similar decline in BP.

254 Supplementary Figures 1a to 7b show the adjusted (Model 2) average shaped SBP
255 trajectories and Supplementary Figures 8a to 14b the adjusted (Model 2) average shaped
256 DBP trajectories for Pakistani and white British women by HDP category and maternal risk
257 factor. These trajectories were consistent with what we would expect from patterns seen
258 with associations of each risk factor with each HDP. Supplementary Tables 3 and 4 show the
259 adjusted mean difference (with 95% CI) in SBP and DBP at eight weeks and change in SBP
260 and DBP in each period of gestation by GH and PE for each ethnic group.

261 Table 6 shows the associations of BP at eight weeks and changes in BP with time to delivery.
262 SBP and DBP at 8 weeks gestation and BP change in any period of pregnancy was not
263 associated with gestational length in either ethnic group, with the exception of SBP, but not
264 DBP change between 8 and 24 weeks which was weakly associated with a reduction in
265 gestational length for white British women only.

266 Supplementary Table 3 shows similar predicted lengths of gestation at 2 SDs above and
267 below the average SBP and DBP at 8 weeks and changes in SBP and DBP in each period of
268 gestation for Pakistani and white British women.

269 **Discussion**

270 We found that Pakistani women start pregnancy with lower SBP and DBP than white British
271 women. This is true for women who remain normotensive throughout their pregnancies and
272 for those who go on to develop GH or PE. Generally, normotensive Pakistani and white
273 British women, show gradual increases in BP until 36 weeks when SBP and DBP for both
274 ethnic groups increases more steeply until term. Compared with white British women,
275 Pakistani women who developed either GH or PE had a steeper rise in BP in the third
276 trimester and this difference in BP trajectories influenced the comparison of HDP between
277 the two groups. GH was more common in white British women due to: (a) the combination
278 of the same diagnostic criteria being used across ethnicities, (b) Pakistani women having
279 lower BP levels until the final trimester of pregnancy, and (c) women in both ethnic groups
280 who developed GH doing so evenly between 20-36 and after 36 weeks gestation. By
281 contrast, the proportion with PE was similar in both ethnic groups, because in both groups
282 those developing PE were much more likely to do so after 36 weeks, which meant that the
283 steeper third trimester rise in BP in Pakistani women had a greater impact on their PE rate
284 than their GH rate.

285 A number of previous studies have reported ethnic differences in the occurrence of GH and
286 PE and the association of risk factors with GH and PE and adverse outcomes for different
287 ethnic groups^{36,46,47} and one study found similar rates of PE in South Asian and non-Hispanic
288 white Americans³⁶. However, few previous studies have examined ethnic differences in BP

289 trajectories across pregnancy³⁸. A recent study compared Norwegian women with women
290 from eastern Europe, middle east, Africa and south and east Asia, however only 200 south
291 Asian women were included and BP was recorded just twice antenatally³⁹.

292 The lower rate of GH in Pakistani compared with white British women in our study is not
293 surprising given the lower SBP and DBP of Pakistani women at the start of pregnancy and
294 between 20 and 36 weeks gestation, and that criteria for the diagnosis of HDP are the same
295 in both ethnic groups²².

296 Our findings provide some evidence for considering the rate of change or trajectory patterns
297 of gestational BP, particularly for understanding ethnic differences in rates of HDP. The use
298 of BP change across pregnancy (usually an increase of >15mmHg or more from baseline
299 reading) to identify women at increased risk of an adverse outcome has been explored for
300 over 30 years⁴⁸. However inconsistencies in study methods and their reported findings,
301 together with the complexity of incorporating such measures into clinical guidelines and
302 practice has so far prevented its adoption^{49,50}.

303 We did not show a decline in BP from early pregnancy to 18/20 weeks that previous studies
304 found^{18,38,51}. This is unlikely to reflect a lack of measurements at 18 to 24 weeks because
305 antenatal measurements were obtained at regular intervals across pregnancy and this did
306 not change over the period of the study^{22,41}. The transitory decline in BP in early pregnancy
307 is thought to reflect the early haemodynamic changes of pregnancy and its end point
308 (around 20 weeks) is used as the marker after which GH and PE are diagnosed. Our finding
309 of a gradual increase in BP in the first two trimesters of pregnancy for normotensive women
310 and women with GH and PE (with the exception of white British women with PE, who
311 showed a decline from 24 to 30 weeks), in both ethnic groups, may reflect the changing

312 characteristics of pregnant women over time and the greater prevalence of risk factors for
313 HDP¹⁹. For example, the proportion of women who were overweight and obese included in
314 our previous UK cohort which recruited women in pregnancy in 1990 were 15% and 6%,
315 respectively^{18,52}; in this study, which recruited women 20 years later and from an area of the
316 UK with more deprivation, 29% were overweight and 22% obese⁴⁰.

317 Our study is large with many repeated measurements, which allowed a consistent approach
318 to the classification of GH and PE. Use of routinely collected data will introduce a degree of
319 variability, for example use of different BP measurement devices or use of different sized
320 arm cuffs, however all measurements were collected in accordance with clinical care at the
321 time of recruitment and accurately reflect the measurements used to inform clinical
322 decisions related to the diagnosis and treatment of HDP in our study population.

323 The most recent International Society for the Study of Hypertension in pregnancy (ISSHP)
324 guidance defines PE as high BP on at least two occasions after 20 weeks with concurrent
325 significant proteinuria defined as 2+ on a reagent strip²¹, the UK NICE do not comment on
326 how many + are appropriate²². Our use of 1+ proteinuria on reagent strip in defining PE
327 reflects the ISSHP criteria for diagnosing PE and what was used in clinical practice in the UK
328 at the time of our study (which preceded the most recent ISSHP guidelines)⁵³.

329 Compared with the most recent ISSHP criteria our use of 1+ proteinuria may have increased
330 the overall prevalence of PE in our study. However, this will not have altered the ethnic
331 differences in BP trajectories nor the similarity in PE between white British and Pakistani
332 women, which was driven by the steeper increase in BP in Pakistani women. Some of the
333 weaker associations of risk factors with PE compared with GH shown in Table 3 may be
334 because of a lower proteinuria threshold, but given the relatively small numbers with PE and

335 imprecise estimates with wide confidence intervals, we would not be able to distinguish
336 differences in associations with a stricter definition of proteinuria, as the number with PE
337 would be smaller.

338 Other measures of proteinuria, for example protein-creatinine ratio, may more accurately
339 estimate proteinuria, but they were not consistently recorded at each antenatal visit.

340 Indicators of severe PE, such as evidence of multi-organ involvement were unavailable in
341 our study, so we cannot assess severe PE or eclampsia.

342 In conclusion, BP trajectories differ by ethnicity. Pakistani women had lower BP at the start
343 of pregnancy and during the first two trimesters, irrespective of the development of a HDP
344 or the presence of a risk factor. Compared to white British women, Pakistani women who
345 developed GH or PE showed steeper increases in BP towards the end of pregnancy. Because
346 GH developed evenly between 20-36 weeks and after 36 weeks gestation and PE occurred
347 more commonly after 36 weeks in both ethnic groups, lower BP up to the third trimester in
348 Pakistani women resulted in a considerably lower GH rate, whereas PE rate, influence by
349 the steep third trimester BP increase is similar in the two groups. Criteria for diagnosing
350 HDP may benefit from considering ethnic differences in BP change across pregnancy.

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356 **Contribution:** DF and DAL designed this study, with input from other co-authors. DF
357 collected the BP measurements, DF, JW, DT, and DAL were involved in the design and data
358 collection of the Born in Bradford study. DF, CM-W, GS, TAS and DAL developed the analysis
359 protocol and GS, CM-W and DF performed the analyses. DF wrote the first draft of the paper
360 with initial further iterations involving all remaining authors and with all authors
361 contributing to the final submitted versions. All authors approved the final version.

362

363 **Ethics approval:** Ethics approval was granted by the Bradford National Health Service
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365

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382

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384 International governments and charitable funders, Medtronic and Roche Diagnostics for
385 research unrelated to that presented here. The other authors declare no competing
386 interests.

387

388 **Data availability statement:** The Born in Bradford Study allows bone fide researchers to
389 access data. Full details of how to do this are provided on the study web pages
390 (<https://borninbradford.nhs.uk/research/how-to-access-data/>).

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393 **References**

- 394 1 Abalos, E., Cuesta, C., Grosso, A. L., Chou, D. & Say, L. Global and regional estimates of
395 preeclampsia and eclampsia: a systematic review. *Eur J Obstet Gynecol Repro Biol* 170, 1-7
396 (2013).
- 397 2 Buchbinder, A. *et al.* Adverse perinatal outcomes are significantly higher in severe
398 gestational hypertension than in mild preeclampsia. *Am J Obstet Gynecol* 186, 66-71 (2002).
- 399 3 Hauth, J. C. *et al.* Pregnancy outcomes in healthy nulliparas who developed hypertension.
400 Calcium for Preeclampsia Prevention Study Group. *Obstet Gynecol* 95, 24-28 (2000).
- 401 4 Muti, M., Tshimanga, M., Notion, G. T., Bangure, D. & Chonzi, P. Prevalence of pregnancy
402 induced hypertension and pregnancy outcomes among women seeking maternity services in
403 Harare, Zimbabwe. *BMC Cardiovasc Disord* 15, 111 (2015).
- 404 5 Duckitt, K. & Harrington, D. Risk factors for pre-eclampsia at antenatal booking: systematic
405 review of controlled studies. *BMJ* 330, 565 (2005).
- 406 6 Hutcheon, J. A., Lisonkova, S. & Joseph, K. S. Epidemiology of pre-eclampsia and the other
407 hypertensive disorders of pregnancy. *Best Pract Res Clin Obstet Gynaecol* 25, 391-403,
408 doi:<http://dx.doi.org/10.1016/j.bpobgyn.2011.01.006> (2011).
- 409 7 Centre for Maternal and Child Enquiries (CMACE). Saving Mothers' Lives: reviewing maternal
410 deaths to make motherhood safer: 2006–08. The Eighth Report on Confidential Enquiries
411 into Maternal Deaths in the United Kingdom. *BJOG*. 118, 1-203 (2011).
- 412 8 Khan, K. S., Wojdyla, D., Say, L., Gülmezoglu, A. M. & Van Look, P. F. A. WHO analysis of
413 causes of maternal death: a systematic review. *Lancet* 367, 1066-1074,
414 doi:[http://dx.doi.org/10.1016/S0140-6736\(06\)68397-9](http://dx.doi.org/10.1016/S0140-6736(06)68397-9) (2006).
- 415 9 Bramham, K. *et al.* Association of Proteinuria Threshold in Pre-Eclampsia with Maternal and
416 Perinatal Outcomes: A Nested Case Control Cohort of High Risk Women. *PloS one* 8, e76083,
417 doi:10.1371/journal.pone.0076083 (2013).

- 418 10 Tranquilli, A. L., Landi, B., Giannubilo, S. R. & Sibai, B. M. Preeclampsia: No longer solely a
419 pregnancy disease. *Pregnancy Hypertens* 2, 350-357,
420 doi:<http://dx.doi.org/10.1016/j.preghy.2012.05.006> (2012).
- 421 11 Ayansina, D. *et al.* Long term effects of gestational hypertension and pre-eclampsia on
422 kidney function: Record linkage study. *Pregnancy Hypertens* 6, 344-349 (2016).
- 423 12 Ahmed, R. J. *et al.* The Cost Implications of Less Tight Versus Tight Control of Hypertension in
424 Pregnancy (CHIPS Trial). *Hypertension* 68, 1049-1055 (2016).
- 425 13 Mistry, H., Dowie, R., Franklin, R. C. G. & Jani, B. R. Costs of neonatal care for low-
426 birthweight babies in English hospitals. *Acta Pædiatr* 98, 1123-1129 (2009).
- 427 14 National Institute for Health and Care Excellence. Hypertension in pregnancy. Costing report.
428 Implementing NICE guidance. *NICE clinical guideline 107* (2010).
- 429 15 Cluver, C., Novikova, N., Koopmans, C. M. & West, H. M. Planned early delivery versus
430 expectant management for hypertensive disorders from 34 weeks gestation to term.
431 *Cochrane Database Syst Revs*, doi:10.1002/14651858.CD009273.pub2 (2017).
- 432 16 Duley, L., Meher, S. & Jones, L. Drugs for treatment of very high blood pressure during
433 pregnancy. *Cochrane Database Syst Revis*, doi:10.1002/14651858.CD001449.pub3 (2013).
- 434 17 Duley, L., Gülmezoglu, A. M., Henderson-Smart, D. J. & Chou, D. Magnesium sulphate and
435 other anticonvulsants for women with pre-eclampsia. *Cochrane Database Syst Revs*,
436 doi:10.1002/14651858.CD000025.pub2 (2010).
- 437 18 Macdonald-Wallis, C. *et al.* Blood pressure change in normotensive, gestational
438 hypertensive, preeclamptic, and essential hypertensive pregnancies. *Hypertension* 59, 1241-
439 1248 (2012).
- 440 19 Magriples, U. *et al.* Blood pressure changes during pregnancy: impact of race, body mass
441 index, and weight gain. *Am J Perinatol* 30, 415-424, doi:10.1055/s-0032-1326987 (2013).

- 442 20 Reiss, R. E., Oshaughnessy, R. W., Quilligan, T. J. & Zuspan, F. P. Retrospective comparison of
443 blood-pressure course during preeclamptic and matched control pregnancies. *Am J Obstet*
444 *Gynecol* 156, 894–898 (1987).
- 445 21 Tranquilli, A. L. *et al.* The classification, diagnosis and management of the hypertensive
446 disorders of pregnancy: A revised statement from the ISSHP. *Pregnancy Hypertens* 4, 97-104
447 (2014).
- 448 22 National Institute for Health and Care Excellence. Hypertension in pregnancy: The
449 management of hypertensive disorders during pregnancy. *NICE clinical guideline 107* (2010).
- 450 23 Mol, B. W. J. *et al.* Pre-eclampsia. *Lancet* 387, 999-1011 (2016).
- 451 24 Macdonald-Wallis, C., Tilling, K., Fraser, A., Nelson, S. M. & Lawlor, D. A. Established
452 preeclampsia risk factors are related to patterns of blood pressure change in normal term
453 pregnancy: findings from the Avon Longitudinal Study of Parents and Children. *J Hypertens*
454 29, 1703-1711 (2011).
- 455 25 Macdonald-Wallis, C. *et al.* Gestational-age-specific reference ranges for blood pressure in
456 pregnancy: findings from a prospective cohort. *J hypertens* 33, 96-105 (2015).
- 457 26 Macdonald-Wallis, C. *et al.* Antenatal blood pressure for prediction of pre-eclampsia,
458 preterm birth, and small for gestational age babies: development and validation in two
459 general population cohorts. *BMJ* 351 (2015).
- 460 27 British Heart Foundation Health Promotion Research Group. Ethnic differences in
461 Cardiovascular disease *Department of Public Health* 10th edition (2010).
- 462 28 McKeigue, P. M., Pierpoint, T., Ferrie, J. E. & Marmot, M. G. Relationship of glucose
463 intolerance and hyperinsulinaemia to body fat pattern in South Asians and Europeans.
464 *Diabetologia* 35, 785-791 (1992).
- 465 29 Lawlor, D. A. *et al.* Pregnancy glycaemia and cord-blood levels of insulin and leptin in
466 Pakistani and white British mother–offspring pairs: findings from a prospective pregnancy
467 cohort. *Diabetologia* 57, 2492-2500 (2014).

- 468 30 West, J. *et al.* UK-born Pakistani-origin infants are relatively more adipose than White British
469 infants: findings from 8704 mother-offspring pairs in the Born-in-Bradford prospective birth
470 cohort. *J Epidemiol Community Health* 67, 544-551 (2013).
- 471 31 Farrar, D. *et al.* Association between hyperglycaemia and adverse perinatal outcomes in
472 south Asian and white British women: analysis of data from the Born in Bradford cohort.
473 *Lancet Diabetes Endocrinol* 3, 795-804 (2015).
- 474 32 Poston, L. *et al.* Preconceptional and maternal obesity: epidemiology and health
475 consequences. *Lancet Diabetes Endocrinol* 4, 1025-1036 (2016).
- 476 33 Weissgerber, T. L. & Mudd, L. M. Preeclampsia and Diabetes. *Curr Diab Rep* 15, 9 (2015).
- 477 34 Naimy, Z., Grytten, J., Monkerud, L. & Eskild, A. The prevalence of pre-eclampsia in migrant
478 relative to native Norwegian women: a population-based study. *BJOG* 122, 859-865 (2015).
- 479 35 Caughey, A. B., Stotland, N. E., Washington, A. E. & Escobar, G. J. Maternal Ethnicity,
480 Paternal Ethnicity, and Parental Ethnic Discordance: Predictors of Preeclampsia. *Obstet*
481 *Gynecol* 106, 156-161 (2005).
- 482 36 Gong, J., Savitz, D. A., Stein, C. R. & Engel, S. M. Maternal ethnicity and pre-eclampsia in New
483 York City, 1995–2003. *Paediatr Perinat Epidemiol* 26, 45-52 (2012).
- 484 37 Ghosh, G. *et al.* Racial/ethnic differences in pregnancy-related hypertensive disease in
485 nulliparous women. *Ethn Dis* 24, 283-289 (2014).
- 486 38 Bouthoorn, S. H. *et al.* Ethnic Differences in Blood Pressure and Hypertensive Complications
487 During Pregnancy: The Generation R Study. *Hypertension* 60, 198-205 (2012).
- 488 39 Waage, C. W. *et al.* Ethnic differences in blood pressure from early pregnancy to
489 postpartum: a Norwegian cohort study. *J Hypertension* 34, 1151-1159 (2016).
- 490 40 Wright, J. *et al.* Cohort profile: The Born in Bradford multi-ethnic family cohort study. *Int J*
491 *Epidemiol* 4, 1–14 (2012).
- 492 41 National Institute for Health and Care Excellence. Antenatal care: Routine care for the
493 healthy pregnant woman. *NICE clinical guideline* 62 (2008).

494 42 Office of National Statistics. Ethnic group statistics: A guide for the collection and
495 classification of ethnicity data. *ethnic-group-statistics_tcm77-186499.pdf*, Last accessed
496 January 2018 (2003).

497 43 Di Renzo, G. C., Rosati, A., Sarti, R. D., Cruciani, L. & Cutuli, A. M. Does fetal sex affect
498 pregnancy outcome? *Gender Med* 4, 19-30 (2007).

499 44 World Health Organization. *Definition, diagnosis and classification of diabetes mellitus and
500 its complications. Report of a WHO consultation. Part 1: diagnosis and classification of
501 diabetes mellitus.* (WHO, 1999).

502 45 World Health Organization. Body Mass Index - BMI. [http://www.euro.who.int/en/health-](http://www.euro.who.int/en/health-topics/disease-prevention/nutrition/a-healthy-lifestyle/body-mass-index-bmi)
503 [topics/disease-prevention/nutrition/a-healthy-lifestyle/body-mass-index-bmi](http://www.euro.who.int/en/health-topics/disease-prevention/nutrition/a-healthy-lifestyle/body-mass-index-bmi),
504 doi:[http://www.euro.who.int/en/health-](http://www.euro.who.int/en/health-topics/disease-prevention/nutrition/a-healthy-lifestyle/body-mass-index-bmi)
505 [lifestyle/body-mass-index-bmi](http://www.euro.who.int/en/health-topics/disease-prevention/nutrition/a-healthy-lifestyle/body-mass-index-bmi) (last accessed January 2018).

506 46 Bryant, A. S., Worjolah, A., Caughey, A. B. & Washington, A. E. Racial/Ethnic Disparities in
507 Obstetrical Outcomes and Care: Prevalence and Determinants. *Am J Obs Gynecol* 202, 335-
508 343 (2010).

509 47 Shahul, S. *et al.* Racial Disparities in Comorbidities, Complications, and Maternal and Fetal
510 Outcomes in Women with Preeclampsia/Eclampsia. *Hypertens Pregnancy* 34, 506-515
511 (2015).

512 48 Redman, C. W. G. & Jefferies, M. Revised definition of pre-eclampsia. *Lancet* 1, 809-812
513 (1988).

514 49 Bullarbo, M. & Rylander, R. Diastolic blood pressure increase is a risk indicator for pre-
515 eclampsia. *Arch Gynecol Obstet* 291, 819-823 (2015).

516 50 Levine, R. J. *et al.* Should the definition of preeclampsia include a rise in diastolic blood
517 pressure of ≥ 15 mm Hg to a level < 90 mm Hg in association with proteinuria? *Am J Obstet*
518 *Gynecol* 183, 787-792 (2000).

519 51 Easterling, T. R., Benedetti, T. J., Schmucker, B. C. & Millard, S. P. Maternal Hemodynamics in
520 Normal and Preeclamptic Pregnancies: A Longitudinal Study. *Obstet Gynecol* 76, 1061-1069
521 (1990).

522 52 Golding, J., Pembrey, M., Jones, R. & The Alspac StudyTeam. ALSPAC–The Avon Longitudinal
523 Study of Parents and Children. *Paediat Perinat Epidemiol* 15, 74-87 (2001).

524 53 Brown, M. A., Lindheimer, M. D., de Swiet, M., Assche, A. V. & Moutquin, J. M. The
525 classification and diagnosis of the hypertensive disorders of pregnancy: statement from the
526 International Society for the Study of Hypertension in Pregnancy (ISSHP). *Hypertens*
527 *Pregnancy* 20 (2001).

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Table 1 Characteristics for all participants and by ethnicity. Values are n (%) unless otherwise indicated

	All women N=8,212	White British women N=3,867	Pakistani women N=4,345	P-value³
Gestational age at delivery, weeks ¹	39 (38-40)	40 (39-40)	39 (38-40)	<0.001
Hypertensive disorder of pregnancy (HDP)				
No HDP	7,376 (90)	3,326 (86)	4,050 (93)	<0.001
Gestational hypertension	622 (8)	428 (11)	194 (5)	
Pre-eclampsia	214 (3)	113 (3)	101 (2)	
Maternal age at infant birth (years)				
<25	2,788 (34)	1,516 (39)	1,272 (29)	<0.001
25-29	2,641 (32)	1,090 (28)	1,551 (36)	
30-34	1,778 (22)	767 (20)	1,011 (23)	
≥35	1,005 (12)	494 (13)	511 (12)	
BMI at pregnancy booking (kg/m ²) ² (mean)	26.2	26.9	25.6	<0.001
Underweight (BMI ≤19)	503 (6)	154 (4)	349 (8)	
Normal (BMI 19-24)	3,482 (42)	1,608 (42)	1,874 (43)	
Overweight (BMI 25-29)	2,446 (30)	1,126 (29)	1,320 (30)	
Obese (BMI ≥30)	1,781 (22)	979 (25)	802 (19)	
Parity:				
Nulliparous	3,291 (40)	1,875 (49)	1,416 (33)	<0.001
Multiparous	4,921 (60)	1,992 (52)	2,929 (67)	
Smoked during pregnancy				
Yes	1,452 (18)	1,302 (34)	150 (4)	<0.001
Highest educational achievement				
<5 GCSE equivalent	1,883 (23)	766 (20)	1,117 (26)	<0.001

	All women N=8,212	White British women N=3,867	Pakistani women N=4,345	P-value³
5+ GCSE equivalent	2,694 (33)	1,332 (35)	1,362 (31)	
A-level equivalent	1,221 (15)	659 (17)	562 (13)	
Higher	1,867 (23)	744 (19)	1,123 (26)	
Other	547 (7)	366 (10)	181 (4)	
Infant gender				
Male	4,212 (51)	1,990 (52)	2,222 (51)	0.771
Female	4,000 (49)	1,877 (49)	2,123 (49)	
Gestational diabetes	660 (8)	191 (5)	469 (11)	<0.001
Family history of diabetes	2,045 (25)	514 (13)	1,531 (35)	<0.001
Family history of hypertension	2,167 (26)	901 (23)	1,266 (29)	<0.001
Pre-existing diabetes	15 (<1)	9 (<1)	6 (<1)	0.316

¹ Median and interquartile range

² Body mass index

³ Difference between white British and Pakistani women using Wilcoxon rank sum test or chi-squared test as appropriate

Table 2 Total numbers developing gestational hypertension at any time in pregnancy: 428 White British and 194 Pakistani women; Total numbers developing preeclampsia at any time in pregnancy:113 White British and 101 Pakistani women.

Hypertensive disorder of pregnancy	Number of weeks of gestation at which each hypertensive disorder of pregnancy developed	
	20-36 weeks	≥36
Gestational hypertension N (%)		
White British	231 (54)	197 (46)
Pakistani	86 (44)	108 (56)
Preeclampsia N (%)		
White British	15 (13)	98 (87)
Pakistani	25 (25)	76 (75)

Table 3 Unadjusted associations of risk factors with hypertensive disorders of pregnancy

Difference in means (continuous variables), Odds ratios (binary) or relative risk ratios (multinomial) [95% CI]

Risk factor	White British women			Pakistani women		
	No HDP (N=3326) Reference	Gestational hypertension (N=428)	Preeclampsia (N=113)	No HDP (N=4050) Reference	Gestational hypertension (N=194)	Preeclampsia (N=101)
BMI (km/m ² , reference <25))						
25 to 29	1	2.15 (1.61, 2.86)	2.01 (1.24, 3.26)	1	1.35 (0.93, 1.96)	1.61 (0.97, 2.66)
30+	1	5.59 (4.31, 7.24)	3.34 (2.10, 5.32)	1	3.56 (2.53, 5.01)	3.77 (2.34, 6.06)
Maternal age (years, reference <25)						
25 to 29	1	1.22 (0.94, 1.58)	1.01 (0.64, 1.60)	1	1.02 (0.71, 1.48)	0.73 (0.45, 1.20)
30 to 34	1	1.30 (0.97, 1.72)	0.94 (0.55, 1.60)	1	1.08 (0.73, 1.59)	0.67 (0.39, 1.16)
35+	1	2.21 (1.65, 2.96)	1.14 (0.63, 2.06)	1	1.32 (0.84, 2.08)	0.92 (0.49, 1.74)
Parity (≥1 versus 0)	1	0.77 (0.63, 0.94)	0.55 (0.38, 0.81)	1	0.54 (0.40, 0.72)	0.40 (0.27, 0.59)
Gestational diabetes (Yes vs No)	1	1.48 (0.98, 2.22)	1.14 (0.49, 2.63)	1	1.12 (0.72, 1.75)	1.02 (0.54, 1.91)
Maternal education (reference 5 GCSE)						
<5 GCSE	1	0.88 (0.65, 1.20)	1.09 (0.66, 1.80)	1	0.96 (0.66, 1.40)	0.76 (0.46, 1.28)
A level	1	1.37 (1.02, 1.83)	0.76 (0.42, 1.39)	1	0.83 (0.51, 1.36)	0.56 (0.27, 1.17)
Degree	1	1.45 (1.10, 1.92)	1.09 (0.66, 1.83)	1	0.93 (0.64, 1.37)	0.79 (0.47, 1.32)
Other	1	1.39 (0.97, 1.98)	0.64 (0.28, 1.44)	1	1.21 (0.61, 2.40)	1.00 (0.39, 2.58)
Smoking in pregnancy (Yes vs No)	1	0.55 (0.44, 0.70)	0.63 (0.41, 0.97)	1	0.72 (0.29, 1.78)	0.55 (0.13, 2.26)
Infant gender (Female vs Male)	1	0.88 (0.72, 1.07)	0.77 (0.52, 1.12)	1	0.86 (0.65, 1.16)	1.20 (0.81, 1.78)

Table 4 Mean difference (95% CI) in SBP at 8 weeks and change in SBP in each period of gestation for each hypertensive disorders of pregnancy

Hypertensive disorder	Mean Difference in SBP at 8 wk, mmHg	Mean difference in average SBP change, mm Hg/wk			
		8-24 weeks	24-30 weeks	30-36 weeks	≥36 weeks
No hypertensive disorder					
White British women	0	0	0	0	0
Pakistani women	-4.00 (-4.71 to -3.29)	0.02 (-0.04 to 0.08)	-0.15 (-0.27 to -0.03)	0.09 (-0.02 to 0.21)	0.01 (-0.19 to 0.20)
Gestational hypertension					
White British women	0	0	0	0	0
Pakistani women	-4.93 (-7.74 to -2.12)	0.15 (-0.09 to 0.38)	-0.40 (-0.91 to 0.11)	-0.15 (-0.37 to 0.67)	0.89 (0.03 to 1.74)
Pre-eclampsia					
White British women	0	0	0	0	0
Pakistani women	-2.58 (-7.16 to 2.00)	-0.11 (-0.50 to 0.27)	0.84 (0.59 to 1.62)	0.32 (-0.63 to 1.27)	-0.06 (-1.89 to 1.77)

Adjusted model adjusted for time to delivery and for maternal age, pregnancy booking BMI, parity, smoking in pregnancy status, education, gestational diabetes and infant gender

In reference category: No hypertensive disorder (white British women) mean SBP at 8 wk (mm Hg) 105.84 (104.32 to 107.37); mean SBP change (mm Hg/wk): 8–24 wk 0.15 (0.03 to 0.28); 24–30 wk 0.20 (-0.05 to 0.45); 30–36 wk 0.13 (-0.12 to 0.38); >36 wk 1.33 (0.93 to 1.73)

In reference category: gestational hypertension (white British women), mean SBP at 8 wk (mm Hg) 108.59 (102.95 to 114.23); mean SBP change (mm Hg/wk): 8–24 wk 0.29 (-0.18 to 0.76); 24–30 wk 0.57 (-0.43 to 1.56); 30–36 wk 0.47 (-0.54 to 1.48); >36 wk 3.25 (1.74 to 4.76)

In reference category: pre-eclampsia (white British women), mean SBP at 8 wk (mm Hg) 112.27 (102.17 to 122.37); mean SBP change (mm Hg/wk): 8–24 wk 0.09 (-0.83 to 1.00); 24–30 wk 0.47

(-1.31 to 2.24); 30–36 wk 1.58 (-0.16 to 3.32); >36 wk 1.13 (-2.25 to 4.52)

Table 5 Mean difference (95% CI) in DBP at 8 weeks and change in DBP in each period of gestation for each hypertensive disorders of pregnancy

Hypertensive disorder	Mean Difference in SBP at 8 wk, mmHg	Mean difference in average DBP change, mmHg/wk			
		8-24 weeks	24-30 weeks	30-36 weeks	≥36 weeks
No hypertensive disorder					
White British women	0	0	0	0	0
Pakistani women	-1.92 (-2.44 to -1.40)	0.04 (0.01 to 0.08)	-0.06 (-0.14 to 0.03)	-0.10 (-0.19 to 0.01)	0.19 (0.04 to 0.34)
Gestational hypertension					
White British women	0	0	0	0	0
Pakistani women	-1.58 (-3.60 to 0.44)	0.12 (-0.05 to 0.29)	-0.21 (-0.59 to 0.16)	-0.04 (-0.35 to 0.43)	0.29 (-0.34 to 0.93)
Pre-eclampsia					
White British women	0	0	0	0	0
Pakistani women	0.48 (-2.93 to 3.88)	-0.16 (-0.45 to 0.13)	0.86 (0.28 to 1.45)	-0.32 (-0.98 to 0.34)	0.77 (0.41 to 1.96)

Adjusted model adjusted for time to delivery and for maternal pregnancy booking BMI, age, parity, smoking in pregnancy status, education, gestational diabetes and infant gender

In reference category: No hypertensive disorder (white British women) mean DBP at 8 wk (mm Hg) 62.31 (61.21 to 63.40); mean DBP change (mm Hg/wk): 8–24 wk 0.02 (-0.08 to 0.11); 24–30 wk 0.25 (0.06 to 0.44); 30–36 wk 0.34 (0.15 to 0.54); >36 wk 1.28 (0.96 to 1.59)

In reference category: gestational hypertension (white British women), mean DBP at 8 wk (mm Hg) 64.84 (60.88 to 68.80); mean DBP change (mm Hg/wk): 8–24 wk 0.02 (-0.31 to 0.36); 24–30 wk 1.07 (0.35 to 1.79); 30–36 wk 0.15 (-0.59 to 0.89); >36 wk 3.04 (1.93 to 4.15)

In reference category: pre-eclampsia (white British women), mean DBP at 8 wk (mm Hg) 69.38 (61.89 to 76.89); mean DBP change (mm Hg/wk): 8–24 wk -0.02 (-0.66 to 0.70); 24–30 wk -0.12

(-1.44 to 1.20); 30–36 wk 1.90 (0.56 to 3.25); >36 wk 2.68 (0.13 to 5.24)

1 Table 6 Associations of blood pressure at 8 weeks gestation and changes in blood pressure in each period of
 2 pregnancy with the time to delivery

Blood pressure variable	Length of gestation			
	White British women		Pakistani women	
	% Increase in gestation	95% CI	% Increase in gestation	95% CI
SBP at 8 weeks, mmHg	0.00	0.00, 0.01	0.00	-0.01, 0.00
SBP change, mmHg/wk				
8-24 wk	-0.26	-0.74, -0.01	-0.07	-0.56, 0.28
24-30 wk	0.09	-1.80, 1.20	-0.02	-2.43, 1.14
30-36 wk	-0.54	-32.55, 2.09	-0.09	-2.15, 1.04
≥ 36 wk	-0.21	-1.18, 0.45	-0.09	-0.28, 0.03
DBP at 8 weeks, mmHg	0.00	0.01, 0.02	0.00	-0.01, 0.00
DBP change, mmHg/wk				
8-24 wk	-0.03	-4495.20, 2.27	-0.38	-2.96, 0.29
24-30 wk	-0.19	-5.51, 1.51	-0.11	-4.51, 1.44
30-36 wk	-0.42	-1.42, 0.32	-0.26	-1.36, 0.53
> 36 wk	-0.08	-0.77, 0.49	-0.04	-0.43, 0.30

3
 4 Model 2 adjusted for HDP category and maternal covariates and for SBP/DBP at 8 weeks' gestation and
 5 SBP/DBP change in BP in earlier periods of pregnancy

6
 7
 8 **Table legends**

9 Table 1 Characteristics for all participants and by ethnicity. Values are n (%) unless otherwise indicated

10

11 Table 2 Total numbers developing gestational hypertension at any time in pregnancy: 428 White British and
 12 194 Pakistani women; Total numbers developing preeclampsia at any time in pregnancy: 113 White British and
 13 101 Pakistani women.

14

15 Table 3 Unadjusted associations of risk factors with hypertensive disorders of pregnancy

16

17 Table 4 Mean difference (95% CI) in SBP at 8 weeks and change in SBP in each period of gestation for each
18 hypertensive disorders of pregnancy

19

20 Table 5 Mean difference (95% CI) in DBP at 8 weeks and change in DBP in each period of gestation for each
21 hypertensive disorders of pregnancy

22

23 Table 6 Associations of blood pressure at 8 weeks gestation and changes in blood pressure in each period of
24 pregnancy with the time to delivery

25

26 **Figure legends**

27 Figure-1 Study flow chart

28

29 Figure-2a and 2b Predicted trajectories of systolic blood and diastolic pressure across pregnancy for Pakistani
30 and white British normotensive women

31

32 Figure-2c and 2d Predicted trajectories of systolic and diastolic blood pressure across pregnancy for Pakistani
33 and white British women with gestational hypertension

34

35 Figure-2e and 2f Predicted trajectories of systolic and diastolic blood pressure across pregnancy for Pakistani
36 and white British women with pre-eclampsia

37 Footnote to figures- 2a to 2f

38 – or + denotes where the mean difference in average BP change in the associated time period between White
39 British and Pakistani women is significantly smaller or larger respectively