

Quantifying the causal effects of 20 mph zones on road casualties in London via doubly robust estimation

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Abstract

This paper estimates the causal effect of 20 mph zones on road casualties in London. Potential confounders in the key relationship of interest are included within outcome regression and propensity score models, and the models are then combined to form a doubly robust estimator. A total of 234 treated zones and 2844 potential control zones are included in the data sample. The propensity score model is used to select a viable control group which has common support in the covariate distributions. We compare the doubly robust estimates with those obtained using three other methods: inverse probability weighting, regression adjustment, and propensity score matching. The results indicate that 20 mph zones have had a significant causal impact on road casualty reduction in both absolute and proportional terms.

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Keywords: 20 mph zones, Doubly Robust Methods, Causal Analysis

1 **1. Introduction**

2 It is widely thought that a reduction in vehicle speeds can reduce the severity
3 of road casualties and decrease the number of traffic collisions (Soole et al., 2013;
4 Elvik et al., 2004; Elvik, 2009). There are a number of policy interventions that
5 can be used by governments to reduce traffic speeds in the hope of improving road
6 safety. An example of such measure is designation of 20 mph zones, which are
7 widely applied in the UK particularly in residential areas.

8 While several studies have been undertaken to analyze the impact of 20 mph
9 zones on various outcome of interest, there remains uncertainty regarding the
10 causal effects of 20 mph zones on road safety. A major challenge for evaluation
11 lies in constructing viable counterfactual outcomes that can represent what would
12 have happened to “treated” units in the absence of the treatment (i.e. designation
13 of 20mph status). Since counterfactual outcomes cannot be observed, regression-based
14 statistical models are usually used to model them, particularly via before-after
15 and time-series methods (e.g. Webster and Layfield, 2003; Grundy et al., 2009).
16 The validity of such methods relies on their ability to control for confounders,
17 which are a set of risk factors for the outcome of interest that are also correlated
18 with treatment assignment. The estimator of treatment effects is consistent and

19 unbiased only if the confounders are properly accounted for. This critical issue,
20 however, is inadequately justified in previous studies.

21 This research contributes to the literature by tackling the issue of confounding
22 using a doubly robust (DR) estimator and subsequently uses this method to evaluate
23 the effect of 20 mph zones on road casualties in London. The DR approach
24 combines outcome regression (OR) and propensity score (PS) models to obtain
25 an estimator which is consistent and asymptotically unbiased so long as at least
26 one of the component models (i.e. OR or PS) is correctly specified. It thus
27 provides two opportunities for valid treatment effect estimates which is useful in
28 situations when the quality of data or knowledge about the underlying processes is
29 not uniform. The DR method has been used routinely to estimate causal treatment
30 effects in other areas of science such as medicine and epidemiology, but, to the
31 best of our knowledge, has not been applied previously in road traffic safety
32 research.

33 Another key contribution of our paper lies in development of a panel data
34 sets to capture variance in road network characteristics over time. A limitation
35 of previous research on this topic is that road network effects have been assumed
36 static which could lead to biased treatment effect estimates if such characteristics
37 operate as confounders.

38 This paper is organized as follows. Section 2 reviews previous literature in the
39 field. Methods are described in Section 3 and Section 4. Our results are presented
40 and discussed in Section 5. Conclusions are then drawn in the final section.

41 **2. Literature Review**

42 A wealth of empirical evidence shows a clear relationship between traffic
43 collisions and vehicle speeds. In particular, mean vehicle speeds are found to
44 be positively related with the number and severity of traffic collisions (Elvik et
45 al., 2004; Elvik, 2009). Speed limits specify maximum desirable traffic speeds
46 and these can be used to reduce the number of road traffic casualties. An example
47 of such a measure is traffic calming, which is especially prevalent in residential
48 areas.

49 Numerous studies have been conducted to evaluate the safety impacts of traffic
50 calming. A meta-analysis by Elvik (2001) investigates the effects on road safety
51 of area-wide urban traffic calming schemes from 33 studies, including research
52 reports from Norway, Sweden, Finland, Denmark, Germany, the Netherlands,
53 Great Britain, France, the United States and Australia. The results show that
54 area-wide urban traffic calming schemes reduce the number of injury accidents
55 by about 15% on average, whilst a 25% reduction in the number of accidents

56 is found for residential streets. Another meta-analysis by Bunn et al. (2003)
57 reviews 16 controlled before-after trials of area-wide traffic calming mainly in
58 high income countries. Their review results also suggest that traffic calming
59 can be effective in reducing the number of traffic crashes. However, previous
60 studies reviewed in these meta-analyses tend to use before-after methods with
61 some defined comparison group, which is not able to fully control for confounding
62 effects, such as selection bias, also known as the regression to mean.

63 A number of studies have examined the impact of traffic calming in te UK,
64 including 20 mph zones, on road safety, traffic speeds, environmental and health
65 outcomes, amenity, traffic volumes, and inequality (Casanova and Fonseca, 2012;
66 Grundy et al., 2009; Steinbach et al., 2011; Tovar and Kilbane-Dawe, 2013;
67 Webster and Mackie, 1996; Webster and Layfield, 2003; Williams and North,
68 2013). Webster and Layfield (2003) investigate 78 20mph zones in London applying
69 before-after methods. Allowing for background changes, total and KSI casualties
70 are found to be reduced by 45% and 57% respectively. Grundy et al. (2009)
71 conduct a time series analysis using data of 399 20mph zones in London from
72 1986 to 2006. Time trend effects are taken into account by using conditional
73 fixed effects Poisson models. The authors also suggest that the RTM effect can be
74 controlled for by dropping data for three, four or five years prior to the implementation

75 of the 20 mph zones.

76 There are two key issues that have not been fully addressed in previous evaluation
77 studies on the impacts of 20 mph zones. First, the methods used in previous work
78 are mainly before-after control studies. Usually, a control group is employed
79 to estimate the counterfactual outcomes of the treatment group. Ideally control
80 groups should have the same or similar characteristics to those of the treatment
81 group, i.e. the control group must be representative of the treated sites. However,
82 in previous research, insufficient attention has been paid to selection of such
83 control groups. For example, Webster and Layfield (2003) use all unclassified
84 roads in London as control data for roads in 20 mph zones. However, due to
85 selection bias, the characteristics of treated and “control” units defined in this way
86 may differ.

87 Second, a fundamental assumption required to draw valid causal inference
88 from observational data is that all confounders are measured and represented
89 adequately. Previous studies on 20 mph zones have largely ignored the potential
90 for road casualties to be associated with the road network characteristics. Yet
91 we know from the literature road casualties are significantly associated with road
92 network characteristics, such as road class, road density and the number of nodes,
93 the connectivity and accessibility of the road network, and the curvature of the

94 road network (e.g. Huang et al., 2010; Marshall and Garrick, 2011; Rifaat et al.,
95 2011; Jones et al., 2008; Quddus, 2008). The failure to account for the effects
96 due to road network characteristics in evaluating traffic calming measures can
97 bias estimates of the safety impacts of 20 mph zones. In this paper we develop a
98 detailed panel data set on road network design to address potential confounding
99 from this source.

100 The doubly robust estimator, originally proposed by Robins et al. (1995), has
101 been described in the statistical literature (Bang and Robins, 2005; Robins et al.,
102 1995; Robins, 1999; Lunceford and Davidian, 2004), and applied extensively in
103 various areas of science. However, it has not yet been used for road safety research
104 although in our view it has great potential.

105 **3. Methods**

106 The DR estimator combines PS and OR models developed using insights from
107 the potential outcomes framework for causal inference. In this section we first
108 introduce the potential outcomes framework and draw attention to its relevant
109 assumptions. We then discuss how a doubly robust estimator of causal effects can
110 be obtained by combining outcome regression and propensity score models.

111 *3.1. Potential outcome framework*

112 In presenting the potential outcome framework, it is necessary to introduce
113 relevant notation. D_i is an indicator of treatment enrolment for individual or unit
114 i . To facilitate understanding, consider only binary treatments. $D_i = 1$, if unit i
115 received the treatment, and 0 otherwise. Let $Y_i(D_i)$ be the potential outcomes for
116 unit i . Therefore, $Y_i(0)$ denotes the level of outcome that unit i would attain if
117 not exposed to the treatment. Likewise, $Y_i(1)$ denotes the level of outcome that
118 unit i would attain if exposed to the treatment. The individual causal treatment
119 effect for unit i can be defined as $\delta_i = Y_i(1) - Y_i(0)$ (Individual Treatment Effect).
120 The fundamental problem of causal inference is that since unit i can be either
121 treated or not, we can only observe one of these two potential outcomes. If
122 unit i is subject to the treatment then $Y_i(1)$ will be realized and $Y_i(0)$ will be an
123 unobservable counterfactual outcome and vice versa.

124 In simple control studies, such as those described in the literature review
125 above, the average treatment effect on the treated (ATE), $E[Y(1) - Y(0)|D = 1]$,
126 is estimated by taking comparisons of the average outcomes between treated and
127 control units, which can be defined as:

$$\begin{aligned} 128 \delta_{ATE} &= E[Y(1)|D = 1] - E[Y(0)|D = 0] \\ 129 &= E[Y(1) - Y(0)|D = 1] + \{E[Y(0)|D = 1] - E[Y(0)|D = 0]\} \quad (1) \end{aligned}$$

130 In the above equation, the term in curly brackets is not zero for most cases
131 due to selection bias, i.e. the treatment assignment is usually associated with the
132 potential outcomes that individuals could attain, with or without being exposed to
133 the treatment.

134 In randomized experiments, the probability of assignment to treatment does
135 not depend on potential outcomes. That is,

$$136 (Y(1), Y(0)) \perp D$$

$$137 \text{Then } E[Y(0)|D = 1] = E[Y(0)|D = 0]$$

138 and therefore

$$139 \delta_{ATE} = E[Y(1)|D = 1] - E[Y(0)|D = 0]$$

$$140 = E[Y(1) - Y(0)|D = 1] + \{E[Y(0)|D = 1] - E[Y(0)|D = 0]\}$$

$$141 = E[Y(1) - Y(0)|D = 1] \text{ (ATE with randomized assignment) (2)}$$

142 Equation (2) provides an unbiased estimator of ATE. Randomized experiments
143 are straightforward and allow the greatest reliability and validity of statistical
144 estimates of causal effects. Whilst they are a valuable tool for treatment evaluation,
145 it is not always feasible to implement a randomized experiment due to high costs
146 and ethical issues. Consequently, causal analysis with observational data uses
147 models to approximate randomized distinctions. There are two critical assumptions
148 underpinning such studies.

149 *3.2. Assumptions*

150 *3.2.1. Unconfoundedness*

151 The validity of causal inferences from observational data crucially relies on
152 the assumption of unconfoundedness. The unconfoundedness assumptions, also
153 known as conditional independence (CIA), assumes all observed differences in
154 characteristics between the treated and untreated units are controlled for, and the
155 outcomes that would result in the absence of treatment are the same for both
156 groups. The CIA creates a selection process analogous to that of randomized
157 experiments. More generally, the distribution of the counterfactual outcomes for
158 treated and untreated groups are the same. In these circumstances it is possible to
159 infer the counterfactual outcomes and the treatment effect can be estimated by the
160 differences between treatment and control groups. The unconfoundedness can be
161 described as:

162 $(Y(1), Y(0)) \perp D \mid X, \forall X$

163 The unconfoundedness assumes that all relevant confounders are observed. This
164 assumption is crucial to making causal inferences in observational studies, but is
165 untestable in practice. The unconfoundedness assumption is too strong and may
166 not hold when unobserved factors that may influence outcomes are not included in
167 the model. However, this assumption can be relaxed by using the difference-in-difference

168 (DID) estimator (Heckman et al., 1997). In the DID approach, the dependent
169 variable is the difference between outcomes in pre-intervention and post-intervention
170 periods. Given data from the pre-treatment period, any time-invariant confounder
171 can be controlled for. In addition, the likelihood of satisfying this assumptions
172 can be strengthened by capturing as much information as possible about potential
173 confounders.

174 *3.2.2. Common support*

175 For valid treatment effect estimation it is also required that both treated and
176 untreated units have overlap in the support of the covariate distributions. This is
177 known as the positivity or the overlap condition (Cole and Hernan, 2008). Also,
178 either extremely high or low values of propensity scores can cause problems when
179 inverse weighting by creating large weighted outcome values (Kurth et al., 2006;
180 Emsley, 2007). Similar to the test used in matching approaches, a histogram
181 showing the distribution of propensity scores for both groups can help identify
182 the positivity and avoid the extreme values problem.

183 *3.3. Doubly robust estimation*

184 The DR estimator is described below in the case of binary treatments for
185 Frequentist inference. For a Bayesian treatment of the DR estimator please see

186 Graham et al. (2015).

187 *3.3.1. Outcome regression*

188 The potential outcome framework can be written in terms of a simple linear
189 regression model:

$$190 Y_i(D) = \alpha + \delta D + \varepsilon_i(D) \quad (3)$$

191 Where $\varepsilon_i(D)$ is the potential outcomes error term. Linear functions are used for
192 notational simplicity. Other functional forms, such as Poisson, can be used in
193 practice. In this analysis, the model is specified in the DID form to eliminate the
194 influence of time-invariant characteristics. Hence $Y_i(D)$ is defined as the difference
195 between outcomes in pre-intervention and post-intervention periods.

196 With observational data, the estimator of the average causal effect, δ' , can be
197 described as:

$$198 \delta' = E(Y_i|D = 1) - E(Y_i|D = 0)$$
$$199 = \delta + E[\varepsilon_i(0)|D = 1] - E[\varepsilon_i(0)|D = 0] + E[\varepsilon_i(1) - \varepsilon_i(0)|D = 1] \quad (4)$$

200 This estimator of the treatment effect will be biased due to the dependence between
201 the treatment assignment D and the error term ε_i . If all potential confounders X are
202 observed and correctly specified in the regression model, the treatment assignment
203 D is independent of the error term ε_i , $D \perp \varepsilon_i \mid X$. The proper specification of the
204 model, however, can be difficult when multiple potential confounders exist. The

205 propensity score can be used as a single covariate and methods based on the PS,
206 e.g. inverse probability weighting, can be applied in causal analysis.

207 3.3.2. *Inverse probability weighting*

208 Different from the outcome regression methods, the inverse probability weighting
209 (IPW) controls for confounding by using a single index, the propensity score. It
210 is the probability that a unit is selected into the treatment group conditional on
211 observed covariates. The first step when implementing the IPW is to estimate
212 the propensity score. For a binary treatment variable, logit and probit models are
213 usually preferred to a linear probability model, which may generate predictions
214 outside the [0, 1] bounds of probabilities. Logit and probit models usually yield
215 similar results, hence the choice between them is not critical (see further discussion
216 of this point in Smith, 1997). In this paper, a logit model is used:

$$217 P(T = 1 | X) = \frac{EXP(\alpha + \beta'X)}{1 + EXP(\alpha + \beta'X)} \quad (5)$$

218 Where α is the intercept and β' is the vector of regression coefficients.

219 The estimator of propensity score, P' , can be predicted based on the estimated
220 parameters and observed covariates for both treated and control individuals. Besides
221 matching, another way of using PS to control for confounding is to weight the
222 observed data. The IPW is defined as the inverse of the conditional probability of
223 an individual's actual treatment status.

224 In observational data the sample is not randomized, but rather one in which
 225 individuals from certain subpopulations are over- or under-sampled. The idea is
 226 that weighting by the IPW estimator creates a pseudo population in which the
 227 distributions of confounders among the treated and untreated are the same as the
 228 overall distribution of those in the original total population (Sturmer et al., 2006).
 229 This indicates that the potential outcomes are independent of the treatment, which
 230 is consistent with the unconfoundedness assumption. The IPW is $1/P'$ for the
 231 treated and $1/(1-P')$ for the untreated. The IPW estimator of the ATE can be
 232 modelled as (Lunceford and Davidian, 2004):

$$233 \delta_{IPW} = N^{-1} \sum_i^N \left(\frac{D_i Y_i}{P'_i} \right) - N^{-1} \sum_i^N \left(\frac{(1-D_i) Y_i}{1-P'_i} \right) \quad (6)$$

234 Similarly, the IPW estimator can be biased if the model for calculating the PS is
 235 misspecified.

236 3.3.3. Doubly robust estimator

237 The doubly robust methods proposed by Robins et al. (1995) combine the
 238 outcome regression and inverse probability weighting in one single model. The
 239 DR estimator can be expressed as the following equation:

$$240 \delta_{DR} = N^{-1} \sum_i^N \left[\frac{D_i Y_i}{P'_i} - \frac{(D_i - P'_i) Y'_{i,D=1}}{P'_i} \right] - N^{-1} \sum_i^N \left[\frac{(1-D_i) Y_i}{1-P'_i} - \frac{(D_i - P'_i) Y'_{i,D=0}}{1-P'_i} \right] \quad (7)$$

241 Where $Y'_i = E(Y | D, X)$ is the predicted value from the outcome regression model
 242 given $D=0,1$ and the baseline covariates X . The two average terms are estimates

243 of the mean potential outcomes, $Y_{X=1}$ and $Y_{X=0}$, if everyone were to be treated
244 and untreated. As a consequence, the difference in means is the effect due to the
245 treatment.

246 In *equation (7)*, the first terms in each average are the IPW estimators for
247 $E(Y_{X=1})$ and $E(Y_{X=0})$ respectively. The second terms are called augmentations
248 (Funk et al., 2011) as this component is formed by taking the product of two bias
249 terms: one from the PS model and one from the outcome regression model. If
250 either bias term equals zero, then it excludes the other non-zero bias term from
251 the incorrect model. That is the DR estimator will be consistent for the true
252 average treatment effect, if either model is correctly specified. (For more detailed
253 demonstration of the DR property, please refer to the work by Lunceford and
254 Davidian, 2004).

255 The standard error can be obtained by bootstrapping the whole sequence of
256 regressions, including the estimation of the propensity score. This can be realized
257 in the STATA package *dr* (Emsley et al., 2008). Figure 1 shows the diagram of
258 applying the DR methods to the estimation of treatment effects.

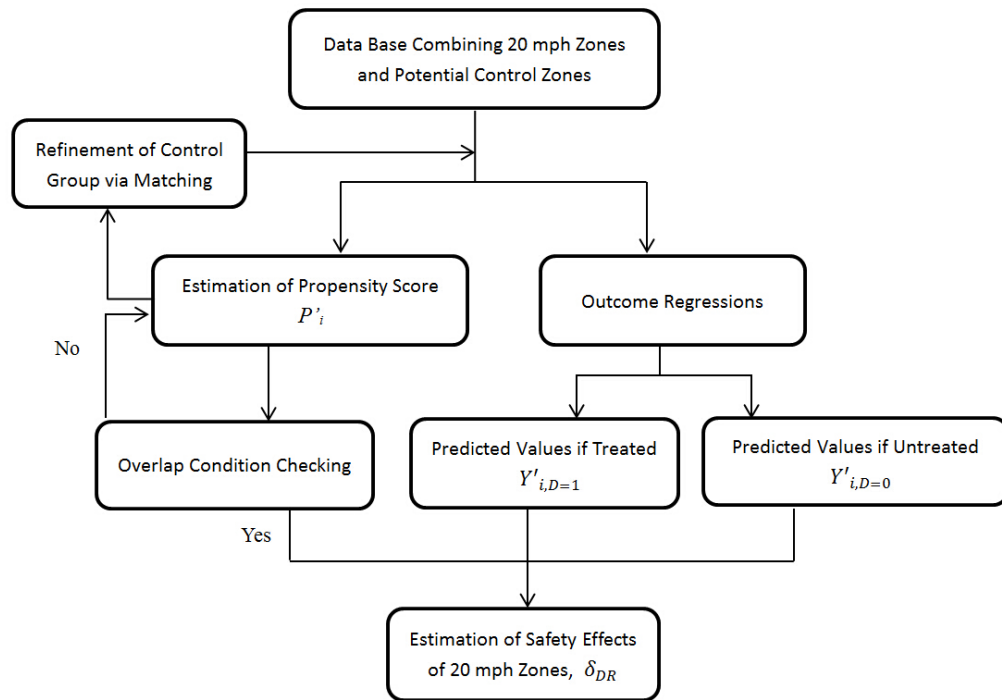


Figure 1: The diagram of the application of the doubly robust method to the evaluation of safety effects of 20 mph zones

259 **4. Data**

260 *4.1. Confounders*

261 The validity of the DR methods heavily relies on the “no unmeasured confounders”
 262 assumption, which is unfortunately untestable. However, its influence can be
 263 lessened by capturing as much information about potential confounders as possible.

264 Theoretically, covariates that affect the treatment assignment and potential
 265 outcomes should be included in the models. In practice, however, selection of such
 266 covariates can be complex due to the lack of precise knowledge of the relations

267 among outcomes, treatment and confounders.

268 Although including additional covariates can increase the precision of the DR
269 estimator (Lunceford and Davidian, 2004), this could generate problems with the
270 common support (Bryson et al., 2002). And although the inclusion of non-significant
271 covariates will not affect the unbiasedness and consistency of the estimates, it can
272 reduce their efficiency, especially with small samples (Augurzky and Schmidt,
273 2000).

274 It is also suggested that omitting important covariates can cause serious bias
275 in estimation (Heckman et al., 1997). Rubin and Thomas (1996) recommend that
276 a covariate should only be excluded if there is consensus that the covariate is
277 unrelated to either the outcome or participation. If there are doubts about this, it
278 is advised to include the relevant covariates.

279 A simulation study by Brookhart et al. (1996) illustrates how the choice of
280 variables included in the propensity score model can affect the bias, variance,
281 and mean squared error of estimated treatment effects. Their results suggest that
282 the optimal practice, in terms of bias and precision, is to include all covariates
283 that affect the outcome regardless of whether they have impacts on treatment
284 assignment. In contrast, however, adding a covariate unrelated to the outcome
285 but related to treatment assignment will increase the variance without decreasing

286 bias.

287 *4.2. Covariates included in DR*

288 The covariates inclusion would be less complicated if criteria for treatment
289 participation were available. Where such criteria are not available, it is still
290 possible to choose covariates based on previous empirical findings. In this study
291 two sets of covariates are considered to be included in the DR models.

292 *4.2.1. Covariates suggested as criterion for 20 mph zones selection*

293 Although the requirements for 20 mph zones have been prescribed in a number
294 of legislation and regulations, the criterion of selecting 20 mph zones remains
295 unclear. The most relevant document is setting local speed limits by DfT (2013),
296 which provides guidance to highway authorities and local traffic authorities who
297 are considering setting local speed limits, including 20 mph zones. The key factors
298 that should be taken into account in any decisions on local speed limits are shown
299 below:

- 300 • History of collisions, including frequency, severity, types and causes;
- 301 • Road geometry and engineering (e.g. bends, junctions);
- 302 • Road function;

- 303 • Presence of vulnerable road users;
- 304 • Existing traffic speeds.

305 According to another report by Steer Davies Gleave (2014), there is considerable
306 variability as to the implementation of 20 mph zones in different authorities.
307 However, most boroughs prioritize areas as 20 mph zones based on collision
308 history, resident requests, and in some cases the presence of schools. Selection
309 of 20 mph zones, therefore, is primarily based on accident history. Pre-treatment
310 accident records are valuable covariates when estimating the DR estimator because
311 they are important predictors of treatment entry and potential outcomes in post-treatment
312 period. The accident data was collected from the STATS 19 data base and was
313 further classified by severity type. The location of an accident was recorded
314 using the British National Grid coordinate system. Each individual accident was
315 located on the map using Geographical Information System (GIS) software, such
316 as MapInfo and Arcmap.

317 Existing traffic speeds, such as the 85th percentile speed and percentages of
318 vehicles over the speed limit are not normally publicly available for all sites on
319 UK roads, however. We address this issue by randomly selecting a large sample
320 of potential control zones within London area. In doing so, it is expected that both
321 treated and untreated zones are observed at every level of pre-treatment traffic

322 speeds, so the overlap condition is met.

323 To account for the impacts of the presence of vulnerable road users on the
324 decision to implement 20 mph zones, the Index of Multiple Deprivation (IMD)
325 is obtained from the office for the Deputy Prime Minister. The Index of Multiple
326 Deprivation integrates data on the following seven deprivation domain indices into
327 one overall deprivation score: income, employment, housing and services, health,
328 education, crime and environment.

329 *4.2.2. Covariates suggested as important factors affecting road casualties*

330 Notwithstanding the covariates discussed above there are areas not meeting
331 the criteria (e.g. the collision history) which may still be selected as 20 mph
332 zones for one or more of the other reasons, such as community concern and
333 engineering factors (DfT, 2013). In other words, there are unknown factors that
334 affect treatment assignment but are not explicitly described in the criterion for 20
335 mph zones selection. As suggested by Rubin and Thomas (1996) and Brookhart
336 et al. (2006), unless there is consensus that the covariate is unrelated to treatment
337 participation, covariates that affect the outcome should be included in the model,
338 because they decrease the variance of the estimated treatment effect without increasing
339 bias. Hence covariates suggested as important factors for analyzing road casualties
340 are also considered.

341 One constraint in previous research on 20 mph zones is that no longitudinal
342 or panel data of road network characteristics has been employed. The statistical
343 relationship between road casualties and the characteristics of a road network
344 has been investigated in the literature as described above, showing in general
345 statistically significant effects. In this study, information regarding the road network
346 was obtained from Ordnance Survey (OS) Meridian, which is a vector map dataset
347 of Great Britain at a scale of 1:50000. This dataset is updated annually and is
348 collected for study period excluding for 2005 due to data availability. A set of
349 variables is extracted from Meridian data set to describe the characteristics of the
350 road network at zone level.

- 351 • Traditional road network characteristics. The length, as well as the density,
352 of the road network is calculated according to road class, e.g. A road, B
353 road, Minor road. Road network nodes are defined as meeting points of two
354 or more roads. The total number and density of nodes is also calculated.
- 355 • Connectivity and accessibility of the road network. It has been suggested
356 that the degree of connectivity and accessibility of a road network can
357 influence the number of crashes (Marshall and Garrick, 2011). The measure
358 used in this study is the link-to-node ratio, which is calculated by dividing
359 the number of links by the number of nodes. A high link-to-node value

360 indicates a more connected road network than one with a low link-to-node
361 value. A node with only one link, also known as a dead end, is usually
362 associated with a residential area. The density of dead ends is used in this
363 study as a measure of the accessibility of a network.

- 364 • Curvature of the road network. Road curvature has been suggested as an
365 important factor influencing road casualties (Jones et al., 2008; Quddus,
366 2008). The literature indicates that straighter roads have more crashes than
367 roads with more bends. The variable used in this research to measure
368 curvature is the number of vertices per km. The number of vertices are
369 obtained using ArcGIS and divided by the road length in each zone.

370 Previous research has also suggested an association between road traffic crashes
371 and socio-demographic characteristics, such as employment, deprivation and land
372 use (Wier et al., 2009; Dissanayake et al., 2009; Graham and Stephens, 2008).
373 In particular, a positive relationship has been found in relation to the size of the
374 population and the level of employment, which implies that more casualties may
375 occur in areas with more residents and job opportunities. To consider this effect,
376 the data for population and employment, as well as the information of land use was
377 obtained from the Office for National Statistics (ONS). In summary, the covariates
378 that we included in the DR model are shown in Table 1. All of these covariates

are included in both outcome regression and propensity score models.

Table 1: Covariates included in the doubly robust model

Covariates	Description
KSI (baseline)	Killed and seriously injured casualties in three years before the intervention
Slightly injured (baseline)	Slightly injured casualties in three years before the intervention
A roads (%)	Percentage of A roads
B roads (%)	Percentage of B roads
Minor roads (%)	Percentage of minor roads
IMD	The index of multiple deprivation
Domestic (%)	Percentage of domestic buildings, e.g. residential area
Non-domestic (%)	Percentage of non-domestic buildings, e.g. business and office district area
Green space (%)	Percentage of green spaces and gardens
Population density	Residential population per m ²
Employment density	Number of employees per m ²
Ratio of Emp to Non-Emp	Ratio of employment to non-employment
Density of dead ends	Ratio of nodes with only one link to all nodes
Links per node	Ratio of road links to nodes
Vertices density	Number of horizontal vertices per km

379

380 4.3. Sample size

381 Figure 2 shows the map of 20 mph zone and control zones in London. The 20
382 mph zones in the dataset cover a large period of time of 1989-2007. Due to data
383 restrictions, only 20 mph zones established between 2002 and 2007 are included
384 in the treatment group. Besides 234 20mph zones, a total of 2844 potential
385 control zones were selected randomly within London area. It is possible that
386 the implementation of 20 mph zones may have impacts on neighboring zones, so
387 zones within 150 meters of each 20 mph zone are not included in the potential
388 control group (Grundy et al., 2009). To ensure that three years data before and

389 after are available for all 20 mph zones. The STATS 19 data used for this analysis
390 includes road accidents in the UK from 1999 to 2010.

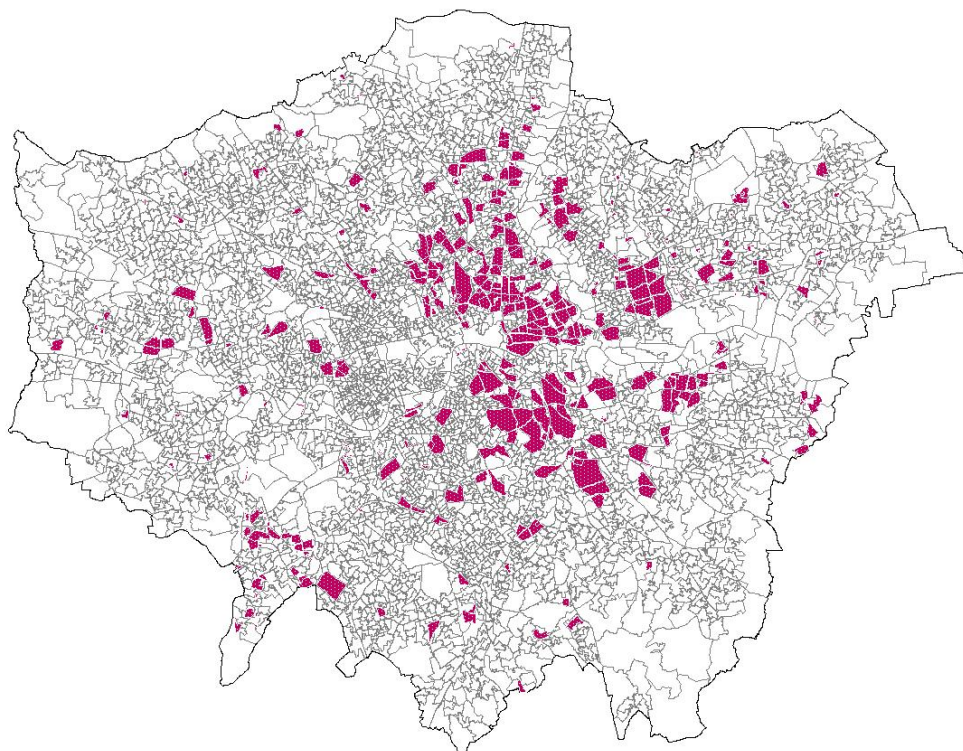


Figure 2: Map of 20 mph zones in London with Lower Layer Super Output Areas Boundary

391 **5. Results**

392 In this section, the safety effects of 20 mph zones are investigated using the
393 DR method. The DR method combines two separate models: the propensity score
394 model and outcome regression model. As discussed earlier, the optimal practice
395 is to use the same set of covariates in both the propensity score and outcome

396 regression models. The regression results from both models are presented, followed
397 by the estimation of 20 mph zones effects using the DR method.

398 *5.1. Propensity score estimation*

399 The first step in the doubly robust method is to estimate the propensity scores.
400 The logit and probit models are usually used in the PS model and give similar
401 results. In this study, the logit model is preferred due to a higher BIC value.
402 The estimation model shows a low Pseudo R-squared value. As Westreich et
403 al. (2011) emphasized, however, the primary purpose of PS model is not to
404 predict treatment assignment, but to balance covariates in order to control for
405 confounding. Previous studies (Brookhart et al, 2006; Myers et al., 2011; Austin,
406 2009) have also shown that better predictive performance does not improve the
407 balance of risk factors for the outcome. It is recommended to use measures of
408 covariate balance to evaluate PSM models (Austin, 2009; McCaffrey et al., 2004).

409 The logit model is regressed on the covariates, which could influence both
410 the treatment assignment and the potential outcomes. The covariate “Minor roads
411 (%)” is dropped due to multicollinearity. Table 2 shows that most covariates are
412 significantly related to the treatment assignment, indicating that they are important
413 in predicting the possibility of being treated. Specifically, 20 mph zones are more
414 likely to be implemented in areas with a higher historical record of slightly injured

415 casualties, which is consistent with the guidelines by DfT (2013). However, the
416 relation between the number of KSI in baseline years and the propensity score is
417 not significant. This indicates that slightly injured casualty is more predominant
418 when making decisions on 20 mph zones. In addition, deprived areas have substantially
419 more 20 mph zones, which is consistent with previous findings (Rodgers et al.,
420 2010). In terms of land use, the propensity score is found to be negatively related
421 to the percentages of non-domestic buildings and green space. In addition, areas
422 with higher density of residential population and lower density of employees have
423 higher propensity of being selected as 20 mph zones. Regarding the characteristics
424 of road network design, only the covariate links per node has a significant impact
425 on treatment assignment, which is not surprising, because they are assumed to
426 have more impacts on road casualties.

427 As discussed in the previous section, a total of 2844 potential control zones
428 were selected randomly within the London area. However, the characteristics of
429 treated and potential control zones may differ in the absence of any treatment.
430 Only untreated zones with similar characteristics to those treated can be used
431 to approximate the counterfactual outcomes of the 20 mph zones. So before
432 proceeding to the doubly robust estimation, the control group need to be refined
433 via matching, which can improve the balance of characteristics between treated

Table 2: Propensity score model

	Coef.	Std. Err.	z	$P > z $
KSI (baseline)	—			
Slightly injured (baseline)	0.007	0.002	3.19	0.001
A roads (%)	0.884	0.288	3.07	0.002
B roads (%)	1.148	0.391	2.94	0.003
IMD	0.015	0.004	4.05	< 0.001
Domestic (%)	—			
Non-domestic (%)	-4.581	1.536	-2.98	0.003
Green space (%)	-0.735	0.386	-1.9	0.057
Population density	173.455	42.180	4.11	< 0.001
Employment density	-243.780	82.203	-2.97	0.003
Ratio of Emp to Non-Emp	0.252	0.094	2.68	0.007
Density of dead ends	—			
Links per node	-0.094	0.019	-5.440	< 0.001
Vertices density	—			
Pseudo R Square: 0.31		BIC: 2145.4		

434 and control groups. Table 3 shows the t-test of differences in covariate means
435 before and after radius matching (caliper=0.05). It can be seen that the characteristics
436 between the treated and original control groups are imbalanced. Matching is
437 subsequently used to refine the control group and the bias due to differences in
438 observable characteristics is reduced as shown in table 3. The sample size of
439 control group is now refined to 1415.

440 Next, we check the distributions of propensity scores for both groups. The
441 histograms in figure 3 show that the propensity scores have similar ranges across
442 the two groups and overlap very well, indicating the overlap assumption is plausible.
443 It is also worth noting that the propensity scores do not have either extremely high
444 or extremely low values, which can cause problems when inverse weighting by

445 creating large, weighted outcome values (Kurth et al., 2006).

446

Table 3: T-test of covariate means pre- and post-matching

Covariate	Unmatched Matched	Mean Treated	Control	%reduct bias	t-test t	$p > t$
KSI	U	5.375	5.715		-0.35	0.726
	M	5.375	5.516	58.7	-0.19	0.851
Slightly injured	U	36.088	34.203		0.33	0.742
	M	36.088	37.013	50.9	-0.19	0.852
A roads (%)	U	0.092	0.059		5.16	< 0.001
	M	0.092	0.084	75.4	0.69	0.488
B roads (%)	U	0.046	0.024		5.35	< 0.001
	M	0.046	0.042	84.8	0.38	0.705
M roads (%)	U	0.862	0.917		-7.26	< 0.001
	M	0.862	0.874	79.1	-0.8	0.423
IMD	U	30.558	22.871		9.61	< 0.001
	M	30.558	30.294	96.6	0.23	0.820
Domestic (%)	U	0.137	0.124		3.75	< 0.001
	M	0.137	0.136	94.5	0.14	0.885
Non-domestic (%)	U	0.077	0.060		3.63	< 0.001
	M	0.077	0.075	85.6	0.38	0.702
Green space (%)	U	0.356	0.423		-4.86	< 0.001
	M	0.356	0.358	97.1	-0.11	0.909
Population density	U	0.009	0.007		10.53	< 0.001
	M	0.009	0.009	98.8	-0.07	0.941
Employment density	U	0.004	0.003		8.17	< 0.001
	M	0.004	0.004	96.3	-0.2	0.842
Ratio of Emp to Non-Emp	U	2.031	2.197		-4.4	< 0.001
	M	2.031	2.045	91.5	-0.23	0.817
Density of dead ends	U	0.055	0.053		0.26	0.795
	M	0.055	0.065	-467.8	-0.86	0.393
Links per node	U	3.030	3.026		0.2	0.844
	M	3.030	3.017	-243.9	0.4	0.687
Vertices density	U	19.275	17.816		2.2	0.028
	M	19.275	19.149	91.4	0.14	0.891

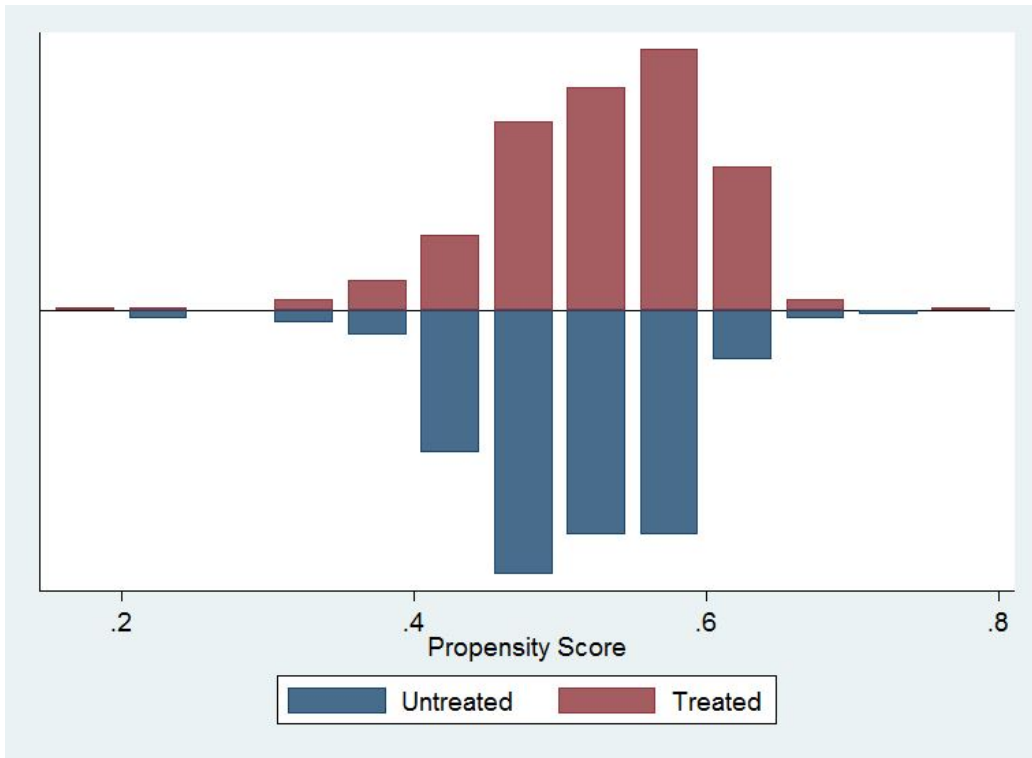


Figure 3: Propensity score distribution by treatment status

447 *5.2. Outcome regression models*

448 We apply generalized linear regression models using the pre-treatment covariates
 449 listed in Table 1 as predictors. The STATS 19 data classifies the casualty by
 450 severity (KSI and slightly injured) and by types (Cycle-, Pedestrian-, and Motor-related).
 451 Regression models for outcomes are fitted on the predictors for the treatment and
 452 control groups separately, and the predicted values will be obtained for the whole
 453 population.

454 Table 4 shows the regression results for different casualty severities and types

455 by groups. Most covariates are significantly associated with the number of casualties
456 for both treated and control groups. Specifically, the number of KSI and slightly
457 injured casualties in baseline years are positively related to the casualty number
458 after the treatment. The density of residential population and employees are
459 used to control for traffic exposure within each zone. Most models show there
460 are positive effects from the level of population and employment. This implies
461 that more casualties may occur in zones with a higher density of residents and
462 job opportunities. Socio-economic deprivation has previously been shown to be
463 positively related to road traffic casualties (Graham and Stephens, 2008), and this
464 has been confirmed by the results of this study which indicate that IMD scores
465 have positively effects on all types of casualties.

466 We further investigate the effects of land use characteristics on casualties.
467 Three main types of land use are examined: domestic, non-domestic and green
468 space. The non-domestic area studied in this paper includes office district area and
469 business area, such as large trade area, warehousing, and wholesaling. The results
470 suggest higher percentages of domestic and non-domestic areas are associated
471 with more casualties, whilst there are fewer casualties in areas with higher percentages
472 of green space. This is consistent with previous findings (Pulugurtha et al., 2013),
473 which suggest that land use characteristics such as residential and business areas

474 are generally high traffic activity generators.

475 Regarding road network characteristics, as suggested in many other studies
476 (e.g. Huang et al., 2010), road density is positively associated with road casualties
477 at all types and severity levels. Two covariates were used as indicators of road
478 network connectivity: the links per node and the number of nodes with one link
479 (Chin et al., 2008). It can be hypothesized that areas with a better-connected
480 road network will have more casualties, because pedestrians, cyclists and motor
481 vehicles have better accessibility and total traffic activities tend to be more frequent.
482 The results indicate that an increase in links per node is associated with an increase
483 in the casualty numbers for all severities. Lower densities of nodes with one link,
484 also known as dead ends, usually indicate limited access to streets. The results
485 show that higher densities of dead ends are associated with fewer casualties.
486 The results also suggest that road networks with a greater degree of horizontal
487 curvature, i.e. more vertices per km, are associated with fewer casualties. This
488 result is consistent with previous findings (e.g. Jones et al., 2008; Quddus, 2008).
489 The mechanisms for this could be complex, however, one possible reason is that
490 vehicles have lower speeds when passing curving road sections.

491 The adjusted R-square values are estimated to show the degree to which the
492 outcome regression models fitted the treatment and control groups are appropriate

493 to prediction. The adjusted R-square values are more than 60% for most models
494 and are 70% for slightly injured models. The high values suggest the predicted
495 outcomes from the regression models are valid.

Table 4: Outcome regression models

Covariates	Slightly Injured		Killed and Seriously Injured		Cycle-related Casualties		Pedestrian-related Casualties		Motor-related Casualties	
	Treatment=1	Treatment=0	Treatment=1	Treatment=0	Treatment=1	Treatment=0	Treatment=1	Treatment=0	Treatment=1	Treatment=0
KSI	0.049 ***	0.004 ***	0.043 ***	0.013 ***	0.069 ***	0.010 ***	0.046 ***	0.001 ***	0.032 ***	0.004 ***
Slightly injured	0.003 ***	0.010 ***	0.005 ***	0.009 ***	2.0E-04 ***	0.009 ***	0.004 ***	0.010 ***	0.006 ***	0.011 ***
A roads (%)	1.353 ***	1.373 ***	1.050 ***	1.402 ***	1.282 ***	1.542 ***	1.054 ***	1.066 ***	1.474 ***	1.483 ***
B roads (%)	1.209 ***	0.552 ***	0.664 ***	1.076 ***	0.673 ***	0.889 ***	1.467 ***	0.581 ***	1.371 ***	0.490 ***
IMD	0.029 ***	0.004 ***	0.033 ***	0.002 ***	0.023 ***	-2.0E-04 ***	0.026 ***	0.009 ***	0.041 ***	0.005 ***
Domestic (%)	4.636 ***	0.223 ***	1.535 ***	-0.318 ***	0.672 ***	-2.288 ***	6.819 ***	3.688 ***	6.010 ***	0.638 ***
Non-domestic (%)	0.600	0.389	1.078 ***	-0.198 ***	0.156 ***	0.740 ***	2.394 ***	2.345 ***	-0.272 ***	-1.713 ***
Green space (%)	-0.230 *	-0.061 ***	0.012 ***	-0.339 ***	-1.395 ***	-1.052 ***	0.260 ***	-0.221 ***	0.427 ***	0.429 ***
Population density	44.9 ***	32.4 ***	59.7 ***	40.9 ***	177.3 ***	40.7 ***	54.4 **	2.3 ***	-5.5 ***	29.6 ***
Employment density	48.8 ***	71.3 ***	118.5 ***	114.0 ***	423.4 ***	191.8 ***	143.9 **	-14.9 **	132.5 ***	-6.7 *
Ratio of Emp to Non-Emp	0.067 ***	0.013 ***	0.138 ***	0.058 ***	-0.256 ***	0.172 ***	0.228 ***	0.058 ***	0.318 ***	-0.058 ***
Density of dead ends	-1.970 ***	-0.838 ***	-3.875 ***	-0.625 *	-3.295 ***	-2.051 ***	-2.011 ***	-0.196 ***	-1.709 ***	-0.405 **
Links per node	0.207 **	0.038 ***	0.016 ***	0.189 ***	0.516 ***	0.212 ***	-0.117 **	0.031 ***	0.093 ***	0.222 ***
Vertices density	-0.013 ***	-0.004 ***	-0.015 **	-0.002 **	-0.010 ***	-0.003 **	-0.016 ***	-0.002 ***	-0.014 ***	-0.005 ***
R-square	0.704	0.696	0.545	0.516	0.660	0.669	0.619	0.587	0.623	0.583
Obs	234	1415	234	1415	234	1415	234	1415	234	1415

Notes: Figures are significant at: *90%, **95% and ***99%.

497 *5.3. Effects of 20 mph zones on road casualties*

498 Given the satisfaction with the component models, propensity score and outcome
499 regression as discussed in the above sections, now we proceed to estimate effects
500 of 20 mph zones using the doubly robust method. For comparison, three other
501 methods are also applied: inverse probability weighting, regression adjustment
502 and propensity score matching. Besides inverse probability weighting, another
503 application of propensity score is matching. The basic idea is to match each
504 treated unit to untreated units with similar propensity scores. Conditional on the
505 propensity score, differences in observed outcomes between the two groups can
506 be solely attributed to the intervention impacts. The matching algorithm used in
507 this paper is radius matching (caliper=0.05). For detailed discussion of matching
508 algorithms, please refer to the work by Heinrich et al. (2010). Table 5 presents the
509 estimations of the safety effects of 20 mph zones by casualty types and severities.

510 The 20 mph zones consistently have a significant impact on reducing casualties
511 in both absolute number and percentages. The results are very similar for all
512 four methods, with a reduction in slightly injured casualties of around 1.7 (10%
513 in percentage), and KSI of around 0.73 (24% in percentage) respectively. The
514 number of pedestrian-related casualties decreases by 0.85 (21% in percentage),
515 which is significant at the 99% level for all four methods. In terms of motor-related

516 casualties, only the absolute number of casualties is found to be significantly
517 reduced by 1.5, whilst this effect is not significant when estimated in percentage.
518 No significant effects of 20 mph zones are found on cycle-related casualties.
519 The similar results from four methods increase confidence in the doubly robust
520 method.

521 To investigate the robustness of DR method to model misspecification, we
522 further examine false models by omitting confounders from both regression and
523 propensity score models. The omitted confounders are significant predictors of
524 outcomes but insignificant for propensity score estimation. This is similar to the
525 routine done by Lunceford and Davidian (2004), and Bang and Robins (2005).
526 The DR method should offer protection against the bias due to the misspecification
527 of regression model. The results are shown in Table 5. All false models are
528 distinguished by superscript “#”. It can be seen that the false OR models lead
529 to unstable estimates with relatively large standard errors due to the omission of
530 significant covariates, while false DR as well as IPW estimators are consistent
531 with the original results for most models. This shows that the DR method is
532 superior for affording protection against misspecification.

533 It is worth noting that the effects of 20mph zones on reducing casualties estimated
534 in this paper are smaller than the results from previous studies. For example, the

535 reduction in casualties varies from 22% to 61% according to previous reports
536 (Webster and Mackie, 1996; Webster and Layfield, 2003; Grundy et al., 2009;
537 Steinbach et al., 2011). There are several possible reasons for this. First, is that the
538 implementation period of 20 mph zones investigated in previous studies is from
539 1991 to 2008, and the effects of 20 mph zone may diminish over time (Grundy
540 et al., 2009). If we focus on 20 mph zones implemented in recent years, the
541 previous findings are more consistent with the ones in this paper. For example,
542 Grundy et al. (2009) used conditional fixed effects Poisson models to estimate
543 the effects of 20 mph zones using the same data. They first used the data from
544 1987 to 2006 and found significant reduction in casualties and collisions. Their
545 initial findings are much higher than the ones of this study. Then they restricted
546 analyses to 2000-2006, the period with the lowest annual numbers of casualties.
547 The results are very similar to the ones of this study this time. For example, they
548 found that the percentage reductions are 28.4% for KSI and 21.6% for pedestrian
549 injuries, and no significant effect for cyclists. These results are, to a large extent,
550 consistent with our findings. Second, and as discussed earlier, the over-estimation
551 of treatment effects in previous studies could be also due to the selection bias.
552 For example, Webster and Layfield (2003) use all unclassified roads in London
553 as “control” data for roads in 20 mph zones. The characteristics of treated and

554 control zones, e.g. historical records of casualties differ in the absence of the
555 treatment, and the counterfactual outcomes approximated by such “control” zones
556 will be biased. Finally, use of detailed panel data on road network characteristics
557 provides adjustment for sources of confounding that have not been addressed in
558 previous studies.

Table 5: Effects of 20 mph zones on road casualties

	Slightly Injured		Killed and Seriously Injured		Cycle-related Casualties		Pedestrian-related Casualties		Motor-related Casualties					
	Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.				
Doubly Robust	-1.825	0.58	***	-0.727	0.172	***	-0.129	0.419	-0.858	0.263	***	-1.564	0.471	***
Inverse Probability Weighting	-1.762	0.638	***	-0.746	0.182	***	-0.123	0.417	-0.867	0.273	***	-1.518	0.492	***
Regression Adjustment	-1.777	0.583	***	-0.723	0.173	***	-0.11	0.424	-0.842	0.267	***	-1.547	0.47	***
Propensity Score Matching	-1.628	0.708	**	-0.787	0.242	***	-0.567	0.438	-0.68	0.307	**	-1.167	0.62	*
Doubly Robust #	-1.844	0.58	***	-0.645	0.231	***	-0.108	0.412	-0.841	0.271	***	-1.54	0.484	***
Inverse Probability Weighting #	-1.841	0.62	***	-0.639	0.237	***	-0.099	0.416	-0.853	0.282	***	-1.527	0.499	***
Regression Adjustment #	-1.306	0.834	*	-0.607	0.237	*	-0.01	0.414	-0.689	0.315	**	-1.213	0.584	*
	Slightly Injured (%)		Killed and Seriously Injured (%)		Cycle-related Casualties (%)		Pedestrian-related Casualties (%)		Motor-related Casualties (%)					
	Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.				
Doubly Robust	-0.092	0.04	***	-0.243	0.049	***	-0.027	0.071	-0.217	0.046	***	-0.078	0.062	
Inverse Probability Weighting	-0.066	0.058		-0.255	0.049	***	-0.021	0.074	-0.217	0.047	***	-0.068	0.065	
Regression Adjustment	-0.089	0.042	**	-0.247	0.049	***	-0.026	0.073	-0.211	0.047	***	-0.077	0.061	
Propensity Score Matching	-0.113	0.04	***	-0.224	0.059	***	-0.035	0.067	-0.202	0.053	***	-0.095	0.066	
Doubly Robust #	-0.072	0.041	*	-0.239	0.054	***	-0.019	0.072	-0.223	0.047	***	-0.074	0.057	
Inverse Probability Weighting #	-0.046	0.089		-0.245	0.057	***	-0.028	0.075	-0.221	0.048	***	-0.053	0.07	
Regression Adjustment #	-0.069	0.032	*	-0.215	0.079	*	-0.021	0.073	-0.266	0.081	*	-0.075	0.058	

559 Notes: Figures are significant at: *90%, **95% and ***99%.

560 **6. Discussion and Conclusions**

561 Several studies have been conducted to evaluate the effects of 20 mph zones
562 on road casualties in the UK. A key issue with causal analysis concerns how the
563 statistical methods employed account for confounding. The ability to draw causal
564 inferences from observational data relies on two properties: correctly specified
565 models and comparability between the treatment and control groups under study.
566 Neither of these issues has been addressed rigorously in previous studies. In this
567 paper, we have applied the doubly robust method which affords us two opportunities
568 for obtaining consistent and asymptotically unbiased causal effect estimates. Given
569 the fact that we rarely know the exact relations among potential outcomes, treatment
570 assignment, and confounding factors; the DR property is useful as it increases
571 scope for satisfying model assumptions in practice. In addition, the propensity
572 score incorporated in the doubly robust method can be used as the criterion when
573 constructing the control group.

574 Our results show that the 20 mph zones consistently have significant impact
575 on reducing casualties in both absolute number and percentages, especially for
576 KSI and pedestrian-related casualties. Considering the diminishing effects of 20
577 mph zones over time, our results are consistent with the general conclusions of
578 previous research in this field.

579 This paper also has two other major findings. First, previous studies rarely
580 look at the criteria for 20 mph zones selection. Although there is considerable
581 variability as to the implementation of 20 mph zones in different authorities,
582 propensity score estimation suggests that the main factors affecting the decisions
583 on 20 mph zones are the historical records of casualties and socio-economic characteristics,
584 e.g. deprivation, land use, and population. Second, by developing a panel data of
585 OS Meridian TM 2, the variation in the road network across time is controlled for
586 in our models. The outcome regression models further show that zones with road
587 network of high connectivity and more bends have more casualties.

588 There are also some limitations with the analysis presented in this paper.
589 Due to data availability, the effects of 20 mph zones on traffic speeds are not
590 investigated in the model. And population and employment are used instead of
591 traffic volume to reflect the overall traffic activities. Despite these, the results
592 from both the propensity score and outcome regression models suggest that the
593 covariates included are significantly associated with the implementation of 20
594 mph zones and road casualties.

595 As suggested by Grundy et al. (2009), the effects of 20 mph zones may
596 diminish over time. A study on temporal heterogeneity of treatment effect would
597 make an interesting question. We also suggest researchers to compare the doubly

598 robust method with other widely used causal methods, such as empirical Bayes
599 for future research.

600 **7. Acknowledgments**

601 This work was supported by the Funds for International Cooperation and
602 Exchange of the National Natural Science Foundation of China (Grant No.5151101143),
603 and the Natural Science Foundation of Jiangsu Province (Project No.BK20150615).
604 We would like to thank Chris Grundy at London School of Hygiene and Tropical
605 Medicine for supplying the data of London 20 mph zone.

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