Do We Need Speed Limits on Freeways?*

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Job Market Paper

Abstract

When choosing his speed, a driver faces a trade-off between private benefits (time savings) and private costs (fuel cost and own damage and injury). Driving faster also has external costs (pollution, adverse health impacts and injury to other drivers). This paper uses large-scale speed limit increases in the western United States in 1987 and 1996 to address three related questions. First, do the social benefits of raising speed limits exceed the social (private plus external) costs? Second, do the private benefits of driving faster as a result of higher speed limits exceed the private costs? Third, could completely eliminating speed limits improve efficiency? I find that a 10 mph speed limit increase on highways leads to a 3-4 mph increase in travel speed, 9-15% more accidents, 34-60% more fatal accidents, and elevated pollutant concentrations of 14-25% (carbon monoxide), 9-16% (nitrogen oxides), 1-11% (ozone) and 9% higher fetal death rates around the affected freeways. I use these estimates to calculate private and external benefits and costs, and find that the social costs of speed limit increases are three to ten times larger than the social benefits. In contrast, many individual drivers would enjoy a net private benefit from driving faster. Privately, a value of a statistical life (VSL) of $6.0 million or less justifies driving faster, but the social planner's VSL would have to be below $0.9 million to justify higher speed limits. The substantial difference between private and social optimal speed choices provides a strong rationale for having speed limits. Although speed limits are blunt instruments that differ from an ideal Pigovian tax on speed, it is highly unlikely that any hidden administrative costs or unforeseen behavioral adjustments could make eliminating speed limits an efficiency-improving proposition.

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1 Introduction

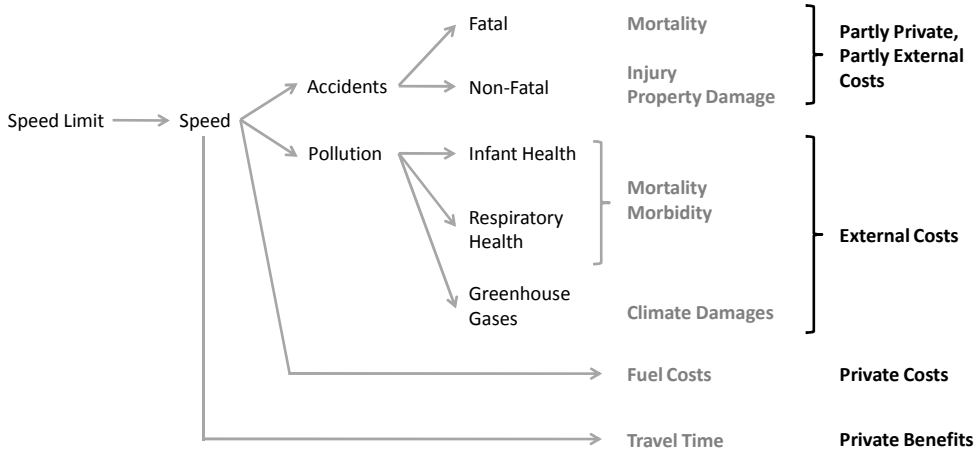
An interesting and actively debated policy question is: should we leave speed choices up to individual drivers? Drivers in many countries are so accustomed to highway speed limits that they take them for granted, but it is nevertheless not obvious that having such limits is socially desirable. Germany does not have speed limits for many of its freeways, and several U.S. states did not have them before 1974 or in the late 1990s. When choosing his speed, a driver faces a trade-off between private benefits (time savings) and private costs (increased fuel use, risk of personal injury, death or damage). It is thus an empirical question if driving faster than the current speed limit is rational. Besides private costs, there are external costs to driving faster: increased pollution, adverse health impacts and damage or injury to other drivers. To justify having speed limits, it is not sufficient to observe that driving faster has external costs as well as private costs. A speed limit is a crude rule that differs from an ideal Pigovian tax on speed. Uniform speed limits for all traffic and weather conditions take away discretion from drivers, and may establish a bad focal point during adverse driving conditions. Therefore, external costs must drive a *substantial* wedge between private and social optimal speed choices for speed limits to increase efficiency. This issue is particularly relevant given the recent debate about speed limits. Early in 2011, Spain reduced the freeway speed limit from 120 to 110 kilometers per hour (kph) to achieve gasoline reductions, while the Netherlands raised it from 120 to 130 kph to reduce travel time.[1] In the United States, travel time reduction inspired Kentucky (2007), Utah (2009), Ohio (2011) and Texas (2011) to increase their posted maximum speed, with similar proposals underway in Illinois and South Carolina.

This paper aims to answer three related questions. First, should we raise speed limits? A social planner would do so only if the social benefits of speed limit increases exceed the social (private plus external) costs. Second, are speed limits binding? That is, would individuals enjoy private net benefits from driving faster if speed limits were raised? Third, could completely eliminating (as opposed to raising) speed limits be an efficiency-improving proposition? In order to answer these questions, I estimate the effect of speed limit increases on a wide range of outcome variables: travel time, accidents, air pollution and health. I use these estimates to calculate the private and external benefits and costs summarized in Figure 1.

I use a unique setting and rich data to address these questions. The 1987 amendment and 1995 repeal of the National Maximum Speed Law in the United States provide quasi-experimental variation in speed limits. Between 1974 and 1987, this law prescribed a maximum speed limit of 55 mph across the entire United States. In 1987, states were allowed to raise the speed limits to 65 mph on rural interstates, but not on other similar urban or rural highways. In 1996, speed limit

[1] Spain's deputy prime minister Alfredo Pérez Rubalcaba expressed it as follows: "We are going to go a bit slower and in exchange for that we are going to consume less gasoline and therefore pay less money." (http://www.guardian.co.uk/environment/2011/feb/25/spain-speed-limit-oil-prices). Dutch transport minister Melanie Schultz van Haegen defended her decision by claiming that "a higher speed limit leads to a travel time reduction of up to eight percent." (http://www.rijksoverheid.nl/ministeries/ienm/nieuws/2011/02/28/130-km-u-van-start-op-afsluitdijk.html). Other governments proposed to decrease speed limits to reduce traffic accidents (United Kingdom, 2009) or pollution and associated adverse health effects (Texas, 1992; green parties in Europe).

Figure 1: An Overview of the Costs and Benefits of Speed Limit Changes



Notes: Higher speed limits may lead to a higher average travel speed. This higher speed has a direct benefit (reducing travel time), but also three direct costs: higher accident rates, increased pollution and increased fuel expenditures. The pollution channel has indirect negative effects on infant and respiratory health, and climate change. Time savings benefits are private, while some of the costs are externalities.

authority was returned to the states, which decided to raise speed limits on a variety of highways. This provides a rare opportunity to use difference-in-differences and ratio-in-ratios (count data) methods to identify the effect of speed limit changes on travel speed, accidents, pollution and health. To account for potential statewide trends in these outcome variables, I need to predict what would have happened on the affected highways in absence of the changes in the law. I therefore construct control highways or areas that are unaffected by the speed limit changes, but otherwise very similar to the affected highways or areas. Also, I exploit geographically precise micro data to make within-state difference-in-differences comparisons while holding constant weather, daylight, hour-of-day, traffic density, road construction, and much else.

I explicitly address whether control highways are indirectly affected by the speed limit changes through traffic substitution towards roads with higher speeds. I find that this is only a minor concern. I also demonstrate that my results stand up against potential identification challenges. One such challenge is that governments are unlikely to assign speed limits randomly. Fortunately, the 1987 speed limit changes provide a source of almost random variation as states were only permitted to raise speed limits on their rural interstates but not on other – otherwise very similar – major highways. I show that there are no differential pre-existing trends for the treatment and control highways, and present robustness checks with subsamples of control highways that match, e.g., the accident rate of treatment highways. The results are robust to such specifications.

My paper uses a detailed data set to evaluate the effects of speed limit changes. First, I use location descriptions of speed limit changes in California, Oregon and Washington. These states are selected because of superior data quality and availability. Second, I collect hourly measurements of actual traffic speed. Third, I use a data set of all highway accidents. Fourth, I use daily air pollution

measurements at various monitoring stations. Fifth, I requested all birth records in California to estimate the effect on infant health. Finally, I use geographical mapping techniques to augment these data sets with meteorological and geographic information wherever applicable.

In terms of the specific outcome variables, I find that a 10 mph speed limit increase leads to a 3-4 mph increase in travel speed, 9-15% more accidents, 34-60% more fatal accidents, a shift towards more severe accidents, and elevated pollution concentrations of 14-25% (carbon monoxide), 9-16% (nitrogen oxides) and 1-11% (ozone) around the affected freeways. The increased pollution leads to a 0.07 percentage point (9%) increase in the probability of a third trimester fetal death, and a positive but small and statistically insignificant increase in the probability of infant death. I use these estimates to calculate the time saving benefits and the private and external costs from accidents and deteriorated infant health. Moreover, I combine the travel speed estimates with engineering data to compute the increase in fuel consumption at higher speed. Similarly, I combine the air pollution estimates with epidemiology research to compute adverse health effects for adults.

Using these estimates and a wide array of plausible values of a statistical life (VSL) and values of time routinely adopted by governments, I find that the social costs of raising the speed limit from 55 to 65 mph are three to ten times larger than the social benefits. My social cost estimates are two to four times larger than in previous studies, in large part due to the greater comprehensiveness of my approach: I not only consider travel time and fatal accidents, but also non-fatal accidents, climate damages, fuel costs and health. Perhaps surprisingly, the costs from pollution-induced adverse health impacts are about as large as the costs from traffic fatalities. I further find that if travel time is valued only slightly below the average after-tax wage, the net social benefits are negative even when road fatalities and pollution-induced mortality are ignored. In contrast, I find that many individual drivers would enjoy a net private benefit from driving faster as a result of the higher speed limit. This reflects the substantial external costs not considered by a typical individual. Privately, a VSL of \$6.0 million or less justifies driving faster, but the social planner's VSL would have to be below \$0.9 million to justify higher speed limits. While \$6.0 million is within the conventional range of VSL estimates, \$0.9 million falls well below it. These results suggest a surprisingly large difference between the social and private optimal speed choices. Although speed limits are a blunt instrument for dealing with the various externalities involved, it seems highly unlikely that any hidden administrative costs or unforeseen behavioral adjustments could make completely eliminating speed limits an efficiency-improving proposition.

A seminal paper in this literature is Ashenfelter and Greenstone (AG; 2004), who use the 1987 speed limit changes to estimate the value of a statistical life based only on travel time and fatal accidents (see Section 2 for details). My paper's main contributions are threefold.

First, I explicitly distinguish between private and external costs and benefits, and show a stark contrast between the net benefits from the perspective of an individual driver and a social planner.

Second, because I employ an unusually rich data set, I am able to estimate the effect of speed limit changes on additional outcome variables such as non-fatal injuries, property damage from accidents, air pollution and the health of infants and others who live near freeways. To the best

of my knowledge, these results are new to the literature.[2] These estimates allow me to perform a more complete cost-benefit analysis. They are also interesting in their own right. For instance, estimating the effect of speed on pollution informs us about how higher tailpipe emissions translate into higher atmospheric pollutant concentrations. This could help guide environmental legislation such as engine technology standards.

Third, by exploiting within-state variation in speed limits and a wide range of control variables, my approach mitigates potential omitted variable bias. Using within-state speed limit variation is a useful and necessary complementary approach to existing cross-state regressions for speed and fatal accidents. AG use the fact that not all states raised their rural interstate speed limits in 1987 and compare highways of the same type (e.g., rural interstates), but across states with potentially dissimilar driving conditions (e.g., New Jersey vs. Iowa). I compare similar but differently classified highways (e.g., rural interstates and "rural principal arterial"), but within one state so that driving conditions are more similar. I present evidence that this choice of treatment and control highways is reasonable. Moreover, due to data limitations, AG do not include any control variables. This raises omitted variable bias as a possible concern. States differ in weather, climate, highway construction activity, and trends in vehicles types, vehicle safety, and other laws that affect driving (e.g., drunk driving and seat belt laws).[3] My paper addresses this concern by including many control variables, such as weather, road and driver conditions, and demographics.

This research is relevant for today's policy makers, since past experiences can inform them about whether speed limits are desirable and if raising speed limits further would be in society's best interest. Using engineering and epidemiological evidence on the past and current relationships between speed, pollution and health, many results in this paper can be extrapolated to obtain pollution and health effects for current speed limit changes. The large difference between private and social net benefits is likely to persist, especially because governments currently consider speed limits in the 75-90 mph range where the strong upward sloping relationship between speed and emissions remains even for today's new vehicles. This paper also demonstrates that the common approach to evaluating speed limits, based on a single cost or benefit or a single trade-off between travel time and fatal accidents, may lead to incorrect conclusions.

The paper starts by discussing relevant literature. Section 3 lays out the empirical strategy. In Section 4, I briefly discuss the various data sources. Section 5 provides summary statistics on quantities and trends to explore the plausibility of the identification assumptions. Sections 6-9 present the econometric framework and estimation results of the various outcome variables: speed, accidents, pollution and infant health. Section 10 combines these estimates in a cost-benefit analysis that also includes impacts on fuel consumption, climate change and adult health, and contrasts the private and social costs. Sections 11 and 12 offer further discussion and conclusions.

[2] Keller *et al.* (2008) study the effect of a temporary five day speed limit reduction on Swiss freeways in 2003 using an air pollutant dispersion simulation model, and predict that NO_x traffic-related emissions decreased by 4%.

[3] For example, consider the weather. AG's seven control states in the Northeast are far away from many treatment states, and a bad winter could hit the Midwest but not the Northeast. Since most accidents occur in the winter, and if the safety of rural interstates was particularly compromised by winter conditions, the treatment effect would absorb this weather-induced change in accidents.

2 Related Literature

2.1 The Relationship Between Speed Limits, Speed and Accident Incidence

When vehicles collide, the damage is proportional to the square of the difference in the vehicles' relative speed. Hence, conditional on an accident occurring, we expect that higher-speed collisions cause more severe injuries, more injuries that result in death, and more property damage. Research has also shown that vehicles are more difficult to control at higher speed. It is harder to get and retain traction if a sudden change in speed or direction is required. There is also less time to avoid hitting other cars or fixed objects (Wong, 2008). Therefore, both accident incidence and accident severity are expected to increase with speed.

Only one well-known study has produced modern empirical evidence on how speed limits affect speed and accidents (Ashenfelter and Greenstone; AG, 2004). This is surprising, since other driving-related policies have attracted considerable attention from academic researchers. Examples include the impact on accidents of seat belt laws (Cohen and Einav, 2003), highway police enforcement (DeAngelo and Hansen, 2011) and the share of light trucks and SUVs (Anderson, 2008; Jacobsen, 2011). AG use annual data by state and road type in a difference-in-differences framework to estimate the impact of the 1987 speed limit changes on speed and fatal accidents. They can employ cross-state variation in the adoption of the 65 mph speed limit: seven states in the northeast retained the 55 mph limit, whereas the other eligible states adopted the 65 mph limit. They find that the average speed increased by 2.5 mph and fatality rates by 35%. The paper uses the trade-off between travel time saved and increased risk of mortality to calculate an upper bound on the value of a statistical life: $1.54 million (1997 USD) for the full sample, but higher estimates for California ($4.75 million) and Oregon ($5.41 million). Section 10 discusses how my results are different.

AG do not investigate how speed limits affect non-fatal accidents or property damage. The costs of non-fatal accidents may be substantial, since they occur much more frequently than fatal accidents, and can have high medical and lost quality of life costs (National Safety Council, 2011).

2.2 The Relationship Between Speed and Air Pollution

Extensive engineering research shows that vehicle speed and emissions per mile are related in a non-linear way (e.g., Litman and Doherty, 2009). Such engineering studies report results from lab test cycles, in which a number of vehicles are driven at a range of speeds under similar conditions. Exhaust gas samples are collected to plastic bags or by electronic measurement. These studies find that at higher speeds, carbon monoxide (CO) and nitrogen oxide (NO_x) emissions per mile increase rapidly and disproportionately to fuel consumption. These pollutants react in the atmosphere to form ozone (O_3) and small particulate matter (PM_{10}). The Environmental Protection Agency (EPA)'s test results for the 1990 vehicle fleet suggest that CO emissions triple when the vehicle speed increases from 55 to 65 mph. NO_x emissions increase by about 50%. The average fuel economy decreases by 18%. More details are given in Section 8.

2.3 The Relationship Between Air Pollution and Health

There is an extensive literature on the relationship between air pollution and health. Medical and epidemiological studies have documented a strong association between adverse health outcomes for infants and for patients with respiratory diseases. High CO concentrations suppress the body's ability to deliver oxygen to organs and tissues. NO_x has been associated with respiratory problems. PM_{10} can cause heart and lung damage, possibly through inflammations that weaken the immune system. O_3 exposure is thought to lead to breathing difficulties, inflammation, aggravation of asthma and increased susceptibility to pneumonia and bronchitis, as well as permanent lung damage (Seaton *et al.*, 1995; EPA, 2011b). Pollution exposure is also thought to affect fetal health, birth weight and pre-term birth (Huel *et al.*, 1993; Wilhelm and Ritz, 2003). Further, O_3 and PM_{10} have been shown to be risk factors for respiratory related postneonatal mortality and sudden infant death syndrome (Bobak and Leon, 1999; Ha *et al.*, 2003; Woodruff *et al.*, 2008).

Interestingly, although there are no modern empirical studies of how speed limits affect infant health, some highly credible studies of related questions suggest that speed limits increases might have adverse health effects. Currie and Walker (2011) use the reduction in congestion related air pollution from the introduction of electronic toll payments (E-ZPass) to identify the effect of air pollution on infant health. Using birth records data from New Jersey and Pennsylvania, they find that the introduction of E-ZPass decreased prematurity and low birth weight for infants from mothers who live within two kilometers of a toll plaza by 10.8% and 11.8%, respectively. Chay and Greenstone (2003) exploit the 1981-1982 recession as a natural experiment that caused variation in air pollution across counties. They find that a 1% reduction in total suspended particulates leads to a 0.35% decrease in the infant mortality rate. Currie and Neidell (2005) use rich individual level data and find significant effects of CO and PM_{10} on infant mortality in California. Using periods of unusually heavy traffic as an instrument, Knittel *et al.* (2010) find that the impact of pollution on infant mortality is even higher.

The second relevant literature studies the effect of air pollution on adult and children's health. Air pollution leads to premature deaths, mostly due to respiratory causes but also from cardiovascular disease (Schwartz, 2001; Bell *et al.*, 2004). Wilhelm *et al.* (2008) find that children living in high O_3 and PM_{10} areas experienced asthma symptoms more frequently. Children living close to heavy traffic were more often hospitalized with respiratory problems. CO (Neidell, 2004) and O_3 (Lleras-Muney, 2010) have been shown to have significant effects on child hospitalizations for asthma. The EPA uses some of these, and various other, studies to quantify health effects associated with changes in pollution. In Section 10, I follow the EPA's approach.

Health economics studies typically seek to exploit quasi-random variation in pollution exposure. Most epidemiological studies use a case-control design, in which subjects are not randomly assigned to pollutant concentrations. Rather, they assign treatment and control groups based on cross-sectional variation in observed exposure levels and apply a matching estimator.

3 Empirical Strategy

3.1 The Ideal Experiment

Before describing the source of variation in speed limits used in this paper, it is instructive to think about what an "ideal experiment" would look like. In such an ideal experiment, one would consider two freeways that are identical in terms of driving characteristics: speed limit, traffic counts by hour-of-day, number of lanes, curvature, slope, driver experience, vehicle type, weather, nature of the surrounding area, etc. On two such freeways, we would expect both the average and any potential time trends in speed, accidents and traffic related pollution to be the same. Let's further assume that the two freeways are neither substitutes nor complements. The ideal experiment would raise the speed limit on one freeway while keeping the speed limit fixed on the other. Then, one would compute the difference of the average travel speed or number of accidents on the treated and untreated freeway, after the speed limit change. In addition, if the background pollution concentrations and population characteristics of the areas surrounding both freeways were the same, we would do a similar comparison of pollution concentrations and health outcomes.

Still, the treatment effect would be local to the type of freeway studied. Comparing two identical rural two-lane highways would yield a consistent estimate of the causal impact of a speed limit change *on such rural two-lane highways*. To estimate effects for freeways of different types, we would need to identify such "ideal pairs" over a wide range of freeway characteristics. This would require a government that assigns speed limit changes randomly over (otherwise similar) freeways.

Of course, such an experiment is not likely to be observed in practice. For good reasons, governments do not randomly assign speed limits. Nevertheless, there exists a natural experiment that allows me to identify the treatment effects with some additional assumptions.

3.2 A Series of Actual Experiments: The National Maximum Speed Law

In response to the First Oil Crisis, Congress adopted the National Maximum Speed Law in 1974, which prohibited speed limits in excess of 55 mph on any highway in the country. Its goal was to reduce oil imports from the Middle East. States had to comply in order to keep receiving federal highway funding. The law was amended in April 1987, when states were allowed to increase the speed limits to 65 mph *on rural interstates only*. Oregon and Washington responded by increasing the speed limit on (virtually) all of their rural interstates; California increased the speed limit on the majority of its qualified freeways. In December 1995, the National Maximum Speed Law was repealed, returning all speed limit authority to the states. California raised the speed limit to 65 mph on another 2,200 highway miles, and in addition raised the speed limit on 1,272 highway miles to 70 mph (these were primarily the segments that had already been raised to 65 mph in 1987). Washington also further increased speed limits. Oregon adopted no further changes. Across all three states, few highways have experienced further changes in their speed limits since 1996.

3.3 Identification

In absence of an ideal experiment, two potential concerns arise when using the amendment and abolishment of the National Maximum Speed Law to identify a causal impact of speed limit changes on travel speed, accidents, pollution and health.

1. *Local treatment effects*: the effect can be estimated for a specific type of highway only and is not necessarily reflective of the effect of the same speed limit change on other highways.

2. *Non-random variation*: speed limits can change in a non-random way, e.g., they only increase on highway segments that are considered safe.

The first issue applies mostly to the 1987 speed limit changes. These happened on rural interstates only. Therefore, we cannot be certain that the estimated impact of these changes is representative for, e.g., urban highways. Nevertheless, this local treatment effect is a particularly interesting one: rural highways are likely candidates to experience (further) speed limit increases today, especially those still posted at 55 mph. In 1996, speed limits increased on a more diverse range of rural and urban highways.

Regarding the second issue, the 1987 speed limit changes actually provide a source of almost random variation: rural interstates were allowed higher speed limits, whereas rural principal arterials and urban highways must be kept at 55 mph. The Federal Highway Administration (FHWA) classifies both interstates and principal arterials (mainly federal – "US" – highways) as highways that "serve corridor movements having trip length and travel density characteristics indicative of substantial statewide or interstate travel" (FHWA, 1989). I now discuss why the distinction between these road types is somewhat arbitrary in many respects.

The number of highway miles that states can designate as interstates is limited, and de facto rationed. States can apply to add a principal arterial to the interstate system. To obtain approval, a road needs to be a "logical addition" to the interstate system from a national defense perspective, and meet all interstate standards (e.g., sufficient length, connections to other interstates on either end, and safety and environmental standards). If a principal arterial is deemed to duplicate an existing interstate, inclusion will be denied. The year of construction and national defense reasons, as opposed to safety standards, often determine why one highway is designated as an interstate while another similar highway is not. In fact, some highway miles that are ineligible to become interstates (because of rationing) are signed as "non-chargeable" interstates, to avoid driver confusion. Also, strict design standards can make it undesirable for states to turn principal arterials into interstates. If a highway meets all interstate standards but has a short segment without full access control (e.g., US-101 near Salinas, CA), inclusion will be denied. Since adding interstates hardly increases federal highway funding and limits the state to add new access points, states do not always try to add likely candidates as interstates (FHWA, personal communication, 26 August 2011). These examples illustrate that the distinction between interstates and principal arterials can be quite arbitrary.

Therefore, rural principal arterials share many important characteristics with rural interstates. Both road types had a 55 mph speed limit before 1987, are designed for speeds at or above 70 mph,

and are typically divided highways with multiple lanes and full or at least partial access control. Rural principal arterials have fewer lanes than rural interstates on average, but wide shoulders make driving conditions similar. Urban highways are even more similar to rural interstates in terms of access control and number of lanes, but face a higher traffic density.

Unfortunately, there is only limited data to verify to what extent rural interstates differed from rural principal arterials and urban highways in 1987. For example, Washington has no reliable data before 2004 on vehicle miles traveled (VMT) by highway, which makes it impossible to compare traffic densities. Although these roads obviously share many similarities, Table 1 demonstrates that there was at least one observable difference: accident rates.

Table 1: Accident Rates by Road Type Before the 1987 Speed Limit Change (Oregon)

	Adopted 65 mph (rural interstates)	Retained 55 mph (rural principal arterials)	Retained 55 mph (urban highways)
Fatal accidents	24	130	26
per 100 million VMT	0.82	3.76	0.46
Incapacitating accidents	111	411	191
per 100 million VMT	3.83	11.91	3.37
Non-incapacitating accidents	240	813	520
per 100 million VMT	8.30	23.57	9.16
Total accidents	1,010	3,412	3,143
per 100 million VMT	34.94	98.86	55.34
AADT per lane	3,554	1,823	5,091
VMT (million)	2,892	3,451	5,567

Notes: Annual averages over 1985-1987. Oregon is chosen because of data availability. AADT = average annual daily traffic (supplied by ODOT). VMT = vehicle miles traveled (Federal Highway Statistics).

Table 1 shows that rural interstates have lower accident rates than rural principal arterials. Non-fatal injury rates are similar across rural interstates and urban highways, but fatal accidents are more likely on rural interstates while non-injury (property damage only) accidents are more likely on urban highways. These differences are similar to those reported by Ashenfelter and Greenstone (2004) for all states that adopted the new 65 mph speed limit.

I take these observable differences in 1987 for granted and state the conditions under which a causal treatment effect is identified. Sections 5-9 present several tests of the plausibility of these assumptions. A naive identification approach would consider the highways where the speed limit changed, and compare speed, accidents, pollution and health outcomes before and after the change. One might be concerned about trends that affect all highways, such as a gradual decline in fatal accidents because of improved vehicle engineering. In such cases, a single difference estimator will be biased. A difference-in-differences estimator uses a treatment and a control group to account for statewide trends. For speed and accidents, the treatment group consists of rural interstates and the control group consists of (subsets of) rural principal arterials and urban highways. For pollution and health, I compare treatment areas close to highways where the speed limit changed with control areas further away. I assume that treatment and control groups would have followed the same trend in absence of the speed limit changes:

Identification Assumption 1: (Speed, pollution, health) Absent the policy change, there would have been equal time trends *in levels* for treatment (T) and control (C) highways:

$$E[speed_1^T|\Delta SL = 0] - E[speed_0^T] = E[speed_1^C] - E[speed_0^C] \tag{1}$$

where ΔSL denotes the speed limit change, 0 corresponds to the pre-period ($t < t_{\Delta SL}$) and 1 to the post-period ($t \geq t_{\Delta SL}$). This is the standard identification assumption for linear difference-in-differences estimators. The expressions for pollution and health are analogous, but the treatment group is now defined as an area close to the treatment freeways.

Identification Assumption 2: (Accident rates) Absent the policy change, there would have been equal time trends *in ratios* for T and C highways:

$$E\left[\frac{accidents_1^T/VMT_1^T}{accidents_0^T/VMT_0^T}|\Delta SL = 0\right] = E\left[\frac{accidents_1^C/VMT_1^C}{accidents_0^C/VMT_0^C}\right] \tag{2}$$

To assert the reader of the plausibility of these assumptions, Section 5 motivates the choice of identification assumption 2 and presents evidence that there were no differential pre-existing trends in the outcome variables for treatment and control highways. Section 6 demonstrates that the effect on speed is robust when I include only those control highways that are most similar to rural interstates. Importantly, Section 7 shows that the effect on accidents is remarkably similar when I only include control highways with comparable accident rates to the treated highways.

The speed limit changes in 1996 were not as randomly assigned as in 1987, since states faced no federal restrictions and based their decisions on accident histories. Therefore, the identification assumptions of equal trends are less convincing (although Section 6 will show that there were no differential pre-existing trends in speed). Nevertheless, I investigate how changes in the outcome variables are associated with this speed limit change. This is only possible for speed data, since it has proven impossible to collect a comprehensive list of speed limit changes in 1996 beyond the speed limits pertaining to the specific locations of the speed stations. The estimated effect on speed is similar to the estimate using the 1987 speed limit changes.[4]

4 Data

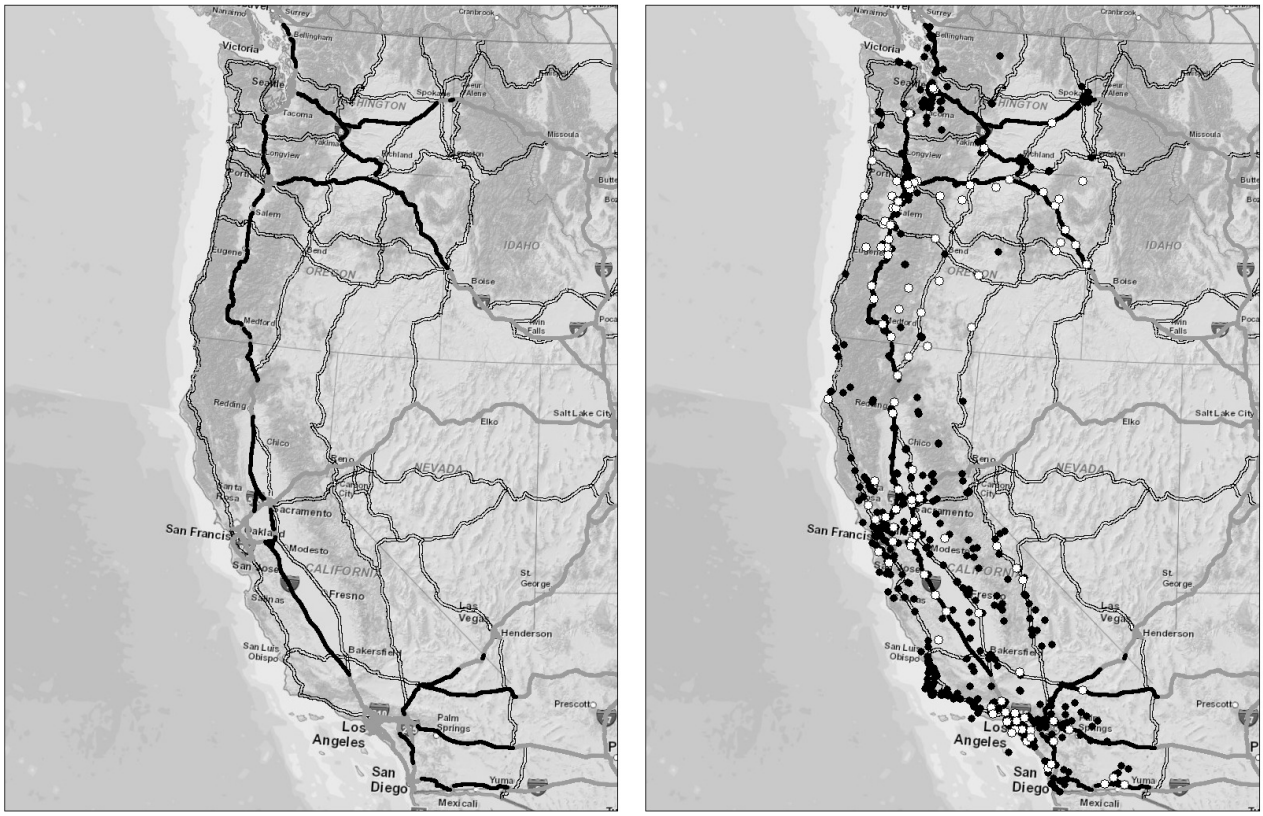
This section summarizes the data sources. Several of these sources were collected using Freedom of Information Act (FOIA) requests. The data collection faced the further challenge that many state agencies do not retain data for more than 10 years. More details are provided in Appendix A.

First, I collected a list of the freeway segments on which the speed limits changed in 1987 from the three Departments of Transportation (CA: Caltrans; OR: ODOT; WA: WSDOT). Following

[4]This may reflect that drivers may not have a strong perception of the safety of a particular segment. Given that most highways had "engineering design speeds" well above 55 mph (typically, 70 mph), drivers may have considered them all relatively safe (Caltrans, personal communication, 28 January 2011).

the April 1987 amendment to the National Maximum Speed Law, California (May 1st), Oregon (December 8th) and Washington (April 23rd) changed the speed limit for 1,157, 604 and 526 rural interstate miles, respectively.[5] Figure 2 (left panel) displays the interstates on which the speed limits increased in 1987, as well as highways with no change in the speed limit.[6] The speed limit changes in 1996 are more difficult to summarize and discussed in Appendix A.

Figure 2: Rural Interstates with Increased Speed Limits in 1987 (Left) and the Distribution of Pollution and Speed Monitoring Stations (Right)



Notes: (Left panel) Gray solid lines indicate interstates. Black solid lines indicate the interstate segments in CA, OR and WA on which the speed limit changed from 55 to 65 mph in 1987. Double black lines are a subsample of principal arterials, on which no speed limit change occurred. (Right panel) Black dots indicate pollution monitoring stations that were active in the period 1984-1990. White dots indicate speed monitoring stations.

Second, I have obtained detailed speed data for a reasonable range of monitoring stations in the period 1994-1998 (CA), 1983-1992 (OR) and 1994-2001 (WA). These data were extracted from archived databases and scanned paper records. Depending on the state, I observe counts by speed bin (e.g., 55-60 mph), by year-month-day-hour, by direction, by lane. California data are available for one month per quarter. Oregon data are available for one day per quarter. The Washington

[5] This corresponds to 56.3%, 97.3% and 100% of all eligible rural miles per state.

[6] Throughout the entire study period, truck speed limits remained at 55 mph in California and Oregon. In Washington, truck speed limits were raised to 60 mph in 1996 on those segments where the car speed limits were raised to 70 mph. I ignore trucks, since they only constitute a small fraction of vehicles (see footnote 27).

data contain speeds for almost all days. I have data for 61 speed stations in California, 48 of which had a speed limit increase (treatment stations). The Oregon data cover 51 stations (12 treatment stations). In Washington, I only have six stations, but well balanced (three treatment stations) and extensive daily coverage throughout the year. Figure 2 (right panel) shows the speed stations.

Third, I requested the universe of accident records since 1985 (OR) and 1980 (WA). Unfortunately, Caltrans has destroyed all accident data older than 10 years.[7] Detailed information on each accident is reported, such as the date, time, type (fatality, incapacitating injury, non-incapacitating injury, property damage), location (highway number and milepost), city, county, type of highway, urban status and a range of road, weather, daylight and driver characteristics. Using the accident location and speed limit change information, I assign each accident a treatment or control status.

Fourth, the California Department of Public Health's birth cohort files (1984-1990) contain infant health information from all birth records, including birth weight, gestational age, infant deaths in the first year of life and fetal deaths in the second or third trimester of the pregnancy. The files also contain a large number of characteristics of the child, mother and father, as well as medical information about the pregnancy. I use zip code information to approximate the mother's residence during pregnancy, and use ArcGIS to calculate the distance between the zip code's population-weighted average centroid and the closest highway segment where the speed limit changed.

Fifth, daily measurements for CO, NO_2, NO_x, O_3 and PM_{10} were obtained from the California Air Resources Board, the Oregon Department of Environmental Quality, and the Washington Department of Ecology. Figure 2 (right panel) plots the 431 stations that reported emissions for some part of the period 1984-1990. I calculate the distance between each air pollution monitoring station and the closest point on a highway segment where the speed limit changed. Stations are located at various distances from such highways, but only occasionally right next to them. 31 stations were located within three miles of these highways and reported both before and after the speed limit changes (treatment stations), 42 within five miles and 69 within 10 miles. Since few stations monitor all five pollutants, the number of treatment stations by pollutant is more limited.

Finally, I obtained weather data from the National Climatic Data Center's "Global Summary of the Day" (1980-2010). Each weather station in California, Oregon and Washington reports average, maximum and minimum temperature, precipitation, wind speed, plus indicator variables for rain, fog, snow, hail, thunder or a tornado. I follow Knittel *et al.* (2010) and assign each pollution/speed station a weather variable: the inverse distance-weighted average of observations from all weather stations within a 20 mile radius.

[7] The National Highway Traffic Safety Administration has retained California's accident records since 1988, but has removed precise location information. This renders the data set unsuitable for the purposes of this paper.

5 Exploring the Identification Assumptions

5.1 Speed

In Section 3 I discussed that there are certain observable differences between treatment and control highways. A potential concern is that states chose to raise speed limits on freeways where the average speed was already high, to legalize the prevailing speed. This may reduce the treatment effect. Table 2 shows that California and Oregon did indeed raise the speed limit on highways with an average travel speed of about 3-4 mph above the highways that remained at 55 mph. This difference is non-existent for Washington. However, Figure 3 shows that there were no differential pre-existing speed trends between the treatment and control groups in any of the three states. This lends credibility to identification assumption 1 (Section 3). The graphs for California and Oregon reveal a statewide time trend for speed. This underscores the importance of a difference-in-differences approach as opposed to a single difference estimate using the treatment stations only.

Table 2: Speed Data Summary Statistics Before the Speed Limit Changes

California (1996)	65 to 70 mph	55 to 65 mph	Retained 55 mph	
Average speed (mph)	65.2	61.7	57.9	
Oregon (1987)	55 to 65 mph	Retained 55 mph		
Average speed (mph)	60.5	57.1		
Washington (1996)	55 to 60 mph	Retained 55 mph	65 to 70 mph	Retained 50 mph
Average speed (mph)	59.7	59.9	68.0	49.3

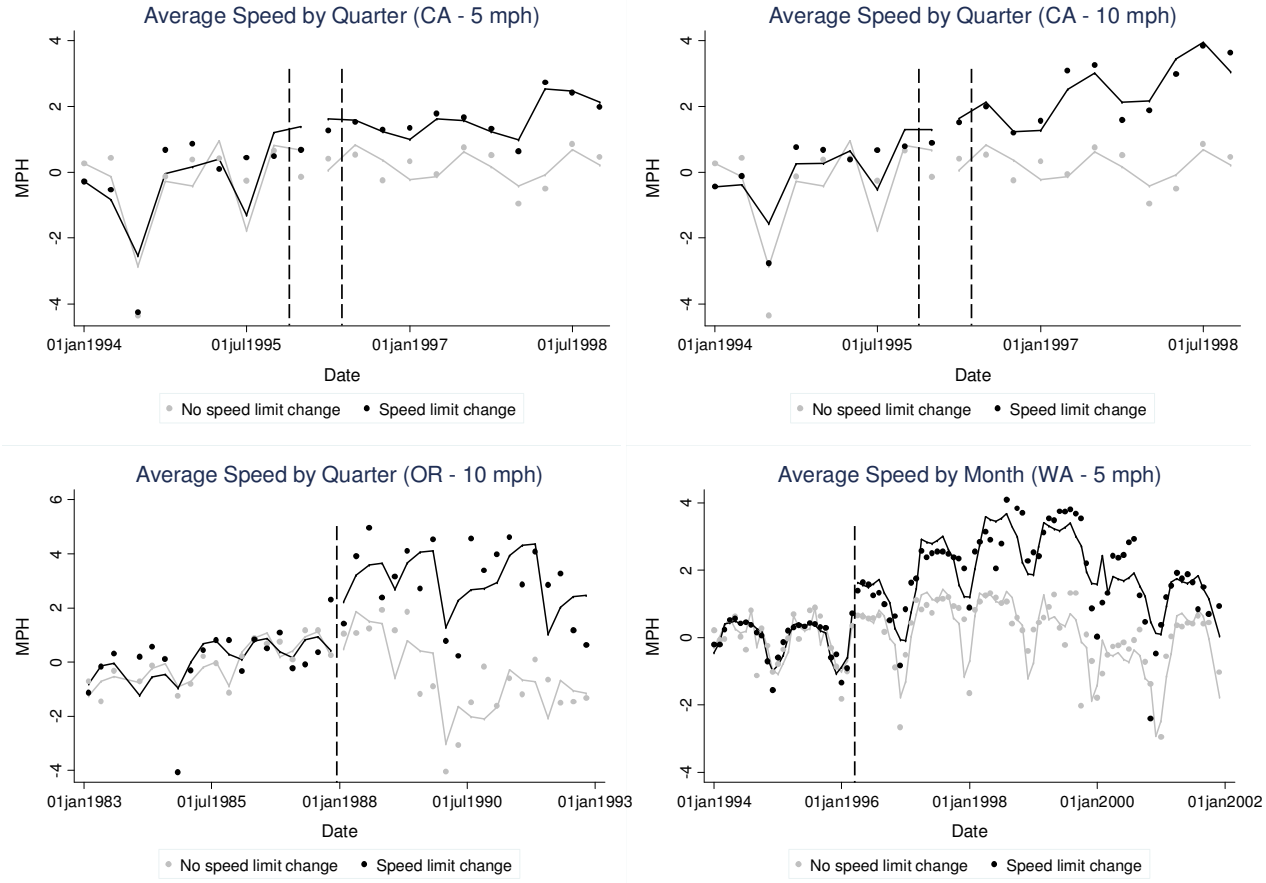
Notes: All statistics are annual averages over the period before the speed limit changes (typically, 2-3 years). Average traffic density in number of vehicles per hour per lane for the control and treatment stations is 285.2 and 347.8 (CA), 138.2 and 145.1 (OR), and 379.2 and 462.4 (WA).

5.2 Accidents

Table 3 presents summary statistics for accidents and VMT for Oregon and Washington to explore the validity of identification assumption 2. The table shows – not adjusting for control variables – that the increase in fatal accidents is highest on rural interstates, both in absolute terms and relative to the road types on which speed limits remained at 55 mph (rural principal arterials and urban highways). The increase in total accidents is highest for rural interstates in relative, but not absolute, terms. Given the large differences in VMT, a fair comparison is based on relative changes. Moreover, since I want to isolate the effect of the speed limit changes from changes in accidents due to time trends in VMT, I compare accident rates (accidents per vehicle mile traveled). This motivates identification assumption 2, which suggests a comparison of relative changes in accident rates.[8]

[8] A comparison of relative changes in accidents instead of accident rates gives similar results. Analyzing accidents per vehicle mile traveled also largely deals with a potentially confounding trend: substitution of traffic from control highways to rural interstates after the speed limit changes. However, even after dividing accidents by VMT, one may still be concerned that traffic substitution makes the treatment roads more congested. If the accident rate per mile is an increasing function of traffic density, an increase in accidents would be the combined effect of the higher speed and the higher traffic density. Section 6 shows that these concerns are of limited importance. First, there is no or hardly any traffic substitution. Second, the effect on travel speed is not affected by controlling for traffic density.

Figure 3: Trends in Speed Before and After the Speed Limit Increases in California (1996), Oregon (1987) and Washington (1996)



Notes: Gray dots indicate average speed for control stations, black dots for treatment stations. Speeds are relative to the pre-treatment average speed. Solid lines are predicted values from regressions of the average speed on year and month(quarter)-of-year dummies. Vertical dotted lines indicate the time of the speed limit change (OR, WA), or the period in which speed limits changed (CA). Panel titles indicate the size of the speed limit change (5 or 10 mph).

Figure 4 graphically illustrates that such a relative comparison is indeed reasonable. The figure plots trends in the accident index (defined as the ratio of the monthly accident rate and the pre-treatment average monthly accident rate). For all accident types, the accident index for treatment highways shifts up relative to the index for control highways. Moreover, the figure shows that there are no differential pre-existing trends in any of the accident indices between the treatment and control groups. This provides suggestive evidence that identification assumption 2 is plausible.

There is considerable variation in the total number of accidents per year. This can to a large extent be explained by the severity of winters: the majority of accidents occurs between November and February. I will control for weather conditions, and present a robustness check to demonstrate that the results are not driven by differential weather impacts (Section 7). Also, I use within-state comparisons, so that weather conditions will be more similar than in cross-state studies.

Table 3: Summary Statistics for Accidents Data (Oregon & Washington Combined)

	Before speed limit change			After speed limit change		
	-3 Years	-2 Years	-1 Years	+1 Years	+2 Years	+3 Years
	Rural interstates (speed limit increased from 55 to 65 mph)					
Fatal	41	62	44	71	70	77
Incapacitating	244	281	249	278	304	295
Non-incapacitating	644	622	557	657	677	717
Total	3,213	3,173	2,814	3,331	3,775	3,713
	Rural principal arterials (no speed limit change)					
Fatal	210	195	180	187	201	179
Incapacitating	781	693	643	575	573	646
Non-incapacitating	1,554	1,492	1,268	1,349	1,391	1,352
Total	7,152	6,858	6,154	6,145	6,667	6,377
	Urban highways (no speed limit change)					
Fatal	75	69	68	58	57	72
Incapacitating	602	601	508	473	543	514
Non-incapacitating	1,954	1,855	1,521	1,498	1,769	1,720
Total	12,536	12,666	10,659	10,680	12,789	12,560
	Vehicle miles traveled					
Rural interstate	5,426	5,504	6,075	6,402	6,747	6,971
Rural principal arterial	6,020	6,262	6,803	6,638	6,659	7,105
Urban highway	13,469	13,818	13,722	15,202	15,745	16,640

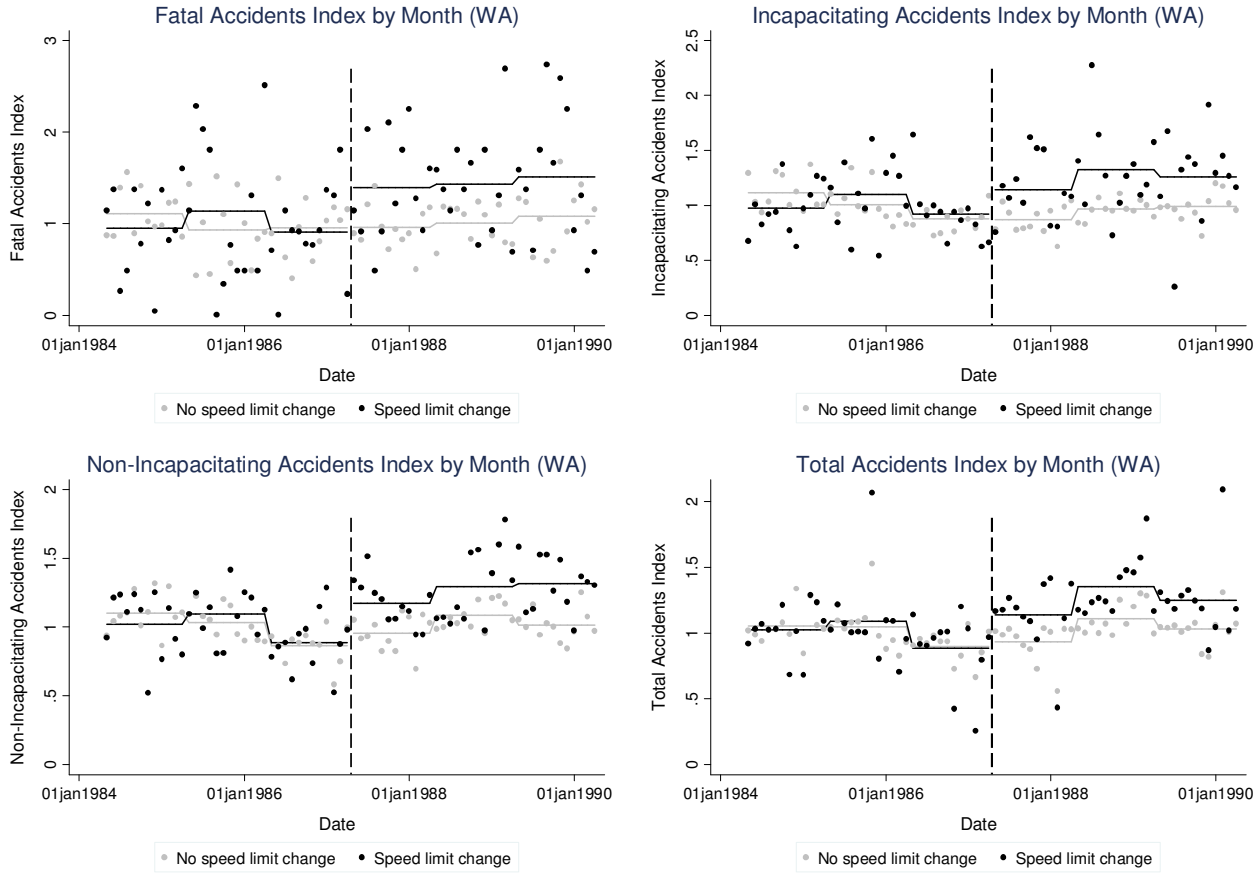
Notes: Count of accidents are for the period Jan 1985 - Dec 1990 (OR) and May 1984 - Apr 1990 (WA), representing three years before and after the speed limit changes in these states. VMT is expressed in millions of miles per year and are obtained from the Federal Highway Statistics (several editions).

5.3 Infant Health and Air Quality

As discussed in Section 3, a naive approach to identify the effect of an increase in speed on infant health (or air pollution) would focus on health outcomes for infants from mothers who live close to the treatment highways (or pollution concentrations close to the treatment highways). If socioeconomic and health characteristics of parents and infants can be credibly controlled for, and in the absence of statewide health trends, a single difference between infant health outcomes before and after the speed limit changes should suffice. Similarly, if one controls for weather conditions that affect the spreading of pollution, a single difference approach should suffice in the absence of statewide pollution trends. However, Table 4 shows that there are trends for some infant health outcomes and pollutant concentrations. There is a slight declining trend in the probability of a fetal death and infant mortality. CO, NO_2 and NO_x decrease over time. This motivates the use of a difference-in-differences approach (identification assumption 1). I therefore distinguish between mothers living in treatment zip codes (centroid within 3 or 5 miles from the treatment freeways) and in control zip codes (centroids further away), and a similar geographic treatment-control definition based on the location of the pollution monitoring station (see Section 8 for details).

To give further credibility to the difference-in-differences identification assumption, Figures 12 and 13 in Appendix A show that there are no differential pre-existing trends in air pollution and infant health. Moreover, Table 20 in Appendix A contains summary statistics of the health outcomes and control variables, for various definitions of treatment and control zip codes. Mothers living close to the relevant highways have comparable birth outcomes to mothers living further

Figure 4: Trends in Accidents Before and After the Speed Limit Increases in Washington (1987)



Notes: Gray dots indicate the accident index (defined as the ratio of the monthly accident rate and the pre-treatment average monthly accident rate) for control highways, black dots for treatment highways. All data are de-seasonalized at the monthly level. Solid lines are predicted values from regressions of the accident indices on year dummies. Vertical dotted lines indicate the time of the speed limit change in Washington (23 April 1987). For reasons of clarity, results are shown for Washington only to reflect a single treatment date.

away. They are also quite similar in terms of other observable characteristics, except for a few differences in racial composition: children born within a three mile zone around treatment freeways are somewhat more likely to be Hispanic, and less likely to be black or Asian. Also, the three mile zone is less urban than the surrounding areas.[9] The various zones are similar in terms of education, age, prenatal care, insurance, unemployment, poverty and housing. Comparing areas with similar poverty and unemployment rates is important since the poor are the most likely to lack access to adequate prenatal care, have higher rates of preventable infant or fetal mortality, and respond differently to pollution increases. The similarity on observables of treatment and control zip codes, combined with the absence of differential pre-existing trends, provides suggestive evidence that the "equal trends" assumption in the difference-in-differences framework is reasonable.

[9]The reason is that some of the treatment highway segments end relatively close to cities, and the wider buffers capture a share of the population living in city suburbs.

Table 4: Summary Statistics for Infant Health and Pollution Data: Time Trends

	1984	1985	1986	1987	1988	1989	1990
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Health outcomes (California)						
Fetal deaths (per 1000)	7.1	6.9	7.0	7.1	6.7	6.9	6.7
Infant deaths (per 1000)	9.5	9.8	9.1	9.3	8.8	8.8	8.0
Birth weight (grams)	3,374	3,377	3,377	3,374	3,374	3,369	3,375
Low birth weight (per 1000)	59.4	60.3	59.8	60.5	60.2	61.2	58.0
Gestational age (days)	278.9	278.4	278.1	277.7	277.7	277.5	277.3
Premature birth (per 1000)	5.8	6.3	6.5	6.7	6.5	6.6	6.1
Number of births	451,915	475,545	487,036	508,739	537,897	574,991	617,742
	Pollution outcomes (California, Oregon & Washington combined)						
CO (ppm)	1.385	1.387	1.323	1.225	1.231	1.274	1.195
NO_2 (ppb)	26.03	26.28	24.12	22.88	22.76	22.59	20.91
NO_x (ppb)	51.08	50.33	45.04	42.41	42.01	43.24	39.57
O_3 (ppb)	26.46	27.70	28.12	30.29	31.25	29.98	28.51
PM_{10} $(\mu g/m^3)$	36.07	54.71	45.87	42.56	40.56	40.00	35.89

Notes: Health outcomes: annual statewide means of the health related outcome variables from the California birth cohort files. Pollution outcomes: annual means of the daily air pollutant concentrations.

6 The Effect of Speed Limit Changes on Travel Speed

6.1 Econometric Framework

I estimate a number of flexible specifications, taking into account that the effect of a speed limit change on speed builds up over time (Figure 3). In fact, the effect may consist of an immediate impact at the time of the speed limit change, and a – potentially time-varying – longer term impact. Under identification assumption 1 (Section 3), I first estimate the latter effect using the following linear difference-in-differences specification:

$$speed_{ijth} = \beta_0 + \beta_1 1\left(i \in T\right) * 1\left(t \geq t_{\Delta SL}\right) * TI + \beta_2 X_{it} + \theta_i + \theta_j + \theta_h + \theta_d + \theta_m + \theta_y + \varepsilon_{ijth} \quad (3)$$

for speed station i, lane-direction j, date t, hour-of-day h, day-of-week d, month-of-year m and year y. Control variables include weather conditions (maximum, minimum and average temperature, wind speed, fog, rain, snow, hail, thunder, tornado) and a measure of traffic density (number of vehicles per lane per hour).

One potential concern is that the speed limit increases caused substitution of traffic towards faster roads. This would make treatment highways more congested, and reduce the effect on speed. In addition, traffic density would be an outcome of the treatment and should not be included as a control. I test for traffic substitution directly by estimating (3) with the number of vehicles as the left-hand side variable (see Table 22 in Appendix B). For all three states, I find that the coefficients on the interaction term are small and statistically insignificant at the 10% level: 5.49% (s.e. 3.42%) in California, 0.02% (s.e. 11.86%) in Oregon, and 0.20% (s.e. 8.70%) in Washington. Treatment and control roads do not appear to be strong substitutes. One reason is that they are often quite far apart, and not many drivers are at the margin between choosing one over the other.

Since California had speed limit changes from 55 to 65 mph and from 65 to 70 mph, the treatment intensity varies: $TI = 1$ if $\Delta SL = 10$ and $TI = 0.5$ if $\Delta SL = 5$. This assumes an equal treatment for every mph increase in the speed limit. I also estimate separate coefficients for the two speed limit changes. Moreover, I estimate separate treatment effects for different traffic conditions, e.g. by hour-of-day (replacing β_1 by β_{1h} in equation (3)).

Finally, I estimate the immediate effect of a speed limit change using a time discontinuity on the treatment stations, while flexibly controlling for other time-varying factors using a time polynomial:

$$\begin{aligned} speed_{ijth} = \beta_0 + \beta_1 1\left(t \geq t_{\Delta SL}\right) * TI + \beta_2 X_{it} + f(t, t^2, ..., t^n) \\ + \theta_i + \theta_j + \theta_h + \theta_d + \theta_m + \theta_y + \varepsilon_{ijth}, \qquad i \in T \end{aligned} \tag{4}$$

6.2 Travel Speed Results

Tables 5 and 6 show the main results for the speed limit changes in California (1996), Oregon (1987) and Washington (1996).

Table 5: Regression Results for the Effect of Speed Limit Changes on Travel Speed

	(1)	(2)	(3)	(4)	(5)
LHS variable	$speed_{ijt}$	$speed_{ijt}$	$speed_{ijth}$	$speed_{ijth}$	$speed_{ijth}$
Speed limit	3.05***	3.03***	3.10***	3.06***	2.98***
(CA 1996)	(0.53)	(0.53)	(0.52)	(0.51)	(0.50)
	[0.84]	[0.85]	[0.80]	[0.80]	[0.78]
Observations	15,048	15,048	356,661	356,661	356,661
Speed limit	3.93***		4.09***		4.10***
(OR 1987)	(0.55)		(0.46)		(0.46)
	[0.75]		[0.76]		[0.77]
Observations	732		19,103		19,103
Speed limit	3.69***	3.61***	3.69***	3.60***	3.54***
(WA 1996)	(0.58)	(0.59)	(0.61)	(0.63)	(0.50)
	[0.73]	[0.77]	[0.67]	[0.67]	[0.54]
Observations	24,289	24,289	1,371,156	1,371,156	1,371,156
Fixed effects					
Station	Y	Y	Y	Y	Y
Direction	Y (CA, WA)	Y (CA, WA)	Y (CA, WA)	Y (CA, WA)	Y (CA, WA)
Lane	Y (WA)	Y (WA)	Y (WA)	Y (WA)	Y (WA)
Station-direction	N	Y (CA, WA)	N	Y (CA, WA)	Y (CA, WA)
Year	Y	Y	Y	Y	Y
Month	Y	Y	Y	Y	Y
Day-of-week	Y	Y	Y	Y	Y
Hour-of-day	N	N	Y	Y	Y
Controls	All	All	All	All	Weather only

Notes: The coefficient on the interaction term of 13 separate regressions is reported. Coefficients are normalized to reflect the effect of a 10 mph speed limit change. The dependent variable is travel speed. Standard errors in parentheses clustered at the station by year level in (), and clustered at the station level in [] (used for stars). The time window is 1994-1998 (CA), 1983-1992 (OR) and 1994-2001 (WA). ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Table 5 shows the treatment effect of a 10 mph speed limit increase in California, Oregon and Washington.[10] The table reveals two interesting results. First, the effect is quite stable across states

[10]Standard errors are clustered at the station by year level and at the station level. Clustering at the station level

(a 3-4 mph increase in travel speed for a 10 mph speed limit increase), even though the speed limits were not all raised by the same amount, from the same original speed limit, and at the same point in time. This is reassuring and suggests that the estimated coefficient is representative of a range of speed limit changes between 55 and 70 mph. The results are robust to the inclusion of station-direction fixed effects (columns (2) and (4)) and to aggregation to the daily level (columns (1) and (2)). This is to be expected if the experiment is clean and the panel is well-balanced. Second, the inclusion of traffic density per lane per hour hardly changes β_1 (column (5)). This again suggests that traffic substitution towards roads with increased speed limits is not a major issue. Finally, the estimates are considerably below 10 mph. To see why this might not be surprising, consider two driver types: potential responders and non-responders. Next, divide driving conditions into constrained (e.g., congestion, poor visibility, night time) and unconstrained (e.g., uncongested, good visibility). If potential responders' speed choices are initially constrained by the speed limit, a higher limit would induce them to driver faster at a rate of 1 mph for 1 mph at the margin. However, potential responders' speed will not increase if they are constrained by driving conditions. Non-responders were not initially constrained by the speed limit: their optimal speed choice was below the speed limit, or they ignored the speed limit altogether. Only unconstrained potential responders contribute to a positive treatment effect. This effect is therefore well below 10 mph.

Table 6 reports the effect of the speed limit changes on the distribution of travel speed. The results reveal – consistently across the three states – that the average and 85th percentile speed increase by approximately the same amount. This suggests that most drivers are potential responders that are constrained by driving conditions for a significant fraction of their time: they drive "with the flow of traffic". This result is important, since not only an increase in average speed but also in speed variance at higher speeds could cause more accidents. Table 6 demonstrates that the variance channel is unlikely to drive the accidents results in Section 7. The impact on the unconditional speed variance is negligible in California, and small and statistically insignificant in Washington. However, the variance is imprecisely measured since the lowest speed bin is truncated at 30 mph or below.

Column (4) reports two robustness checks. First, when the coefficients on the two speed limit changes in California (55 to 65 mph and 65 to 70 mph) are not constrained to be equal, the resulting estimates are almost identical. Second, when I only include rural control highways in Oregon, the speed increase is slightly lower.[11]

increases the standard errors, but does not affect the conclusions about statistical significance. The station level is relevant if one is concerned about unobserved shocks to all observations within a station, allowing for correlation of the error term across time, lane and direction. A particular concern is that some stations could have been located on segments with long-term road construction, which not only reduces average speed (captured by the station fixed effect) but also the response to speed limit changes (which is likely more limited in construction zones). However, since road construction is typically a shorter-term (e.g., several weeks or months) phenomenon, clustering at the station by year level is likely to be conservative enough while mitigating concerns about a paucity of clusters.

[11] I performed two additional checks, which are not reported in Table 6. First, I ran a *single* difference on the treatment stations only. This gives broadly similar results: 3.45 (CA: 55 to 65 mph), 3.50 (CA: 65 to 70 mph; adjusted), 3.34 (OR) and 2.63 (WA). However, the single difference estimation does not account for statewide time trends in speed (see Section 5.1). Second, I did a placebo experiment in which specification (3) is estimated as if the speed limit change occurred on 1 January 1992, ..., 1996. I should find that the coefficients are insignificant and close

Table 6: Speed Regression Results: Distribution and Robustness

		(1)	(2)	(3)	(4)
LHS variable		$speed_{ijth}$	$speed85p_{ijth}$	$var(speed)_{ijth}$	$speed_{ijth}$
Speed limit	CA 1996	3.06***	3.12***	-0.81	
		(0.51)	(0.56)	(4.04)	
		[0.80]	[0.87]	[5.71]	
Speed limit	CA 1996				3.11***
(65 to 70 mph)					(0.82)
					[1.24]
Speed limit	CA 1996				3.08***
(55 to 65 mph)					(0.52)
					[0.80]
Observations		356,661	356,661	356,501	356,661
Speed limit	OR 1987	4.09***	4.10***		
		(0.46)	(0.45)		
		[0.76]	[0.81]		
Speed limit	OR 1987				2.96***
(U.S. highways)					(0.68)
					[1.00]
Speed limit	OR 1987				3.18***
(Rural arterials)					(0.49)
					[0.81]
Observations		19,103	19,103		6,962; 11,008
Speed limit	WA 1996	3.60***	3.72***	5.58	
		(0.63)	(0.69)	(3.54)	
		[0.67]	[0.86]	[3.77]	
Observations		1,371,156	1,370,345	1,350,409	
Fixed effects					
Station		Y	Y	Y	Y
Direction		Y	Y	Y	Y
Lane		Y (WA)	Y (WA)	Y (WA)	Y (WA)
Station-direction		Y (CA, WA)	Y (CA, WA)	Y (CA, WA)	Y (CA, WA)
y, m, d, h		Y	Y	Y	Y

Notes: The coefficient on the interaction term of 11 separate regressions is reported. Coefficients normalized to reflect the effect of a 10 mph speed limit change. The dependent variable is the travel speed. Standard errors in parentheses clustered at the station by year level in (), and clustered at the station level in [] (used for stars). The time window is 1994-1998 (CA), 1983-1992 (OR) and 1994-2001 (WA). All specifications contain controls. No variance regressions are shown for Oregon, since the low and high speed bin definition changed discontinuously around 1988. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

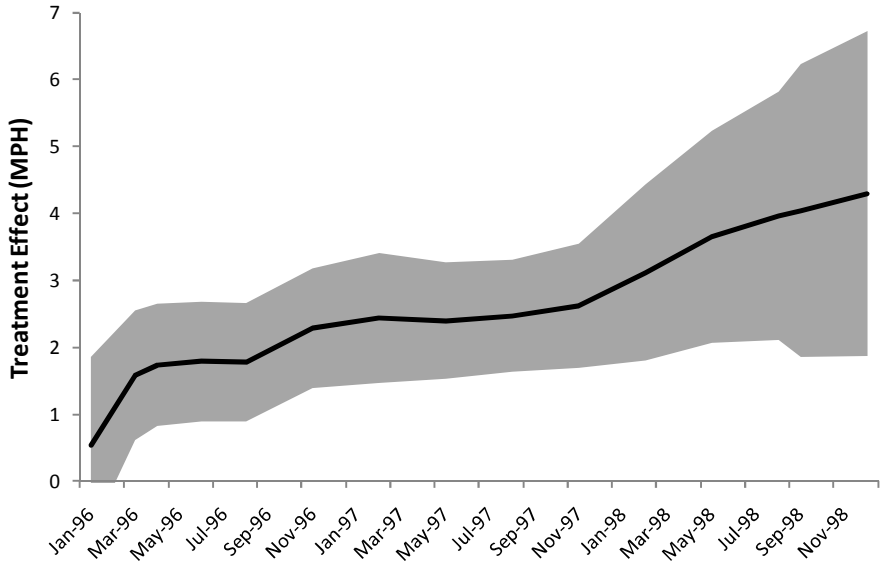
Figure 3 suggests that the treatment effect is not constant over time. I now test this more formally (using CA data[12]) by estimating (3) using the pre-treatment data and only up to one year of post-treatment data. Towards the beginning and end of the post-treatment interval, I use shorter time windows (Figure 5).

Figure 5 shows that the treatment effect is increasing over time. The initial effect is estimated using the time discontinuity specification (4) with a flexible eighth-order time polynomial. The small initial effect could be explained by the fact that not everyone noticed the new signs immediately,

to zero, since no speed limit change occurred in reality. This experiment is only possible for Oregon, since the speed data for California and Washington do not cover a long enough period. Using a symmetric eight year window around the five hypothetical treatment dates, I indeed find small and insignificant (at the 5% level) treatment coefficients: 0.86 (1992), 0.56 (1993), -0.06 (1994), -0.63 (1995) and 0.41 (1996) mph.

[12]Results for OR and WA are similar and available upon request.

Figure 5: Time-Varying Treatment Effects (California)



Notes: This figure shows the treatment effect of various regressions using the pre-treatment data and (up to) a symmetric one-year window of post-treatment data. The estimate for January 1996 is from the time discontinuity regression (4) with a flexible eight-order time polynomial. The gray area represents a 95% confidence interval.

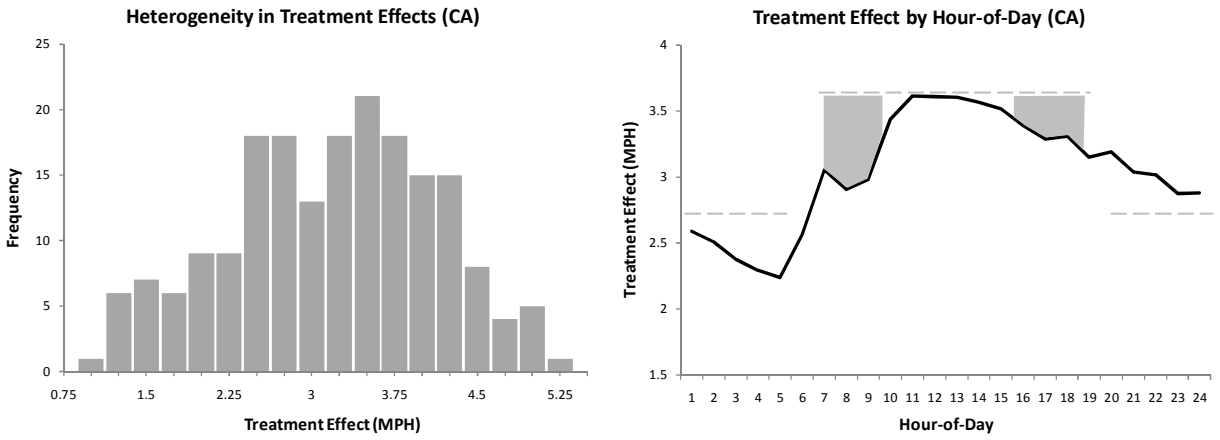
and that the official date of change is not necessarily equal to the date at which the new signs were erected.[13] The latter explanation might bias downwards the time discontinuity estimate. The increasing treatment effect over time reflects adaptation towards the new driving conditions. Similar regressions for Oregon and Washington show that after four years, the treatment effect becomes somewhat smaller. This may be driven by a catch up of speed on other highways and could suggest that drivers indeed perceive these control roads as similar to the treatment roads.

I now estimate heterogeneity in the treatment effect. The distibution of the treatment effects is useful for the cost-benefit analysis since fuel consumption is a non-linear function of speed. These results will also be useful to evaluate flexible speed limits that adjust by time-of-day and road conditions, as used in some European countries. I estimate heterogeneity by allowing for flexible treatment coefficients β_{1uswh} in specification (3), where $u \in \{urban/rural\}$, $s \in \{dry/wet\ road\ surface\}$, $w \in \{weekend/weekday\}$, and h indicates the hour-of-day. This leads to the estimation of $2 * 2 * 2 * 24 = 192$ treatment effects (Figure 6).

Figure 6 (left panel) shows that the treatment effect varies between 1-5 mph across different driving conditions and hours. The average treatment effect is larger for urban than for rural roads (3.60 vs. 2.50 mph), for dry than for wet roads (3.26 vs. 2.82 mph), and for weekends than for weekdays (3.24 vs. 2.84 mph). The larger treatment effect for urban roads likely reflects higher enforcement on urban roads, which means that more drivers were constrained by the original speed limit. Similarly, drivers are more likely to be constrained by the speed limit on dry roads than on wet roads. The larger effect for weekends may reflect less congested driving conditions (i.e., more

[13]This information is not retained in the records of the Departments of Transportation.

Figure 6: Heterogeneity in the Treatment Effect (California)



Notes: This figure shows the heterogeneity of the treatment effect in California, where separate treatment effects are estimated along the following dimensions: urban/rural, dry/wet road surface, weekday/weekend, and hour-of-day. A histogram of all 192 treatment effects is presented in the left panel. The right panel shows the average treatment effect by hour-of-day.

opportunities for "potential responders" to increase their speed).[14]

The right panel shows that the average treatment effect varies by the hour-of-day. During daylight conditions, the treatment effect is generally larger (upper dotted gray line) than during dark hours (lower dotted gray lines). During the morning and afternoon rush hour, the increased probability of congestion causes the treatment effect to be below the "typical" daylight effect. One might have expected that the treatment effect during rush hours would be even below the night time treatment effect. An explanation could be that rush hour drivers (commuters) are disproportionately likely to be "potential responders", since they face penalties for being late at work or want to be back home as soon as possible. This counteracts the effect of increased congestion.

In summary, I find that a 1 mph speed limit increase leads to a 0.3-0.4 mph increase in travel speed. This result is similar across the three states, and for speed limit changes of various intensities between 55 and 70 mph. In all three states, speed limit changes lead to a parallel shift in the speed distribution at average and high speeds. Having established a positive effect on travel speed, I now proceed to estimating various costs related to this increase in speed.

7 The Effect of Speed Limit Changes on Accidents

This section tests two possible hypotheses about the effects of increased speed limits on accidents. The first hypothesis is that traveling at higher speed leads to an increase in the *amount* of accidents. The second hypothesis is that a higher speed causes a shift towards *more severe* accidents.

[14]Not all these effects are statistically significantly different from each other, although a joint F-test of equal parameters is easily rejected (p-value = 0.0000). The average standard error is 0.82, and the differences between the lower and upper end of the distribution are statistically significant.

7.1 Econometric Framework

To test the first hypothesis, I use count data models to estimate the impact of the 1987 speed limit changes in Oregon and Washington on the number of various types of accidents (fatality (*fatal*), incapacitating injury (*inc*), non-incapacitating injury (*non-inc*), property damage only (*pd*)). To test the second hypothesis, I use ordered choice models.

Accidents are discrete events and a natural application of count data models. Moreover, under identification assumption 2 (Section 3) and given the graphical evidence provided in Section 5, it is reasonable to employ a "ratio-in-ratios" estimator. Count data models possess that feature.[15] The two dimensions for the ratio-in-ratios estimator are time (before and after the speed limit change), and the type of highway (treatment: rural interstates vs. control: rural principal arterials and (subsets of) urban highways).

I use two non-linear models for count data: the Poisson model and the negative binomial model.[16] Both are estimated by maximum likelihood. The negative binomial model is more flexible than the Poisson model since it allows for overdispersion[17] instead of equidispersion (footnote 16). It has the following expressions for the conditional mean and variance:

$$\begin{aligned} E(y|X) &= \lambda = exp(X'\beta) \\ Var(y|X) &= \lambda + \alpha^{-1}\lambda^2 = exp(X'\beta) + \alpha^{-1}exp^2(X'\beta) \end{aligned} \tag{5}$$

where $\alpha^{-1} = \sigma^2 > 0$.[18] I estimate the following specification:

$$E(\frac{a_{ijt}}{vmt_{jy}}|X) = exp\left(\beta_0 + \beta_1 1(j = rural\ interstate) * 1(t \geq t_{\Delta SL}) + \beta_2 X_{jt} + \theta_d + \theta_m + \theta_y + \theta_j\right) \tag{6}$$

where a_{ijt} represents the number of accidents of type $i \in \{fatal,\ inc,\ non\text{-}inc,\ pd\}$ on highway type $j \in \{rural\ interstate,\ rural\ principal\ arterial,\ urban\ highway\}$ on date t. X_{jt} includes control

[15] A linear model in logs is similar to a ratio-in-ratios model in principle, but leads to biased results if there are zero count observations – days with no accidents on a particular road type. This happens frequently in accident data, particularly for fatal (88.39%) and (non-)incapacitating injury (22.20%, 57.08%) accidents.

[16] The Poisson model assumes:

$$\begin{aligned} f(y_i|\lambda_i) &= \frac{exp(-\lambda_i)\lambda_i^{y_i}}{y_i!} \\ \lambda_i &= exp(x_i'\beta) \end{aligned}$$

where x_i is a $(k \times 1)$ vector of regressors, and finally that observation pairs (y_i, x_i) are independently distributed. This model has the restrictive property of equidispersion – the conditional mean and variance are the same:

$$\begin{aligned} E(y|X) &= \lambda = exp(X'\beta) \\ Var(y|X) &= \lambda = exp(X'\beta) \end{aligned}$$

The standard negative binomial model instead assumes:

$$f(y_i|\alpha, \lambda_i) = \frac{\Gamma(\alpha + y_i)}{\Gamma(\alpha)\Gamma(y_i + 1)} \left(\frac{\alpha}{\lambda_i + \alpha}\right)^{\alpha} \left(\frac{\lambda_i}{\lambda_i + \alpha}\right)^{y_i}$$

[17] Underdispersion is not often observed in real-world data (Winkelmann, 2008).

[18] $H_0 : \alpha^{-1} = 0$ vs. $H_1 : \alpha^{-1} > 0$ provides a test if the fit of the negative binomial model is better than that of the Poisson model.

variables (weather, road, daylight and driver characteristics). Since the data are collapsed to the daily level, these control variables are averages (or relative frequencies) over all accidents that occurred on road type j. vmt_{jy} denotes vehicle miles traveled. θ indicates fixed effects for day-of-week d, month-of-year m, year y and highway type j. These are necessary to control for seasonality in accidents, longer-term time trends, and fixed relative differences across highway types. In the definition of the dependent variable, the count variable accidents a_{ijt} is scaled by vmt_{jy} to reflect an accident rate. Poisson and negative binomial models are equally suitable when the dependent count variable is scaled to non-integer values.

The average marginal effect is a function of both the covariates and the estimated parameters. However, the parameters have an easy interpretation in terms of relative changes: β is the relative change in $E(y|X)$ associated with a small change in X. If the dependent variable is binary, β is an approximation of the relative change in $E(y|X)$ when the dummy variable changes from 0 to 1.

The second hypothesis ("higher speed limits lead to more severe accidents") is tested using ordered choice models. These models test, conditional on an accident occurring, whether the probability of being in a more severe accident type increases. I assume that nature chooses the type of accident based on a latent index of accident severity:

$$\begin{gathered} a^*_{ijt} = \gamma_0 + \gamma_1 1\left(j \in T\right) * 1\left(t \geq t_{\Delta SL}\right) + \gamma_2 X_{ijt} + \theta_d + \theta_m + \theta_y + \theta_j + \eta_{ijt} \\ a = a_{pd} \; if \; a^* \leq 0 \\ \dots \\ a = a_{fatal} \; if \; \mu_2 \leq a^* \end{gathered} \tag{7}$$

for accident type i, highway type j, date t, day-of-week d, month-of-year m and year y. In the ordered logit model (η_{ijt} is assumed to be type I extreme value distributed), the (exponential of the) coefficient on the interaction term has an easy interpretation: when the interaction dummy $1\left(j \in T\right) * 1\left(t \geq t_{\Delta SL}\right)$ switches from 0 to 1, the ratios $\frac{a_{fatal}}{a_{inc}+a_{non-inc}+a_{pd}}$, $\frac{a_{fatal}+a_{inc}}{a_{non-inc}+a_{pd}}$ and $\frac{a_{fatal}+a_{inc}+a_{non-inc}}{a_{pd}}$ are all predicted to increase by that number (the "odds ratio"). In the ordered probit model, the marginal effects are complicated functions of γ_1 and the regressors. The equal odds ratio property does not hold. Since there is no clear theoretical reason to prefer one ordered choice model over another, I present results for both the ordered logit and the ordered probit model.

7.2 Accidents Results

7.2.1 Accident Rate Results

Table 7 summarizes the treatment effect of the speed limit changes in Oregon and Washington (1987) on various types of accidents, using the negative binomial model (6). The estimates from the Poisson model are very similar and are presented in Table 23 in Appendix B. Figure 15 in the same appendix shows that the negative binomial model predicts the observed accident proportions better than the Poisson model for all but fatal accidents. Therefore, the negative binomial model is the preferred specification in the remainder of this section. The upper panel presents the regressions

for Oregon and Washington combined. The lower panels present similar regression results for Oregon and Washington separately. Columns (1)-(4) restrict the sample to fatal, incapacitating, non-incapacitating and property damage accidents, respectively. Column (5) includes all accidents.

Table 7: Impact of 1987 Speed Limit Changes on Accident Rates: Negative Binomial Model

	Fatal	Incapacitating	Non-incapacitating	Damage only	Total
	(1)	(2)	(3)	(4)	(5)
			Oregon & Washington combined		
Interaction (β_1)	0.365***	0.211***	0.140***	0.124***	0.131***
	(0.113)	(0.048)	(0.030)	(0.019)	(0.019)
Exact relative change	0.441	0.235	0.150	0.132	0.140
Share of total accidents	1.5%	6.7%	17.2%	74.6%	100.0%
			Oregon		
Interaction (β_1)	0.469***	0.164**	0.052	0.088	0.084
	(0.163)	(0.083)	(0.070)	(0.061)	(0.059)
Exact relative change	0.598	0.178	0.054	0.092	0.087
Share of total accidents	2.4%	8.8%	19.9%	68.9%	100.0%
			Washington		
Interaction (β_1)	0.292***	0.213***	0.150***	0.099***	0.114***
	(0.092)	(0.056)	(0.056)	(0.036)	(0.037)
Exact relative change	0.339	0.238	0.161	0.104	0.120
Share of total accidents	1.0%	5.6%	15.8%	77.5%	100.0%
Observations	6,573	6,573	6,573	6,573	6,573

Notes: The coefficient on the interaction term of 15 separate regressions is reported. The dependent variable is the number of accidents per VMT per day. Highway type, year, month-of-year and day-of-week fixed effects are included. Controls are included. The exact relative change is calculated as $exp(\beta_1) - 1$. Standard errors clustered at the highway type by year level in parentheses. Observations are taken within a six year symmetric time window around the dates of the speed limit changes. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

The results indicate that the rates of various types of accidents on rural interstates went up sharply and significantly following the 1987 speed limit increases. The effect is strongest for fatal accidents, with estimated increases of 44.1%, 59.8% and 33.9% in the combined sample, Oregon and Washington, respectively.[19] This effect may appear large at first, but could be explained by the fact that the impact of a collision increases in the square of the difference in speed between two colliding objects. Thus, any given collision becomes substantially more likely to be fatal at higher speed. Moreover, I find that collisions (of any type) happened more frequently after the speed limit changes. For the combined sample and for Washington separately, all increases in the rates of less severe types of accidents are substantial and statistically significant at the 1% level, and vary between 10.4% and 23.8%. For Oregon, the increases in non-fatal accidents are positive but not statistically significant. Total accident rates increase by 8.7-14.0%. Non-fatal accidents, not included in previous speed limit research, have the potential to be important contributors to total accident costs. Their coefficients are 2-3 times smaller than the coefficient on fatal accidents,

[19]Standard errors are clustered at the highway type by year level. This assumes common unobserved shocks to accidents on a particular highway type within a given year. This is a conservative choice, since I control for road characteristics, road construction and weather. Moreover, accidents happen at different locations and times within a particular day and highway type, and are plausibly uncorrelated conditional on observables.

but their incidence rates are about 4-50 times larger (for the combined sample).

7.2.2 Accident Severity Results

The estimates above show that the speed limit changes not only led to a sizable increase in accidents, but also suggest that they caused a shift towards more severe accidents. The ordered choice models (7) presented in Table 8 confirm this.

Table 8: Impact of 1987 Speed Limit Changes on Accident Severity: Ordered Choice Models

	OR & WA 6-year window (1)	OR & WA 8-year window (2)	Oregon 6-year window (3)	Washington 6-year window (4)	Washington 8-year window (5)
			Ordered logit model		
Interaction	1.0605*	1.0704**	1.0919***	1.0754*	1.0875**
	(0.0370)	(0.0367)	(0.0362)	(0.0431)	(0.0402)
			Ordered probit model		
Interaction	0.0390*	0.0456**	0.0535**	0.0426**	0.0521**
	(0.0210)	(0.0206)	(0.0234)	(0.0218)	(0.0216)
Effect on a_{pd}	-0.0124*	-0.0143**	-0.0188**	-0.0126*	-0.0155**
	(0.0067)	(0.0066)	(0.0083)	(0.0065)	(0.0065)
Percentage change	-1.82%	-1.95%	-2.09%	-1.77%	-1.91%
Effect on $a_{non-inc}$	0.0070*	0.0082**	0.0096**	0.0076*	0.0093**
	(0.0038)	(0.0037)	(0.0042)	(0.0039)	(0.0039)
Percentage change	4.37%	5.01%	3.52%	4.98%	5.74%
Effect on a_{inc}	0.0043*	0.0050**	0.0071**	0.0042*	0.0051**
	(0.0024)	(0.0023)	(0.0032)	(0.0022)	(0.0022)
Percentage change	7.91%	8.43%	6.88%	9.55%	9.65%
Effect on a_{fatal}	0.0011*	0.0012**	0.0021**	0.0008*	0.0010**
	(0.0006)	(0.0006)	(0.0009)	(0.0004)	(0.0005)
Percentage change	13.89%	13.43%	13.40%	17.51%	15.30%
Observations	131,282	165,427	44,466	86,816	114,207

Notes: The coefficient on the interaction term of 10 separate regressions is reported. The dependent variable is accident severity $\in \{1{:}\ a = a_{pd},\ 2{:}\ a = a_{non-inc},\ 3{:}\ a = a_{inc},\ 4{:}\ a = a_{fatal}\}$. Highway type, year, month-of-year and day-of-week fixed effects are included. Controls are included. Average marginal effects for the ordered probit model are calculated at the means of the independent variables and for discrete changes in treatment status and pre-post period. The percentage change is calculated as the ratio of the average marginal effect on a_j and the predicted $Pr(a = a_j)$. Standard errors clustered at the highway type by year level in parentheses. The time window is relative to the dates of the speed limit changes. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

The results from the various ordered choice models in Table 8 consistently indicate that there was a statistically significant shift towards more severe accidents following the 1987 speed limit changes in both states. The odds ratio from the ordered logit models varies between 1.06 and 1.09. This means that, conditional on being in an accident, the probability of being in a fatal accident relative to a non-fatal accident increased by 6-9%. Similarly, the probability of being in an incapacitating or fatal accident increased by 6-9%. For the ordered probit models, the marginal effects indicate that, conditional on being in an accident, the probability of being in a property damage only accident decreased by about 2%, while the probability of being in a more severe type of accident increased by approximately 4-6% (non-incapacitating), 7-10% (incapacitating) and 13-18% (fatal). In summary, Tables 7-8 provide evidence that both hypotheses hold: higher speed limits led to both an increase in the amount of accidents and a shift towards more severe accidents.

7.2.3 Alternative Specifications and Robustness Checks

I now proceed to investigate if these results are robust to alternative econometric specifications. First, I include more flexible, highway type specific, control coefficients. Second, I limit the control highways to a subsample of highways with (more) similar accident rates to the treatment highways, to alleviate the concerns about observable differences between rural interstates and other highways discussed in Section 3. Third, I investigate how the treatment effect changes over time. Fourth, I briefly discuss a placebo test. Additional robustness checks are presented in Appendix B. For reasons of brevity, I limit the discussion to the combined sample for Oregon and Washington and to the accident rate models, unless indicated otherwise.

Section 4 discussed that accidents follow strong seasonal trends: the majority of accidents occur between November and February. It is conceivable that the effect of a speed limit change on accidents varies by season. A potential bias could arise if accidents on different highway types are affected by the weather in different ways, but this is not allowed in the econometric specification. If the winters following the speed limit changes were more severe than before, and if accidents on rural interstates responded more strongly to winter conditions, the treatment effect would absorb this weather-induced change in accidents. Specification (6) restricts β_2 to be equal for all highway types. Table 9 relaxes this assumption. The first specification allows for highway type specific weather coefficients β_{2j}. In the second specification, the coefficients vary by highway type for all control variables.[20]

Section 3 discussed that rural interstates typically have lower accident rates per vehicle mile traveled than rural principal arterials. A critical robustness check would be to test if the treatment effect is similar when a subsample of control highways with the same accident rates as the treatment highways is used. Using data on highway specific VMT in Oregon, I calculate total accident rates for all highway segments and remove the least safe rural principal arterials and urban highways until all three highway types have the same total accident rate. For Washington, highway specific VMT data only go back to 2004. I therefore cannot compare accident rates around 1987 directly.[21] Instead, I compute the total accident rate by highway using 2004 VMT data, and keep the control highways with accident rates in the bottom half. The results are reported in panels 3-5 of Table 9.

The first (second) panel includes highway type specific weather (control) coefficients. It is reassuring to observe that the estimates are similar to the main specification in Table 7: allowing for differential impacts of the control variables on accidents rates does not change the results.[22] The next three panels demonstrate that the main results in Table 7 are remarkably similar to those when the control highways are limited to a set of highways with (more) similar accident rates to

[20] Table 24 in Appendix B reports specifications which allow for season specific treatment effects β_{1s}. The treatment effect in winters is higher than in the summer. This likely reflects that driving at higher speed is especially dangerous under less favorable winter conditions.

[21] The Poisson and negative binomial models are invariant to the level of VMT by highway type. Therefore, under the assumption that trends in VMT on individual roads were the same as the VMT trend for the corresponding highway type, the highway type specific VMT data can be used to estimate the treatment effect using subsets of highways of a particular type.

[22] In panels 1-2, a joint F-test rejects that the control coefficients are equal across highway types (p-value 0.0000).

Table 9: Robustness Checks - Flexible Control Coefficients and Comparable Control Highways

	Fatal	Incapacitating	Non-incapacitating	Damage only	Total
	(1)	(2)	(3)	(4)	(5)
			Highway type specific weather coefficients		
Interaction (β_1)	0.323***	0.194***	0.136***	0.131***	0.135***
	(0.103)	(0.038)	(0.022)	(0.016)	(0.013)
Exact relative change	0.381	0.214	0.146	0.140	0.145
			Highway type specific control coefficients		
Interaction (β_1)	0.344***	0.216***	0.166***	0.165***	0.169***
	(0.082)	(0.040)	(0.030)	(0.027)	(0.024)
Exact relative change	0.410	0.241	0.180	0.179	0.184
			Oregon & Washington - safe control highways		
Interaction (β_1)	0.510***	0.237***	0.154***	0.142***	0.151***
	(0.097)	(0.047)	(0.038)	(0.020)	(0.023)
Exact relative change	0.666	0.267	0.166	0.153	0.163
			Oregon - safe control highways		
Interaction (β_1)	0.604***	0.160*	0.072	0.096	0.098
	(0.169)	(0.096)	(0.078)	(0.070)	(0.070)
Exact relative change	0.829	0.174	0.072	0.101	0.103
			Washington - safe control highways		
Interaction (β_1)	0.340***	0.276***	0.182***	0.124***	0.136***
	(0.096)	(0.046)	(0.051)	(0.030)	(0.031)
Exact relative change	0.406	0.318	0.199	0.132	0.146

Notes: The coefficient on the interaction term of 35 separate negative binomial regressions is reported. The dependent variable is the number of accidents per VMT per day. Highway type, year, month-of-year and day-of-week fixed effects are included. Controls are included. "Safe control highways" have the same average accident rates per vehicle mile traveled as rural interstates (OR), or include only the safest rural principal arterials per vehicle mile traveled (US-97, US-195, US-395, US-730). The exact relative change is calculated as $exp(\beta_1) - 1$. Standard errors clustered at the highway type by year level in parentheses. The number of observations is 6,573 (default: six year symmetric time window around the dates of the speed limit changes), 8,307 (eight year window) or 4,383 (four year window). ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

the rural interstates. In other words, the treatment effect is unlikely to be biased because of the inclusion of the more accident prone control freeways (mostly unsafe rural principal arterials). This finding strongly contributes to the credibility of identification assumption 2 (Section 3).[23]

Table 10 shows how the treatment effect varies over time by limiting the sample to three years of pre-treatment data plus one year of post-treatment data. Interestingly, and in contrast with the results for the effect on speed in Figure 5, a strong effect on accidents appears directly in the first year after the speed limit changes. The effect does not change much over time. This suggests that speed limit changes have an immediate negative effect on road safety, even if the average speed does not change much initially. One potential explanation is that congestion caused by increased accidents puts downward pressure on the average speed. Over time, drivers adjust and travel at faster speeds without experiencing a further increase in accidents.

Table 25 in Appendix B presents additional robustness checks. These demonstrate that the

[23]When control highways are selected based on similar *fatal* accident rates, the results appear robust as well. In Washington, this would include the US-101 in the sample and exclude the US-97. The coefficients are 0.424, 0.269, 0.179, 0.113 and 0.130 for the five categories of accidents. The coefficients of the main specification are 0.365, 0.211, 0.140, 0.124 and 0.131, respectively. In contrast, when the sample of control freeways is extended with more dissimilar freeways (all *minor* urban and rural arterials), the treatment effects become smaller: 0.279, 0.165, 0.125, 0.061 and 0.081, respectively.

Table 10: Time-Varying Treatment Effects

	Fatal	Incapacitating	Non-incapacitating	Damage only	Total
	(1)	(2)	(3)	(4)	(5)
			History plus year 1		
Interaction (β_1)	0.405***	0.220***	0.150***	0.124***	0.134***
	(0.119)	(0.045)	(0.036)	(0.011)	(0.014)
Exact relative change	0.500	0.246	0.162	0.132	0.143
Observations	4,383	4,383	4,383	4,383	4,383
			History plus year 2		
Interaction (β_1)	0.332***	0.256***	0.090***	0.131***	0.131***
	(0.126)	(0.055)	(0.021)	(0.030)	(0.030)
Exact relative change	0.394	0.291	0.094	0.140	0.140
Observations	4,380	4,380	4,380	4,380	4,380
			History plus year 3		
Interaction (β_1)	0.454***	0.170***	0.174***	0.134***	0.143***
	(0.108)	(0.045)	(0.028)	(0.037)	(0.034)
Exact relative change	0.575	0.186	0.189	0.143	0.153
Observations	4,380	4,380	4,380	4,380	4,380

Notes: The coefficient on the interaction term of 15 separate negative binomial regressions using the combined Oregon and Washington sample is reported. The dependent variable is the number of accidents per VMT per day. "History" refers to three years before the speed limit changes. Highway type, year, month-of-year and day-of-week fixed effects are included. Controls are included. The exact relative change is calculated as $exp(\beta_1) - 1$. Standard errors clustered at the highway type by year level in parentheses. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

main results are not driven by alcohol or drugs related accidents, or by truck related accidents. The results of a time discontinuity specification – similar in nature to equation (4) for travel speed – are somewhat similar in magnitude to the main results, but the estimates are sensitive to the choice of the order of the time polynomial.

Finally, I present a placebo test for the accidents results. I run regressions in which a hypothetical speed limit change was introduced on rural interstates 6, 7, 8, 9 and 10 years after the real speed limit change, using symmetric time windows of 6, 8 and 10 years. This adds up to a total of 75 regressions (15 for each category of accidents). I find that only 4 of these 75 regressions have t-statistics larger in absolute value than the corresponding t-statistics from the regressions with the 1987 speed limit changes. This corresponds to a "p-value" of 5.3%. An analogous analysis with the magnitude of the coefficients gives similar results: 5.3% of the placebo regressions have coefficients larger than the corresponding coefficients for 1987 (8.0% have larger coefficients in absolute value). These results suggest that the probability that the treatment effects found in 1987 are a result of pure chance is small. Appendix B discusses the caveats of this test and offers a graphical representation (Figure 16).

7.2.4 Potential Confounding Trends

Notwithstanding the various robustness checks above, there remains a risk of confounding unobserved trends that affected accidents in the period around the speed limit changes. A first possibility is changes in seat belt laws (Cohen and Einav, 2003) and child seat laws (IIHS, 2011). However, changes in these laws affect occupants of vehicles on all highways. A confounding effect would only

be present if there was a differential impact of the seat belt laws on rural interstates. Moreover, most of the changes happened outside the six-year window.[24] Even so, Table 11 explicitly takes into account the only change inside the window: Washington required the use of seat belts on 11 June 1986. In Table 11, the year fixed effects are replaced by a dummy variable that reflects the introduction of the 1986 seat belt law in Washington. The law had a negative impact on accident rates (although not always statistically significant), while the estimates of the treatment effects remain largely unchanged. Hence, seat belt and child seat laws do not seem to confound the results.

Table 11: Confounding Trend: Seat Belt Laws

	Fatal	Incapacitating	Non-incapacitating	Damage only	Total
	(1)	(2)	(3)	(4)	(5)
			Washington - seat belt law		
Interaction (β_1)	0.300***	0.214***	0.184***	0.105***	0.126***
	(0.077)	(0.045)	(0.046)	(0.037)	(0.038)
Seat belt law dummy (D_{sb})	-0.096	-0.160***	-0.107**	-0.035	-0.057**
	(0.065)	(0.047)	(0.043)	(0.022)	(0.023)
Exact relative change (β_1)	0.350	0.239	0.202	0.111	0.135
Exact relative change (D_{sb})	-0.092	-0.148	-0.102	-0.034	-0.056

Notes: The coefficient on the interaction term of 5 separate negative binomial regressions is reported. The dependent variable is the number of accidents per VMT per day. Highway type, month-of-year and day-of-week fixed effects, and controls, are included. The exact relative change is calculated as $exp(\beta_1) - 1$. Standard errors clustered at the highway type by year level in parentheses. The number of observations is 6,573. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Macroeconomic conditions could be a second confounding trend, if they affected driving on rural interstates differentially from other highways. For instance, if unemployment in counties with rural interstates decreased relative to other counties following the speed limit change, the relative traffic density of rural interstates could have increased. This could have caused more accidents on the treatment highways even in the absence of a speed limit change. Controlling for VMT largely alleviates this concern. In addition, Table 22 in Appendix B shows that there is no evidence for such differential changes in traffic density in Oregon and Washington.

A final, and more substantive, challenge would be the existence of differential enforcement of speed limits. It is not inconceivable that the higher speed limits were accompanied by a period of increased enforcement. If that indeed happened, increased enforcement on rural interstates could have put a downward pressure on traffic speed and accidents. In that case, the estimates presented in this section should be interpreted as the combined effect of higher speed limits plus increased enforcement. That said, the highway patrol in all three states stated that there had been no official orders to increase enforcement following the speed limit increases (see Appendix A).[25]

[24] Oregon implemented a similar seat belt law on 7 December 1990. This falls almost outside the study period. Oregon and Washington introduced child seat laws on 1 January 1984, which is outside the six year windows for both states.

[25] None of the states has collected data on enforcement by highway type, so I cannot test if the change in accidents can be partially attributed to changes in enforcement. Figure 14 in Appendix A plots statewide trends in speeding tickets and highway trooper employment. However, even an absence of statewide trends does not rule out the possibility of redistributing police resources towards rural interstates.

8 The Effect of Speed Limit Changes on Pollution

This section estimates the changes in air pollutant concentrations as a result of the 1987 speed limit changes. In Section 10, I use epidemiological "concentration-response functions" to translate these estimates to adult health impacts not covered in my data set.

8.1 Speed and Air Pollution

Vehicle emissions are important sources of local air pollution and global greenhouse gas emissions. Carbon monoxide (CO), nitrogen oxides (NO_x), volatile organic compounds (VOCs) and carbon dioxide (CO_2) are all direct byproducts of the internal combustion process. In 2009, the transportation sector's share of total CO_2 emissions in the United States was 31.2% (EPA, 2011a). Moreover, motor vehicles are responsible for the majority of CO emissions (67-97%), and a large share of NO_x emissions (33-50%) (Chatterjee *et al.*, 1997; Hallmark, 1999; EPA, 2011b). Vehicles are also indirectly responsible for the formation of ground level ozone (O_3), through a reaction between NO_x and VOCs in the atmosphere in the presence of sunlight. Direct emissions of particulate matter smaller than 10 micrometers in diameter (PM_{10}) from gasoline car engines are minimal, but NO_x can react with other atmospheric pollutants to form PM_{10} indirectly (EPA, 2011b).[26]

Vehicle speed and emissions per mile are related in a non-linear way (e.g., TRB, 1995; Hallmark, 1999; Litman and Doherty, 2009). The EPA's MOBILE and MOVES models provide emissions by speed, for various vehicle types and model-years. Figure 7 shows the U-shaped relationship between vehicle speed, per-mile tailpipe emissions (CO, NO_x) and the rate of fuel consumption for the vehicle fleet in 1990, a year in between the two major speed limit changes in 1987 and 1996.[27]

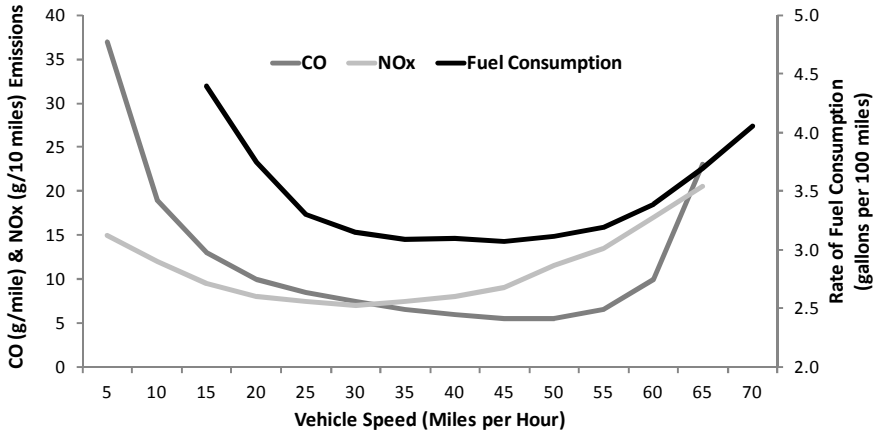
At low speeds, emissions per mile and the rate of fuel consumption are high. At moderate speeds (30-50 mph), fuel consumption and emissions per mile reach a minimum. At higher speeds, CO and NO_x emissions per mile increase rapidly and disproportionately to fuel consumption.[28]

[26] Several empirical studies have confirmed the strong relationship between traffic and local air pollution. Hu *et al.* (2009) document elevated air pollutant concentrations in a wide area surrounding freeways. Davis (2008) analyzes the effect of Mexico City's *Hoy No Circula* policy on local air pollution and finds no discernible impact. Knittel *et al.* (2010) find a strong relationship between unusually high local traffic density and pollution levels in California.

[27] Both gasoline and diesel engine emissions exhibit this U-shaped relationship, although the relationship between speed and CO is less steep for diesel (TRB, 1995; Barlow and Boulter, 2009). I focus on gasoline vehicles, since diesel vehicles represented only 0.0-4.3% of new retail car sales and 1.7-8.5% of light-duty truck sales in the United States between 1975-2009 (Davis *et al.*, 2010). The majority of commercial trucks are diesel fueled, but these represent only 3.2-3.6% of the vehicle fleet (and 6.8-7.6% of vehicle miles traveled) in the period 1980-2009 (Ward's Automotive Yearbook, 2010). Moreover, truck speed limits did not change in most of the cases considered in this paper.

[28] The laws of physics and vehicle engineering explain the positive relationship between speed and emissions at higher speeds. In terms of physics, the energy required per unit of distance to overcome air resistance is proportional to the square of speed. Thus, keeping technology fixed, fuel economy will eventually decline rapidly with speed. In terms of engineering, vehicle engines are designed for specific speed ranges and are optimized to perform well at both city and freeway speeds. Driving at speeds above the design range leads to less efficient combustion. In addition, differences in temperature and fuel-to-air ratios lead to sharp changes in the exhaust concentrations of local pollutants (TRB, 1995). Within some range, manufacturers can tune their engines to be most efficient at a higher speed following a speed limit increase. In fact, today's cars operate more efficiently between 55 and 65 mph than the 1990 fleet did. However, this tuning makes the vehicle more polluting at lower speeds. Since the EPA has not changed its local emissions test cycle since 1990, the re-optimized vehicles would fail the test if the requirement was binding initially. Even if the test were non-binding, it takes several years to bring new, re-optimized, vehicles to the

Figure 7: The Relationship Between Vehicle Speed, Emissions and Fuel Consumption



Notes: 1990 fleetwide average for gasoline vehicles. Source: Litman and Doherty (2009) based on the EPA's MOBILE5a model (http://www.epa.gov/oms/m5.htm).

CO emissions triple when the vehicle speed increases from 55 to 65 mph. NO_x emissions increase by about 50%. These numbers are based on testing a limited number of vehicles. They can be taken as evidence for a strong relationship between speed and pollution, but the exact magnitudes in Figure 7 should be interpreted with caution.

In summary, the engineering literature suggests that we may expect that the 1987 and 1996 increases in speed limits above 55 mph have led to substantial increases in CO and NO_x concentrations. The effect on O_3 could be weaker, since it forms indirectly through a chemical reaction between NO_x (which increases rapidly with speed) and VOCs (74% of which are emitted by non-traffic sources and do not vary with speed). We should expect no effect on PM_{10}, since direct emissions from gasoline engines are negligible and transportation is only responsible for a small share of indirectly formed PM_{10} (EPA, 2011b).

8.2 The Spreading of Air Pollution

There is a large literature in engineering, epidemiology and atmospheric modeling on the spreading of air pollution. One strand of papers focuses on *pollution gradients* at short distances from freeways. The typical experiment measures pollutant concentrations both upwind and downwind from a highway, and establishes how far the downwind station has to be moved from the highway to observe pollution levels within (e.g.) 5% of the upwind ("background") station (see Zhou and Levy (2007) for an overview). Most papers find that such gradients exist over relatively short distances, up to 1000 feet. However, several papers found gradients to stretch out over much longer distances up to two miles (Hu *et al.*, 2009), or even that there is hardly any spatial decay in downwind concentrations during stable atmospheric conditions (Roorda-Knape *et al.*, 1998).

market. Then, even if all new models were adjusted (an unlikely scenario), penetration would be slow due to limited vehicle fleet turnover: the average age of the U.S. light vehicle fleet between 1995-2009 was 8.4-10.2 years (Ward's, 2010). Today's engines still exhibit a U-shaped emissions pattern, albeit shifted to the right by about 10 mph.

The EPA reports that many pollutants can spread very far: pollution from the U.S. Midwest reaches the East Coast (EPA, 2011b). Discussion with EPA modelers has confirmed that there are two relevant air pollution effects: changes in pollution gradients and changes in pollution background concentrations. These two phenomena can co-exist: at any point in time, a pollution gradient can exist as long as atmospheric conditions (e.g., wind speed and direction) do not change much. Over the course of one or several days, wind directions change and the increased pollution disperses within a larger area ("buffer zone") around the freeway. There is no clear evidence on how wide such buffer zones are in practice, and this is in fact one of the challenges for EPA modelers. It is not unreasonable to assume effects for distances up to 10 miles (EPA, personal communication, 2 June 2011). A pure focus on short distance pollution gradients is even problematic insofar as it ignores changes in background concentrations. The relevant width of the pollution buffer is to some extent an empirical question which could help inform atmospheric modelers.

Pollution gradients are relevant to people living very close to freeways by exposing them to high temporary emissions levels. Pollution buffers are relevant to people within a wider area around the freeway by exposing them to generally elevated pollutant concentrations. Section 4 discussed the limited availability of pollution stations very close to freeways. For that reason, I can only test for the "pollution buffer effect" of speed limit changes with varying buffer distances. To keep a reasonable sample size, distances smaller than three miles are not used in this analysis.

8.3 Econometric Framework

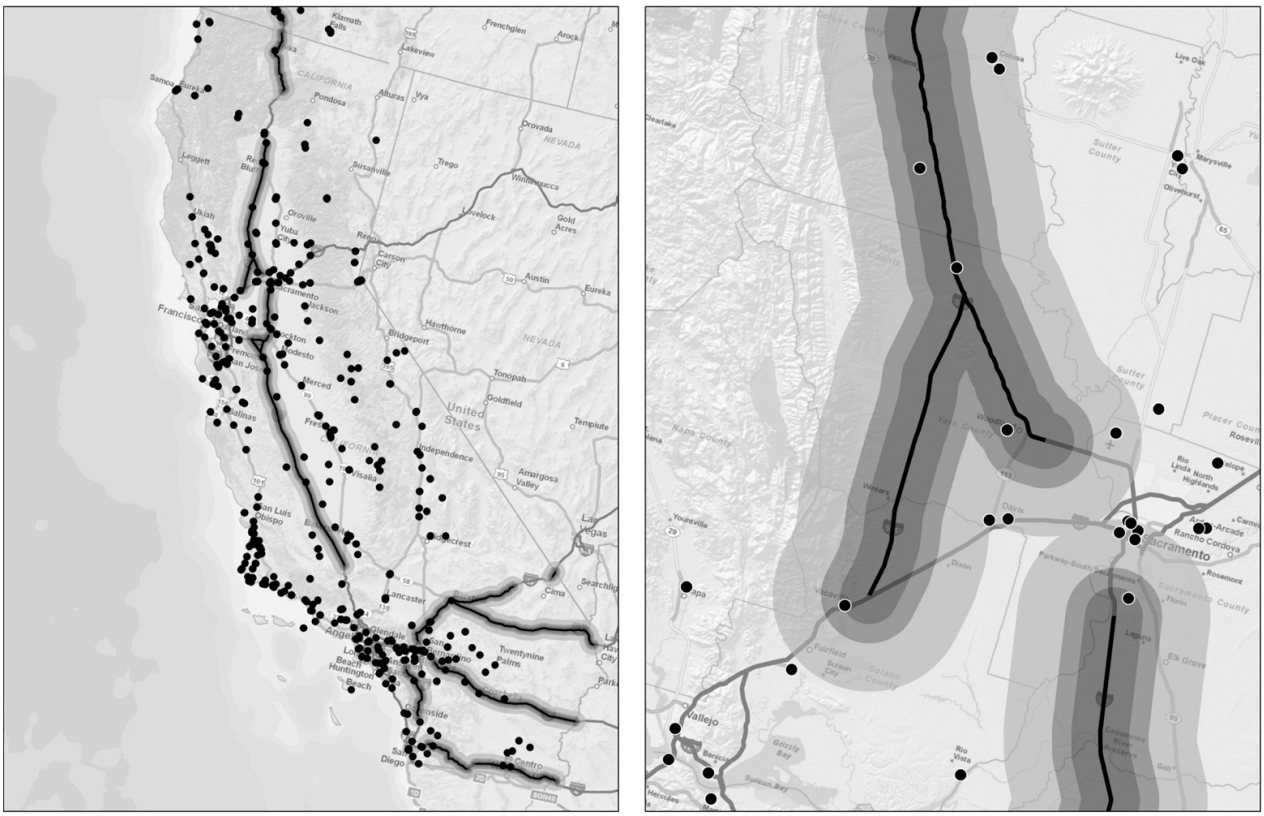
To estimate the effect of speed limit changes on pollution, I use a difference-in-differences estimator. I group stations based on their proximity to the relevant freeway segments. This estimator is preferred over a single difference design, since air pollution concentrations are not only determined by local emissions, but also by imported pollution from other regions and by state and federal environmental policies. There has been a declining trend in the concentrations of many pollutants over the last three decades. To estimate the impact of the 55 to 65 mph increase on rural interstates in 1987, I define treatment stations as being located at most x miles away from the 10 mph change. Control stations are located at least y miles away from the 10 mph change, where $y \geq x$ (Figure 8). The central case is $(x, y) = (3, 3)$, but I report the robustness to the buffer distance.

Air pollution is highly seasonal at both the monthly and daily level, and has an annual trend. Moreover, pollution concentrations depend on meteorological conditions. For example, PM_{10} concentrations tend to be lower on rainy days. In addition, the chemical reactions between pollutants depend on outside temperature. I therefore employ the following specification:

$$ln(p_{it}) = \beta_0 + \beta_1 1(dist \leq x) * 1(t \geq t_{\Delta SL}) + \beta_2 X_{it} + \theta_i + \theta_d + \theta_m + \theta_y + \varepsilon_{it}, \qquad dist \notin (x, y) \quad (8)$$

where $p_{it} \in \{CO, NO_2, NO_x, O_3, PM_{10}\}$ indicates the pollution concentration for pollution station i at date t. X_{it} includes weather variables (average, maximum and minimum temperature, wind speed and indicator variables for rain, fog, snow, hail, thunder or tornado). θ represents fixed

Figure 8: Treatment and Control Pollution Stations Using Various Pollution Buffer Definitions



Notes: (Left panel) 3, 5 and 10 mile buffers around treatment freeways in California, 1987. Black dots: air pollution monitoring stations. (Right panel) 3, 5 and 10 mile buffers near Sacramento, CA. In this example, a $(3, 3)$ buffer corresponds to using 5 treatment stations. Using a $(10, 10)$ buffer, there are 15 treatment stations. Using a $(3, 10)$ buffer, there are 5 treatment stations and the additional 10 stations within the 10 mile buffer would be excluded.

effects for pollution station i, day-of-week d, month-of-year m and year y.

Unobserved shocks to industrial activity could bias the estimates. For example, if the speed limit change coincided with the opening or expansion of a high-emitting factory close to a treatment station, the observed increase in pollution would be incorrectly attributed to the speed limit change only. This concern applies mostly to NO_2/NO_x, since CO emissions from industrial sources are minimal. To mitigate this concern, I collected data on the number of industrial establishments and the associated employment levels (by industry SIC code) from the census County Business Patterns. I then selected the SIC codes for which CO, NO_x and PM_{10} emissions exceeded one percent of total emissions during the period 1984-1990, and created county by year counts of "dirty sector" employment and large establishments ($> 1,000$ employees). These are included in X_{it}.

8.4 Pollution Results

Table 12 shows the estimation results for the central case (column (1)) and for varying treatment and control group cutoff distances (columns (2)-(5)), with and without the SIC controls. The

sample includes observations from 1984-1990, approximately a symmetric time window around the speed limit changes. Data availability for treatment stations before 1984 is very limited.

Table 12: Regression Results for the Effect of the 1987 Speed Limit Changes on Pollution, for Various Buffer Distances (California, Oregon & Washington Combined)

	Industry controls	$(x,y)=(3,3)$ (1)	$(x,y)=(3,5)$ (2)	$(x,y)=(5,5)$ (3)	$(x,y)=(5,10)$ (4)	$(x,y)=(10,10)$ (5)
CO	No	0.2431***	0.2541***	0.2457***	0.2423***	0.1385***
	No	(0.0572)	(0.0573)	(0.0406)	(0.0407)	(0.0356)
	No	[0.0570]	[0.0575]	[0.0450]	[0.0451]	[0.0373]
	Yes	0.2313***	0.2425***	0.2437***	0.2396***	0.1325***
	Yes	(0.0576)	(0.0576)	(0.0405)	(0.0406)	(0.0355)
	Yes	[0.0572]	[0.0579]	[0.0449]	[0.0449]	[0.0371]
NO_2	No	0.1491***	0.1503***	0.0928***	0.0955***	0.0891***
	No	(0.0349)	(0.0349)	(0.0231)	(0.0232)	(0.0206)
	No	[0.0355]	[0.0357]	[0.0253]	[0.0256]	[0.0230]
	Yes	0.1477***	0.1486***	0.0888***	0.0914***	0.0889***
	Yes	(0.0350)	(0.0351)	(0.0233)	(0.0234)	(0.0207)
	Yes	[0.0356]	[0.0358]	[0.0256]	[0.0260]	[0.0231]
NO_x	No	0.1596***	0.1622***	0.1036***	0.1085***	0.1168***
	No	(0.0419)	(0.0418)	(0.0265)	(0.0267)	(0.0241)
	No	[0.0432]	[0.0428]	[0.0241]	[0.0244]	[0.0225]
	Yes	0.1589***	0.1613***	0.1017***	0.1063***	0.1167***
	Yes	(0.0419)	(0.0418)	(0.0267)	(0.0269)	(0.0241)
	Yes	[0.0433]	[0.0429]	[0.0247]	[0.0249]	[0.0225]
O_3	No	0.1079***	0.1051***	0.0571***	0.0513**	0.0129
	No	(0.0199)	(0.0200)	(0.0166)	(0.0167)	(0.0133)
	No	[0.0218]	[0.0221]	[0.0197]	[0.0204]	[0.0184]
	Yes	0.1069***	0.1043***	0.0559***	0.0500**	0.0114
	Yes	(0.0199)	(0.0200)	(0.0166)	(0.0166)	(0.0133)
	Yes	[0.0218]	[0.0220]	[0.0196]	[0.0204]	[0.0184]
PM_{10}	No	0.0291	0.0101	-0.0339	-0.0048	-0.0057
	No	(0.0358)	(0.0363)	(0.0278)	(0.0288)	(0.0218)
	No	[0.0376]	[0.0392]	[0.0325]	[0.0344]	[0.0256]
	Yes	0.0236	0.0072	-0.0290	-0.0111	-0.0045
	Yes	(0.0358)	(0.0366)	(0.0287)	(0.0298)	(0.0231)
	Yes	[0.0376]	[0.0394]	[0.0325]	[0.0346]	[0.0261]
N_{avg}	No	180,053	173,283	180,053	169,339	180,053
	Yes	179,308	172,538	179,308	168,594	179,308

Notes: The coefficient on the interaction term of 50 separate regressions is reported. The dependent variable is the log of the pollutant concentration. Standard errors clustered at the station by month level in (), and clustered at the county by month level in [] (used for stars). The time window is 1984-1990. N_{avg} denotes the average number of observations for the specifications in a column. All specifications contain weather controls and pollution station, day-of-week, month-of-year and year fixed effects. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

The central case estimates (without industry controls) show a large and statistically significant percentage increase in concentrations of CO (+24%), NO_x (+16%) and O_3 (+11%). The effect on PM_{10} is small and not significantly different from zero.[29] These estimates suggest that the

[29]Standard errors are clustered at the station by month and county by month level. Clustering by station is reasonable since it is plausible that certain unobserved shocks, such as changes in imported pollution from other regions, lead to correlation between the measurements from a station over time. Clustering at the county level is more conservative, by not treating stations in the same county as independent observations. A meteorological entity such as an air basin may constitute a more natural clustering unit than a political entity such as a county. However, only California has defined such air basins. To determine the relevant time dimension of the clustering, I investigated

speed limit changes led to elevated pollutant concentrations in at least a three mile buffer zone. The relative magnitude of the coefficients makes sense in light of the discussion of Figure 7, which shows that the effect on CO is expected to be larger than the effect on NO_x.[30] The somewhat smaller estimate for O_3 is consistent with this being an indirect pollutant. As argued above, I should find no effect on PM_{10}. The results in Table 12 confirm this. This is an important specification check and adds to the credibility of the results. Columns (2)-(5) indicate that as the size of the buffer zone increases, the effects become smaller in magnitude. This is consistent with a gradual decay of the impact of pollution.[31] The regression results with and without industry controls are very similar, suggesting that sudden changes in industrial composition do not drive the results.[32]

Table 13 shows the timing of the pollution effects. Each column contains the full set of pre-treatment observations but only one year of post-treatment observations. Column (5) repeats the full sample estimates; columns (1)-(4) show the effect from one to four years after the speed limit changes. The effect on pollution concentrations in the first year after the speed limit changes tends to be small, but increases in subsequent years.[33] This is consistent with the finding that the effect on travel speed is increasing over time (Section 6).

In summary, I interpret the pollution results as broadly consistent with the literature on speed and pollution, air pollution dispersion and the effect of speed limit changes on speed. However, given the limited number of treatment stations, the results cannot be estimated at a great degree of precision, and future research (e.g., using some of the recent speed limit changes) is warranted.

9 The Effect of Speed Limit Changes on Infant Health

A final input for the cost-benefit analysis is the effect of the higher speed limits, through increased pollution, on health (Figure 1). As discussed in Section 2, air pollution has been shown to negatively affect several health outcomes, among both adults and infants. In this section I focus on fetal health and infant health at birth. This choice is motivated by three reasons. First, since a fetus can only be exposed to air pollution in a relatively short nine-month window, we can be sure that only recent

the autocorrelation functions (ACFs) of the measurements of all stations and for all five pollutants. I then recorded the first time lag for which the ACF was insignificant, and calculated the average over all stations for each pollutant. The result is 34.5, 33.3, 31.2, 30.7 and 16.5 days for CO, NO_2, NO_x, O_3 and PM_{10}, respectively. The median is slightly lower. Therefore, I conclude that the relevant time dimension for clustering is the monthly level.

[30] It is hard to draw further conclusions on the expected magnitude of the effect based on the emissions-at-source estimates in Figure 7. These are lab test emissions measurements for a limited number of vehicle models only. Moreover, many atmospheric conditions affect how far various pollutants spread.

[31] As an additional robustness check, I excluded control stations more than 20 miles from treatment freeways. The same pattern as in Table 12 persists, but the effect on CO (NO_x) is about 10 (6) percentage points smaller.

[32] Traffic substitution towards treatment highways also does not drive the results. Section 6 shows that such substitution is limited and statistically insignificant. Even if I combine the highest, but insignificant, point estimate (5.49%, for California) with the speed-emissions curves in Figure 7, 93% of the increase in CO and 75% of the increase in NO_x tailpipe emissions can be attributed to higher speed. The remainder would be due to substituted traffic. In the extreme case where I take the upper end of the confidence interval for the substitution coefficient, I can easily rule out that the pollution increase attributable to speed is zero: 87% and 59% of the CO and NO_x increases are still due to higher speed, respectively.

[33] Similar results are obtained when $(5, 5)$ is used as the buffer definition, or when industry controls are excluded. Results available upon request.

Table 13: Time-Varying Treatment Effects - $(x, y) = (3, 3)$

	Year 1	Year 2	Year 3	Year 4	Full
	(1)	(2)	(3)	(4)	(5)
CO	0.1042	0.4339***	0.2744***	0.4297***	0.2313***
	(0.0749)	(0.0953)	(0.0941)	(0.1061)	(0.0576)
	[0.0771]	[0.0945]	[0.0945]	[0.1057]	[0.0572]
N_{CO}	117,174	119,033	118,950	119,069	172,402
NO_2	0.0486	0.1232**	0.1348***	0.0766	0.1477***
	(0.0569)	(0.0477)	(0.0478)	(0.0492)	(0.0350)
	[0.0591]	[0.0493]	[0.0489]	[0.0497]	[0.0356]
N_{NO_2}	120,266	123,550	125,435	125,895	185,257
NO_x	0.0552	0.1257**	0.1457**	0.0834	0.1589***
	(0.0725)	(0.0623)	(0.0634)	(0.0618)	(0.0419)
	[0.0751]	[0.0642]	[0.0654]	[0.0645]	[0.0433]
N_{NO_x}	117,231	120,096	122,041	121,160	181,551
O_3	0.0832**	0.1277***	0.1286***	0.1237***	0.1069***
	(0.0315)	(0.0312)	(0.0315)	(0.0313)	(0.0199)
	[0.0334]	[0.0327]	[0.0349]	[0.0338]	[0.0200]
N_{O_3}	206,496	209,174	210,683	211,551	305,850
PM_{10}	-0.0199	0.0052	0.0029	-0.0720	0.0236
	(0.0426)	(0.0432)	(0.0517)	(0.0507)	(0.0358)
	[0.0451]	[0.0452]	[0.0540]	[0.0542]	[0.0376]
$N_{PM_{10}}$	21,914	23,423	24,777	26,338	51,511

Notes: The coefficient on the interaction term of 25 separate regressions is reported. The dependent variable is the log of the pollutant concentration. Standard errors clustered at the station by month level in (), and clustered at the county by month level in [] (used for stars). N denotes the number of observations. All specifications contain weather and industry controls and pollution station, day-of-week, month-of-year and year fixed effects. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

(prenatal) exposure to pollution can affect health outcomes at birth. Second, the economic costs of (infant and fetal) deaths tend to overshadow non-fatal health costs in most existing cost-benefit analyses (EPA 2002; 2011c). Third, extensive infant health data are available from birth records.

Section 2 discusses evidence on negative effects of pollution on infant mortality and fetal health, typically measured by low birth weight or gestational age at birth. Another direct measure of fetal health is the occurrence of fetal death. Data on fetal deaths are often hard to obtain and incomplete (Sanders and Stoecker, 2011). Fetal deaths are by far the most likely during the first trimester of the pregnancy, and such early fetal losses are rarely officially recorded. Some states or countries require the reporting of late-term fetal deaths. Pereira *et al.* (1998) find a positive association between late-term intrauterine mortality (28 weeks of gestation or more) and prenatal exposure to CO and NO_2 in São Paulo, Brazil. California requires fetal deaths of 20 weeks gestation or more to be registered. Since many of these more developed fetuses would have been viable if born alive, the welfare costs of such late fetal losses should be of particular interest to policy makers.

Currie (2011) describes a selection mechanism that operates when using live birth data to estimate the impact of pollution on fetal health and birth outcomes. An increase in pollution leads to more fetal deaths, but also to the survival of fewer marginal, less healthy, fetuses. This decreases the number of less healthy infants whose birth weight and gestational age gets recorded. It is an empirical question whether this "harvesting effect" dominates the pollution-induced reduction in

average birth weight and gestational age among non-marginal fetuses.

Using the universe of birth records in California during the period 1984-1990, I estimate the impact of the 1987 speed limit changes on four infant health outcomes: fetal death, infant death, low birth weight (less than 2,500 grams) and (extreme) prematurity (gestational age 28 weeks or less). In the cost-benefit analysis (Section 10), I combine these results with estimates from the epidemiology literature to also take into account the effect of the speed limit changes on important adult health outcomes such as premature mortality due to pollution.

9.1 Econometric Framework

The zip code of the mother's residence during pregnancy is the finest available geographic entity in the data. I classify zip codes by the distance between their population-weighted average centroid and the closest highway where the speed limit changed (see Appendix A for details). To estimate the effect of speed limit changes on infant health, I use a difference-in-differences estimator similar to specification (8) for air pollution. Treatment zip codes have centroids at most x miles away from the 10 mph change; control zip codes' centroids are at least y miles away from the change ($y \geq x$). Given the lack of a theoretical basis for the "correct" buffer size, I report results for various buffer definitions and for a different zip code classification method. I employ the following specification:

$$health_{it} = \beta_0 + \beta_1 1(dist \leq x) * 1(t \geq t_{\Delta SL}) * TI + \beta_2 X_{it} + \theta_z + \theta_m + \theta_y + \varepsilon_{it}, \qquad dist \notin (x, y) \quad (9)$$

where $health_{it} \in \{fetal\ death, infant\ death, low\ birth\ weight, premature\ birth\}$ are binary variables indicating the health outcome for baby/fetus i at date t. X_{it} includes controls such as the race of the baby/fetus i and its parents, the mother's age, the amount of prenatal care, and medical complications during pregnancy and delivery. θ represents fixed effects for zip code z, month-of-year m and year y. TI is the treatment intensity that indicates how long the fetus was exposed to increased pollution. $TI = \frac{month}{9}$ where $month = 1$ if the date of birth falls within the first month after the speed limit change, 2 for the second month, etc. For all births in or after the ninth month following the speed limit change, $TI = 1$.

The treatment and control groups are similar in terms of the means of the covariates (Section 5.3). Table 21 in Appendix A shows that there are no differential trends in any of the control variables, except for a relative decline in the percentage of Hispanic newborns in the treatment areas after the speed limit changes.

9.2 Infant Health Results

Table 14 shows the regression results for the four infant health outcomes discussed above.[34] In short, the table presents evidence that the speed limit changes in 1987 resulted in more fetal deaths. I find

[34]Standard errors are clustered at the zip code level, to account for unobserved shocks at a regional scale. An example could be the opening or closing of a hospital, which could change the quality of prenatal care for many inhabitants of a zip code.

that the higher speed limits caused 17-45 additional fetal deaths per year in California, depending on the buffer specification. Of these, 4-36 are fetal deaths in the third trimester of the pregnancy, when the fetus – if born – is considered viable. Taking the average of the treatment coefficients in Table 14, the increased pollution leads to a 0.07 percentage point increase (+9.4%) in the probability of a third trimester fetal death. This relatively large effect is consistent with other findings that fetuses are very sensitive to pollution *in utero* (Sanders and Stoecker, 2011).

The fairly wide range reflects that the results are sensitive to the exact buffer definition. The air pollution results in Section 8 suggest that increases in background concentrations occur in three and five mile buffer zones, beyond which the effect diminishes. It is difficult to argue for a preferred buffer choice[35], but the estimates for the first two specifications in which $(x, y) = (3, 3)$ might be biased downward if much of the additional pollution spreads further than three miles. Ignoring these specifications, the additional fetal death range narrows to 23-45, with 11-36 additional fetal deaths in the third trimester. As a further robustness check, Table 26 in Appendix B summarizes the results using a buffer classification based on the percentage of zip code residents living within a particular distance from the highway (as opposed to the population-weighted average zip code centroid). The results are similar.

I find no significant impact on the other three health outcomes (infant death, low birth weight and premature birth). The effect on infant deaths (conditional on being born in the third trimester; premature birth is estimated separately) is about zero, while infant deaths among children born within the normal window (gestational age at least 37 weeks) increase slightly. The (statistically insignificant) estimates translate to 2-11 additional infant deaths per year. Given the coarse geographic information at the zip code level, it is not surprising that the effect is not statistically significant. Extrapolating Currie and Neidell's (2005) finding that infant deaths increase by 0.18 per 1,000 births for each ppm increase in the CO concentration, one should expect about two additional infant deaths per year from the increase in CO pollution following the speed limit changes.

The effects on low birth weight, premature birth and (by inference) infant deaths before 37 weeks are insignificant but often negative. A potential reason is the "harvesting effect" discussed above: more fetuses do not survive, instead of being born prematurely and/or with low birth weight. Conditional on surviving and on being born at full term, the risk of low birth weight increases.

I conclude that the speed limit changes led to a significant increase in fetal deaths. The effects on infant death risk are insignificant, but of the same order of magnitude as earlier estimates by Currie and Neidell (2005). Since I observe zip code rather than address level information about the mother's residence, the distance variables are measured with error. Because of attenuation bias, I interpret the coefficients as lower bounds on the true effect.

[35] I removed mothers living in zip codes more than 10 or 20 miles away from the treatment freeways, since unobservable differences between geographically distant areas render the treatment-control comparison less convincing.

Table 14: Regression Results for the Effect of the 1987 Speed Limit Changes on Infant Health, for Various Buffer Distances (California)

	$(x,y)=(3,3)$ $dist \leq 10$ (1)	$(x,y)=(3,3)$ $dist \leq 20$ (2)	$(x,y)=(3,5)$ $dist \leq 10$ (3)	$(x,y)=(3,5)$ $dist \leq 20$ (4)	$(x,y)=(3,10)$ $dist \leq 20$ (5)	$(x,y)=(5,5)$ $dist \leq 10$ (6)	$(x,y)=(5,5)$ $dist \leq 20$ (7)	$(x,y)=(5,10)$ $dist \leq 20$ (8)
Fetal death	0.00061	0.00072	0.00101**	0.00083*	0.00083*	0.00163***	0.00131***	0.00132***
(Gestational age ≥ 98)	(0.00050)	(0.00046)	(0.00052)	(0.00046)	(0.00047)	(0.00045)	(0.00038)	(0.00040)
Observations	418,099	1,027,017	333,980	942,898	718,105	418,099	1,027,017	802,224
Fetal death	0.00016	0.00028	0.00060	0.00041	0.00038	0.00131***	0.00104***	0.00102***
(Gestational age ≥ 196)	(0.00040)	(0.00035)	(0.00042)	(0.00035)	(0.00036)	(0.00039)	(0.00032)	(0.00033)
Observations	414,687	1,018,569	331,243	935,125	712,185	414,687	1,018,569	795,629
Infant death	0.00003	-0.00001	0.00006	-0.00001	-0.00002	-0.00007	-0.00006	-0.00006
(Gestational age ≥ 196)	(0.00067)	(0.00059)	(0.00071)	(0.00060)	(0.00060)	(0.00060)	(0.00046)	(0.00046)
Observations	412,777	1,014,103	329,714	931,040	709,132	412,777	1,014,103	792,195
Infant death	0.00010	0.00008	0.00028	0.00013	0.00009	0.00045	0.00033	0.00030
(Gestational age ≥ 259)	(0.00059)	(0.00054)	(0.00063)	(0.00054)	(0.00055)	(0.00051)	(0.00039)	(0.00040)
Observations	376,106	924,135	300,428	848,457	646,562	376,106	924,135	722,240
Low birth weight	-0.0024	-0.0023	-0.0021	-0.0023	-0.0022	-0.0006	-0.0007	-0.0006
(Gestational age ≥ 196)	(0.0021)	(0.0019)	(0.0023)	(0.0020)	(0.0020)	(0.0020)	(0.0016)	(0.0017)
Observations	412,777	1,014,103	329,714	931,040	709,132	412,777	1,014,103	792,195
Low birth weight	0.0022	0.0010	0.0026*	0.0010	0.0004	0.0020	0.0005	-0.0001
(Gestational age ≥ 259)	(0.0014)	(0.0013)	(0.0015)	(0.0013)	(0.0014)	(0.0012)	(0.0010)	(0.0011)
Observations	376,106	924,135	300,428	848,457	646,562	376,106	924,135	722,240
Premature birth	-0.00034	-0.00038	-0.00044	-0.00041	-0.00041	-0.00036	-0.00037	-0.00037
(Gestational age < 196)	(0.00062)	(0.00054)	(0.00063)	(0.00054)	(0.00055)	(0.00057)	(0.00047)	(0.00047)
Observations	414,444	1,018,101	331,051	934,708	711,911	414,444	1,018,101	795,304

Notes: The coefficient on the interaction term of 56 separate regressions is reported. Standard errors clustered at the zip code level in parentheses. Zip code, month-of-year and year fixed effects are included. Controls are included. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

10 Cost-Benefit Analysis

10.1 Methodology

The cost-benefit analysis requires four key ingredients. First, the various costs and benefits need to be quantified. Many are estimated directly in Sections 6-9 (time savings, accidents and infant health). To make the cost-benefit analysis more complete, I use my estimates of the travel speed and pollution increases to infer increases in fuel consumption, greenhouse gases and several effects on adult health. To do this, I use established relationships from the engineering and epidemiology literature. Second, I classify the various costs into private versus external. Third, having quantified a wide range of private and external costs and benefits, I value them using generally accepted literature estimates of, e.g., the value of time and the value of a statistical life. Since the range of such estimates is wide, I perform extensive sensitivity analysis. Fourth, I use the estimated standard errors to introduce uncertainty about several parameter estimates in a Monte Carlo simulation.

10.1.1 Quantifying the Trade-Off Between Costs and Benefits

Table 15 (upper panel) quantifies the trade-offs between the costs and benefits estimated in this paper, without taking a stance on their valuation.[36]

Table 15: Trade-Offs between Speed and Various Costs (Oregon & Washington Combined - 1987)

Increase in travel speed	1%	1%	1 mph	1 mph
	Directly estimated costs			
Change in fatal accidents	7.45%	4.66	12.31%	7.69
Change in incapacitating accidents	3.97%	15.36	6.56%	25.36
Change in non-incapacitating accidents	2.54%	26.55	4.19%	43.83
Change in property damage only accidents	2.23%	46.80	3.69%	77.27
Change in CO concentrations (ppm)	4.15%	0.06	6.86%	0.09
Change in NO_x concentrations (ppb)	1.75%	0.83	2.89%	1.37
Change in O_3 concentrations (ppb)	0.97%	0.27	1.59%	0.45
Change in infant mortality	0.41%	0.95	0.68%	1.57
Change in third trimester fetal deaths	1.59%	2.82	2.62%	4.65
	Indirectly estimated costs			
Change in gasoline consumption (million gallons)	1.33%	3.92	2.20%	6.47
Change in premature adult mortality	0.006%	3.70	0.010%	6.11
Change in respiratory hospitalizations	0.38%	8.35	0.62%	13.79
Change in asthma emergency room visits	0.19%	19.57	0.31%	32.32
Change in outdoor worker productivity ($2006/hour)	-0.13%	-0.02	-0.22%	-0.03

Notes: The numbers are calculated for the treatment highways in Oregon and Washington in 1987 (or the 5 mile buffer zone around them), although the estimates for health and pollution include data from California. The table shows trade-offs between the time benefits of higher speed, the various costs that are directly estimated in this paper (upper panel) and the costs that are inferred using a combination of estimates in this paper and engineering and epidemiological relationships (lower panel). The travel speed estimate is the arithmetic average of the central case estimates for the three states (+3.06 mph (CA 1996), +4.09 mph (OR 1987), +3.60 mph (WA 1996)).

The next step is to quantify other potentially important costs and benefits that are not part of my data set. First, I quantify the increase in fuel consumption and associated greenhouse gases.

[36] These parameters are estimated using different data sets, so I cannot directly compute elasticities and the associated standard errors.

Using estimates for the fuel economy of vehicles of different vintages as a function of speed (Davis *et al.*, 2010), I translate the estimated increase in travel speed into additional gallons of gasoline and tons of carbon emissions using the distribution of estimated speed increases (see Figure 6).

Second, I quantify the effect of increased pollution on several adult health outcomes. For that purpose, I rely on evidence from the health economics and epidemiology literature that the EPA uses to construct "concentration-response functions" to quantify health effects (EPA, 2010; 2011c). Specifically, I focus on premature adult mortality, which has accounted for a large majority of total health costs/benefits in previous cost-benefit analyses of air pollution regulations (EPA, 2002; 2011c). In addition, I quantify the effect of increased pollution on respiratory related hospitalizations, emergency room visits for asthma, and productivity of outdoor workers.[37] The lower panel of Table 15 shows the results. Appendix C describes the construction and sources of the concentration-response functions.

10.1.2 Private Versus Social Cost-Benefit Classification

Time savings fully accrue to the driver as private benefits. Increased fuel use is a private cost, while increased pollution and associated adverse health effects are external costs not taken into account by the driver. A driver need not be driving faster himself to experience higher accident costs of being on a road with a higher speed limit. A rational driver will consider his own increased risk of accidents and property damage, but ignore the risk imposed on others. I therefore split the effect on various types of accidents into private and external components. Fatalities from single-vehicle accidents (representing 52% of fatal accidents on highways) are treated as purely private if the driver or his passengers die in the crash. If a pedestrian or cyclist dies, this effect is treated as purely external. For n-vehicle accidents not involving pedestrians or cyclists, each driver bears a $1/n$ share of the total accident costs on average.

10.1.3 Valuing Costs and Benefits

I now turn to the valuation of the costs and benefits. The two most important inputs are the value of a statistical human life (VSL), and the value of (travel) time (VOT). There exists an extensive literature on the estimation of the VSL. Most studies use revealed preference methodologies in which a mortality risk premium is estimated using wages for workers with different occupational risk, or prices of houses in locations with a different risk of pollution related mortality. An authoritative source is the meta-analysis by Viscusi and Aldy (2003), who report a mean VSL of about $7 million and a standard deviation of $5.6 million based on 49 studies. This demonstrates the considerable variation in VSL estimates. More recent papers find similar mean estimates for the VSL (Aldy and Viscusi, 2008; Kniesner *et al.*, 2011). The EPA currently prescribes a value of $6.3 million (2000 USD) for policy evaluation, which is largely based on the studies mentioned above. I use this value

[37] Table 27 in Appendix C summarizes costs and benefits related to pollution and health that are not quantified in this study. To the extent that the EPA has quantified these effects in their valuation studies, the effects are orders of magnitude below the costs that I have included.

as the central case estimate, but also perform sensitivity analysis.

The second key input is the value of travel time. Traditionally, time savings were valued at the average wage of the relevant population. An often quoted paper is Deacon and Sonstelie (1985), who studied a gasoline price ceiling as a natural experiment. Motorists could choose between waiting in line for low-priced (price capped) gasoline and purchasing higher-priced (not price capped) gasoline without waiting. Their implied VOT is close to individuals' after-tax average wages. Small *et al.* (2005) use combined revealed and stated preference data on people's choices whether or not to pay a toll for travel in a congestion-free express lane. Using revealed preference data, they find that the VOT is 93% of the average wage. They note that this is on the upper end of the range of existing estimates, potentially because commuting time is valued relatively highly and their study area is affluent.[38] In earlier work, Small (1992) reported a VOT of 20-100% of the average wage. In a recent study, Wolff and Watkins (2011) estimate the effect of gasoline prices on unconstrained driving speeds, and find that the implied VOT is 54% of the average wage rate.

The estimates by Deacon and Sonstelie (1985) and Small *et al.* (2005) may not be fully representative of the VOT in the current study for two reasons. First, they estimate a slightly different VOT. Waiting in line at a gas station or in congestion could be perceived differently from spending more time driving at a somewhat lower speed. If driving in congestion is particularly stressful, drivers should pay more to avoid congestion time than to avoid time spent in flowing traffic.

Second, it is well established that the VOT varies by trip purpose. In a meta-analysis of VOT studies, Wardman and Abrantes (2011) analyze 1,749 valuations from 226 studies. They find that commuting time is valued 12-35% more than driving time for leisure or personal affairs. Business travel time is valued more than twice as highly as commuting time. The Victoria Transport Policy Institute presents an overview of VOT adjustments by trip purpose as employed by various governments. The report proposes to value business travel at 150% of the average wage, commuting at 50%, personal errands at 25%, and leisure/vacation at 0% (VTPI, 2011). Based on various literature estimates, the U.S. Department of Transportation currently recommends valuing business travel at 100% of the average wage, all personal local travel at 50% and all personal intercity travel at 70%.[39] While many studies acknowledge that the VOT should be lower for children, the elderly, and the unemployed, they do not provide numerical estimates. I use the average wage as the central case value of travel time, consistent with the Small *et al.* (2005) and Sonstelie and Deacon (1985) findings. I also show results using the U.S. DOT and the age-adjusted VTPI guidelines.

Table 16 lists the key valuation parameters. In addition to the parameters discussed above, I use broadly accepted estimates for non-fatal health costs. The EPA's numbers for hospitalization and emergency room visit costs only include medical expenditures and opportunity costs (lost wages), but not lost quality of life. The National Safety Council's non-fatal accident costs do include the cost of reduced quality of life.

[38] Their estimates using stated preference data are much lower. This is consistent with Calfee *et al.* (2001)'s finding that the VOT is 14-27% of the average wage when stated preference data are used. Such estimates are not generally considered reliable due to doubts about the generalizability of hypothetical to real-world choices.

[39] See http://regs.dot.gov/docs/VOT_Guidance_Revision_1.pdf.

Table 16: Cost-Benefit Valuation Parameters (Oregon & Washington Combined - 1987)

Parameter	Value	Source
Travel time valuation		
Average hourly after-tax wage	\$15.37	Current Population Survey (CPS); NBER
Fatal accidents and health impact valuation		
Value of a statistical human life	\$7,375,305	Environmental Protection Agency (EPA, 2011c)
Cost of a respiratory hospitalization	\$27,496	Environmental Protection Agency (EPA, 2011c)
Cost of asthma emergency room visit	\$438	Environmental Protection Agency (EPA, 2011c)
Non-fatal accidents valuation		
Cost of an incapacitating injury	\$319,272	National Safety Council (NSC)
Cost of a non-incapacitating injury	\$77,866	National Safety Council (NSC)
Cost of a property damage accident	\$7,705	National Safety Council (NSC)
Gasoline cost valuation		
Pre-tax gasoline price	1.70\$/gallon	Energy Information Administration (EIA)
Social cost of CO_2	48.10\$/tonne	Intergovernmental Panel on Climate Change (IPCC)

Notes: All values are expressed in 2006 USD. CPS: wage data obtained from http://cps.ipums.org/cps/. The hourly wage is inferred from annual income and hours worked per year. Observations with an hourly wage below \$2.50 or above \$100.00 per hour are dropped. NBER: average federal and state tax rate data obtained from http://www.nber.org/\$\sim\$taxsim/ally/. NSC: valuations obtained from http://www.nsc.org/news_resources/injury_and_death_statistics/Pages/EstimatingtheCostsofUnintentionalInjuries.aspx. The non-economic (QALY) part of the costs for incapacitating and non-incapacitating injuries is scaled by the ratio between the EPA's VSL and the NSC's VSL (\$4,300,000). EIA: gasoline price data available at http://www.eia.gov/petroleum/data.cfm#prices. IPCC: Working Group III contribution to the Fourth Assessment Report (2007), available at http://www.ipcc.ch/publications_and_data/ar4/wg3/en/contents.html.

10.1.4 Introducing Uncertainty About Costs and Benefits

There is considerable uncertainty about both the magnitude of the various effects and the parameters used to value them. I therefore perform a Monte Carlo simulation in which I take into account uncertainty in two ways. First, I draw realizations of the various outcome variables using the standard errors of the coefficients estimated in Sections 6-9, assuming a normal distribution.[40] Second, I take into account uncertainty about external parameters taken from the literature by specifying scenarios with low, central and high values.

10.2 Results

10.2.1 Private Versus Social Cost-Benefit Results

I now show the cost-benefit calculations evaluated at the central case parameter values in Table 16 and using the central health impact scenario in Appendix C. Annual net social benefits are estimated at -\$329 million. The social costs (\$486 million) exceed the benefits (\$156 million) approximately three times. A useful related metric is the VSL that equates the expected costs and

[40] One complication of this approach is that the treatment effects in this paper are estimated using separate data sets. This makes it impossible to estimate a joint variance-covariance matrix from which the realizations of the treatment effects can be drawn. In the Monte Carlo simulation, I draw the realizations from independent normal distributions. The true variance of the net benefits may be either larger or smaller than the simulated variance. Since the same unobserved shocks could affect both travel speed (benefits) and, e.g., accidents (costs), the simulated variance may be too high. However, assuming independence across various correlated costs introduces a downward bias in the simulated variance. To mitigate this concern, I estimate a joint variance-covariance for those costs for which this is possible: accidents. I bootstrap the sample of all reported accidents (with replacement), and re-estimate specification (6) for the four types of accidents 1,000 times. I then compute the variance-covariance matrix.

benefits of the speed limit changes. This number could be interpreted as the upper bound of the social planner's VSL, if the realized social costs and benefits were in line with ex-ante expectations. To justify higher speed limits, the social planner's VSL has to be below this upper bound. Likewise, private VSLs below the private VSL upper bound justify driving faster as a result of the higher limit. Table 17 reports these upper bound VSLs.

Table 17: The Difference Between the Social and Private Trade-Off of Faster Driving

	Social trade-off:					Private trade-off:	
Value of a statistical life below which:	Higher speed limits are justified					Driving faster is justified	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	5.08	3.70	2.41	0.86	-0.09	6.02	2.36
Using the following costs:							
Fatal accidents (private)	✓	✓	✓	✓	✓	✓	✓
Fatal accidents (external)	✓	✓	✓	✓	✓		
Non-fatal accidents (private)		✓	✓	✓	✓	✓	✓
Non-fatal accidents (external)		✓	✓	✓	✓		
Fuel costs (private)			✓	✓	✓	✓	✓
Climate damages (external)			✓	✓	✓		
Infant and adult health (external)				✓	✓		
More conservative time valuation					✓		✓

Notes: The upper bound VSL (the VSL that equates expected costs and benefits) is expressed in million 2006 USD. More conservative time valuation corresponds to the U.S. DOT guidelines.

Columns (1)-(4) in Table 17 show the upper bound VSL for a government that takes into account various subsets of social costs. If, in line with previous literature, governments take into account (private and external) fatal accident costs only, the upper bound VSL is \$5.08 million. However, when the set of costs taken into account gets more complete, the upper bound VSL decreases dramatically to the central case social estimate of \$0.86 million. Column (5) shows that when time benefits are valued using the U.S. DOT guidelines rather than at the average wage, the upper bound VSL is negative. This means that the non-fatal social costs of the speed limit changes alone (non-fatal accidents, fuel costs, climate damages and non-fatal health costs) exceed the benefits. Figure 10 provides more details. From columns (4) and (5), I conclude that raising the 1987 speed limits was not a good decision ex-post from a societal perspective. Moreover, columns (1)-(4) provide justification for estimating the effects on outcome variables beyond fatal accidents.

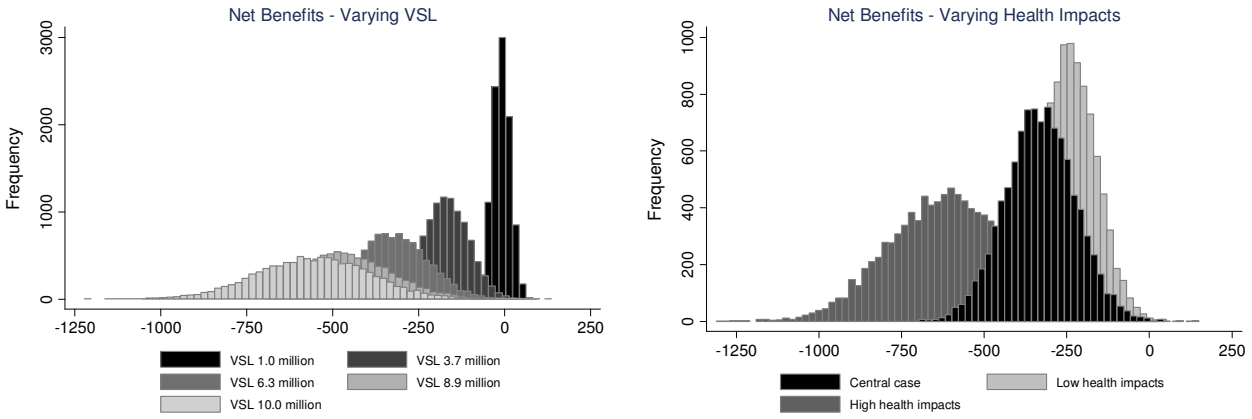
The trade-off is quite different from a private perspective. Privately, VSLs of \$6.02 million or less (column (6)) justify driving faster as a result of the speed limit increase. This value is well within the conventional VSL range and close to the EPA's central value. Given that drivers are heterogeneous in their assessment of the VSL, accident risks and fuel costs, driving faster appears rational for many individual drivers. Applying the U.S. DOT time savings discount, the upper bound VSL decreases to \$2.36 million (column (7)). While this is below the EPA's value, it is still within the range of VSLs in other studies (Viscusi and Aldy, 2003) and in sharp contrast with the negative value in column (5). I conclude that there is a large difference between the social and private optimal speed choices: driving faster appears a rational choice for many drivers but a poor

outcome for society as a whole.[41] Although speed limits are crude rules, this large difference makes it highly unlikely that any hidden administrative costs or unforeseen behavioral adjustments could make completely eliminating speed limits an efficiency-improving proposition.[42]

10.2.2 Simulations and Sensitivity Analysis of Net Benefits

Figure 9 shows the uncertainty around the estimate of net social benefits using Monte Carlo simulations for various VSLs (left panel) and the three health impact scenarios discussed in Appendix C (right panel). The three intermediate VSLs correspond to the EPA's low, central and high estimates. $1 and $10 million are more extreme values. Besides differences in the parameters of the concentration-response functions, the health impact scenarios differ in their treatment of fetal deaths. The low and central health impact scenarios value fetal deaths conservatively at $0. The high health impact scenario values third trimester fetal deaths at the VSL (but second trimester fetal deaths at $0). Travel time is valued at the average after-tax wage for all trips.

Figure 9: Monte Carlo Simulations of the Net Benefits of the Speed Limit Changes in 1987



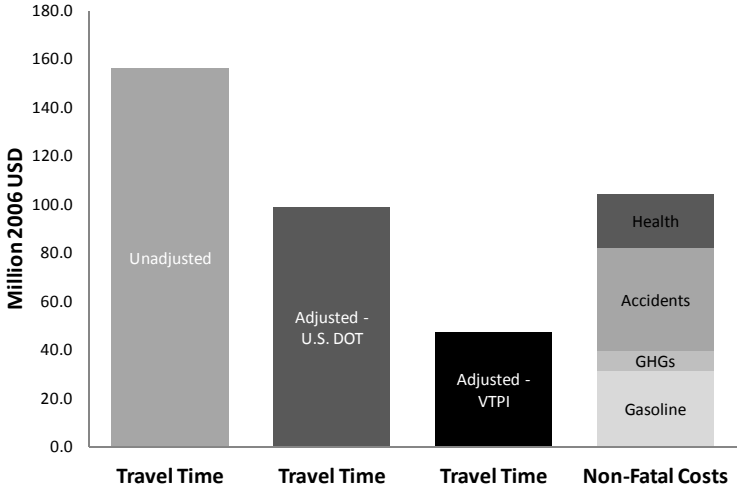
Notes: Histograms show frequencies for 10,000 realizations of the costs and benefits of the speed limit changes in Oregon and Washington in 1987. Results are graphed for five VSLs (left panel) and for three health impact scenarios (right panel). VSL values are expressed in 2000 USD. $6.3 million is the central case (EPA) estimate.

[41] The reported spread between private and social net benefits is conservative. I count all single-vehicle non-pedestrian/cyclist crashes as fully internalized. In reality, other "phantom" vehicles were involved in many of these crashes, but the police had no way to verify this and correctly report the accident as a multi-vehicle crash (National Institute for Safety Research, personal communication, 30 September 2011). For example, if this applies to 50% of the single-vehicle accidents, the private upper bound VSL increases to $7.04 million.

[42] Removing the speed limit (as opposed to raising the limit to 65 mph) would be better than keeping a 55 mph limit only under extreme assumptions about how drivers would react to being given full speed discretion. First, removing the speed limit could lead to a *lower* average speed. Experiences on the German autobahns dismiss this possibility. Second, even if removing the speed limit would lead to a higher average speed (and thus pollution and health impacts), drivers could adopt safer driving habits. For instance, drivers might drive more slowly during adverse weather conditions now that the speed limit is gone as a bad focal point. This "safer driving effect" would have to be so large that it leads to a *reduction* in accidents, as opposed to the sharp increase observed after establishing a 65 mph limit. If the increase in speed were similar to the effect I find for the new 65 mph limit, fatal accidents would have to go down by 17% (instead of increase by 44%) to improve on the old 55 mph limit. This is highly implausible.

The figure shows 10,000 replications of the annual net social benefits of the speed limit changes for Oregon and Washington in 1987. As discussed above, the central case mean estimate is -$329 million. The standard deviation is $106 million. Only in 0.10% of the replications did net social benefits turn out positive. The conclusion that net social benefits are negative is robust to a wide range of VSL assumptions. Even at a VSL of $1 million (left panel), which is widely considered too low, the expected costs exceed the benefits by $9 million. The probability that the net benefits are positive is estimated at 37.13%. The right panel shows the three health impact scenarios. Even in the low health impact scenario, the net benefits are estimated at -$242 million, with a probability of exceeding zero of only 0.17%. In that scenario, the upper bound VSL equals $1.13 million. In the high health impact scenario, the upper bound VSL equals $0.49 million.

Figure 10: Travel Time Benefits and Non-Fatal Costs for the Speed Limit Changes in 1987



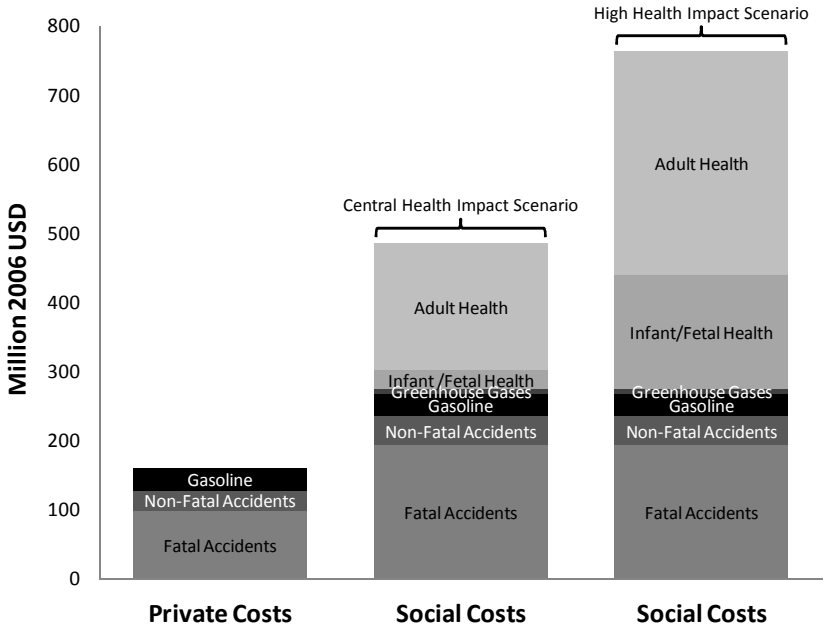
Notes: GHGs denotes greenhouse gases. Benefits and costs evaluated at the central health impact scenario.

Valuing time using the U.S. DOT or VTPI guidelines would reduce the net benefits by another $57-109 million.[43] The costs now exceed the benefits by a factor of 5 and 10, respectively. Figure 10 shows the expected travel time benefits for the three time valuation scenarios versus the *non-fatal* costs (e.g., all costs excluding fatal accidents and infant/fetal/premature deaths). Valuing time according to the U.S. DOT or VTPI guidelines has another important implication: the travel time benefits are outweighed by non-fatal costs alone. This is true regardless of the health impact scenario chosen. This illustrates the negative upper bound VSL in column (5) of Table 17.

[43] I use the adjusted 1990 data from the 1995 National Personal Transportation Survey to compute the fraction of person miles by trip purpose around 1990. This yields 19.6% (commuting), 8.2% (business travel), 5.0% (school/church), 30.8% (family/personal), 22.4% (leisure/vacation) and 13.4% (visiting friends/relatives). Following the U.S. DOT guidelines, I value business travel time at the average wage, and all other purposes at 60% of the average wage (the average of the 50% and 70% prescribed for local and intercity traffic). In the VTPI scenario, I value business travel time at 150% of the average wage, commuting time at 50%, school/church/family/personal/relatives/friends at 25%, and leisure/vacation at 0%. In addition, I multiply the VOT for people aged above 65 or between 16 and 19 by 50%, and for younger children by 25%.

Figure 11 shows the stark contrast between private and social costs. Social costs are 2.5, 3 and 5 times as high as private costs under the low (not shown), central and high health impact scenario, respectively. The figure also shows the relative importance of the various costs. Fatal accidents account for 40% of total costs under the central health impact scenario. However, pollution related premature mortality for adults is almost as large a cost component (38%). Non-fatal accidents (9%), gasoline and climate costs (8%) and infant/fetal health (5%) also cannot be ignored in a cost-benefit analysis. Under the high health impact scenario, fatal accidents only constitute 25% of total costs, with adult health (42%) and infant/fetal health impacts (22%) being the other two major cost components. In the low health impact scenario (not shown), fatal accidents constitute 48% of total costs. This underscores that only including fatal accidents oversimplifies the cost-benefit analysis, potentially leading to incorrect conclusions.

Figure 11: Private Versus Social Costs of the Speed Limit Changes in 1987



Notes: Costs are evaluated at the the central (middle bar) and high (right bar) health impact scenario.

10.3 Comparison with Existing Literature

Table 18 compares my findings with the results from Ashenfelter and Greenstone (AG; 2004), who find an upper bound VSL of $1.54 million (1997 USD) for the full sample, but higher estimates for California ($4.75 million) and Oregon ($5.41 million). AG neither make upward adjustments to their VSL estimates based on average vehicle occupancy, nor downward adjustments because travel time might be valued below the average wage. A fair approach – adopted in Table 18 – compares my central case upper bound VSL (time savings valued at average wage, and incorporating the average vehicle occupancy) with AG's occupancy-adjusted VSL.

I conclude that when time is valued at the average after-tax wage, accounting for costs beyond

Table 18: Comparison of Upper Bounds on VSL Estimates

	This paper OR & WA	This paper OR & WA	This paper OR & WA	AG (2004) Full sample	AG (2004) CA	AG (2004) OR
Time valued at average wage	0.86	6.02	5.08	3.28	10.11	11.52
U.S. DOT time valuation	-0.09	2.36	3.22	2.07	6.40	7.29
VTPI time valuation	-0.94	-0.94	1.53	0.99	3.05	3.47
All social costs	✓					
Private costs only		✓				
Social cost of fatal accidents only			✓	✓	✓	✓

Notes: The upper bound VSL is (the VSL equates expected costs and benefits) is expressed in million 2006 USD and calculated using the central health impact scenario. AG estimate for WA not available. AG estimates adjusted upward by the average vehicle occupancy rate in 1987 (1.695 persons per vehicle; source: National Personal Transportation Survey 1995).

fatal accidents can change the conclusion from the social cost-benefit analysis. While AG's full sample upper bound VSL estimate ($3.28 million) is on the lower end of conventional VSL estimates, their estimates for California and Oregon would lead to the conclusion that the speed limit increases in 1987 were beneficial from a societal perspective. This paper's upper bound VSL of $0.86 million rejects that conclusion. However, when I only include fatal accident costs, the social trade-off is more similar to the results obtained by AG.

If time savings are valued according to U.S. DOT guidelines, AG's estimates for California and Oregon provide only weak guidance on whether speed limits should have been changed, whereas this paper's results strongly decide against raising the speed limits. Using VTPI guidelines, both this paper and AG rule against the 1987 speed limit increases. These results indicate that the additional costs estimated in this paper have the potential to reverse the conclusion about whether or not speed limits should be increased.

11 Discussion

11.1 External Validity

This paper finds that 55 mph was a better speed limit than 65 mph[44] and that there was a large difference in private and social net benefits, using speed limit changes between 55 and 70 mph for mostly rural freeways in the western United States in 1987 and 1996. Whenever comparing across time, state and urban/rural roads was possible, the estimates appeared stable. This suggests that the findings are likely to apply to speed limit changes on similar highways and in similar states in the same period. However, it is reasonable to ask how these findings can be relevant for speed limit changes today, in different countries, and for a different speed range. This is especially relevant because various countries and states are currently debating speed limit changes anywhere in the 55-90 mph range. I discuss two questions. First, how would the various effects of speed limit changes be different today? Second, how would the difference between private and social net benefits change?

[44]This does not imply that the optimal speed limit was much below 55 mph. While I cannot calculate the optimum because I estimate a "slope" at 55 mph but no "curvature", the relationship between speed and pollution becomes quite flat below 55 mph. Therefore, driving slower will not yield large pollution related health benefits.

Regarding the first question, it is possible to extrapolate the pollution estimates using past and current speed-emissions profiles. Such information is available. Cars have become less polluting over the past two decades. The EPA MOVES model's most recent estimates for the current vehicle fleet show that the U-shaped relationship in Figure 7 is still present but less steep. It starts curving upwards sharply around 65 mph instead of 55 mph for the 1990 vehicle fleet (EPA, personal communication, 4 May 2011). The relationship between speed and fuel consumption is still increasing but shifted to the right by approximately 10 mph. This information, combined with up-to-date epidemiological studies, makes extrapolation of pollution and health effects possible. A 55 to 65 mph speed limit increase would lead to smaller pollution and adverse health effects today. These costs would rise again for the currently proposed 65-75 mph and 75-85 mph increases, for which the upward relationship between speed and pollution remains even for today's vehicles.

Extrapolating the effect on accidents is more difficult. The relationship between speed limits and accidents may well be non-linear. Given the recent speed limit changes in the 65-90 mph range and detailed data availability, it will eventually be possible to re-estimate the relationship between speed limits, speed and accidents at higher speeds.

The discussion about extrapolation implies that today's gap between private and social net benefits will be smaller for a 55 to 65 mph speed limit increase. For higher speed limits, the gap is likely to remain substantial because of the steeper speed-emissions profile in that range and the external cost component of accidents. Several factors influence the ratio of private and social costs. Fuel costs are higher than in 1987. This increases private costs relative to external costs. Changing speed limits on urban rather than rural highways achieves the opposite, because the density of the surrounding population that gets exposed to higher pollution levels is higher.

Finally, certain emerging economies have vehicle fleets and roads that bear many similarities to the United States in 1987. The estimates in this paper could be extrapolated to make predictions about costs and benefits of changing speed limits in such countries.

11.2 Interpretation of Cost-Benefit Results

The cost-benefit analysis concludes that, at least ex-post, the Western states should not have raised their speed limits in 1987, and that the implied social upper bound VSL ($0.9 million) is well below the implied private upper bound VSL ($6.0 million). This raises the following questions.

First, why did governments raise speed limits when the costs were three to ten times larger than the benefits? One potential explanation is that governments did not behave as benevolent social planners, and responded to the private desires of some of their constituents, who may have wanted to drive faster. Another – at least theoretical – possibility is that governments were benevolent social planners who thought that the correct VSL was below $0.9 million. However, Figure 10 shows that with a modest reduction in the valuation of travel time (as suggested by both academic literature and governments), the speed limit increases do not even pass the cost-benefit test at a zero VSL. I find it more plausible that a lack of information and incomplete decision making explain the decision to raise freeway speed limits. Governments may have looked at previous speed limit

changes to analyze the impact on travel speed and fatal accidents, but probably did not perform any sophisticated econometric estimation. In addition, non-fatal accidents, pollution and health impacts were hardly or never mentioned in any official documents. The scientific evidence on the effect of pollution on health was largely unavailable in 1987 (see, e.g., Table 28). This leads to a serious underestimation of the total costs. This combination of the absence of a well-founded prior about the effect on fatal accidents and the lack of knowledge about other costs could explain why the cost-benefit trade-off may have looked very different to the government in 1987.

This in turn raises a second issue: is the private trade-off more suitable to infer a VSL upper bound? One view is that if governments decide on trade-offs between money (time) and a small change in mortality risk based on incomplete or wrong information, individuals may do so too. This would invalidate the revealed preference approach to estimating the VSL using either the social or the private trade-off. However, the private trade-off involves fewer factors (travel time, own accident risk, and gasoline consumption) than the more complicated social trade-off which includes pollution and health effects. If drivers have a reasonably accurate prior of the distribution of their private costs and benefits, their choices should be preferred as a means to estimate the VSL.

12 Conclusions

In this paper I estimate the private and external costs and benefits of driving faster on freeways, and investigate if the difference between the individual driver's and social planner's speed choices is large enough to justify a crude policy instrument such as a speed limit. Using variation in speed limits in California, Oregon and Washington in 1987 and 1996, I find that the social costs of raising speed limits in 1987 (from 55 to 65 mph) were 3 to 10 times higher than the benefits. The social costs were also 2.5 to 5 times larger than the private costs. Values of a statistical life below \$6.0 million justify a private driver's decision to drive faster after a speed limit increase. Such VSLs are within the conventional range. In contrast, the social planner's VSL would have to be less than \$0.9 million to justify raising the speed limits. This value is considerably below the estimates in most academic papers and government guidelines. I conclude that private and social net benefits differ substantially: many individual drivers rationally chose to drive faster when they could, but society should not have opted for the higher speed limits. The large wedge between private and social optimal speed choices provides a strong rationale for having speed limits.

This conclusion does not rule out that other instruments could deal with speed externalities more effectively. An ideal Pigovian tax on speed would consist of a combination of a gasoline tax for climate damages, emissions taxes for local air pollutants in exhaust gas (which vary with speed), plus a speed-dependent tax to internalize accident risk imposed on others (which is also a function of traffic conditions). A gasoline tax exists, real-time emissions taxes could conceivably be implemented using sophisticated inboard computers in vehicles, but a speed tax on accidents would face enormous informational requirements and technical challenges. Therefore, speed limits are likely to remain the dominant policy instrument for the foreseeable future.

Flexible speed limits that vary by time-of-day and road conditions provide an interesting avenue for future research. Several states and countries have introduced a network of real-time speed, congestion and accident monitoring on thousands of highway locations. This provides an opportunity to use recent speed limit changes to inform policy makers about how such flexible speed limits should be set, using detailed estimation of the heterogeneity in the effect of higher speed limits on speed and accidents. The availability of a single data set with speed limit, speed and accidents information could also be used to uncover the mechanisms through which higher speed limits lead to more accidents. For instance, it would be possible to test if the effect on accidents is driven by an increase in the average speed, or rather an increase in the variance in the upper or lower end of the speed distribution.

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Appendix A: Data

Speed Limit Changes

Information on the exact timing and location of historical speed limit changes is surprisingly difficult to collect for two reasons. First, this information is not stored in a digital database. Second, the official date of approval does not necessarily correspond to the date that the new road signs were erected. The latter information is not stored in archives. I obtained complete lists of speed limit changes for 1987 using FOIA requests and by visiting local offices. These 29 highway segments are

indicated by milepost and (sometimes) a location description. Using this information, I geo-coded these segments in ArcGIS using a detailed shapefile of U.S. highways (source: Esri Data and Maps).

Speed limit changes in 1996 have been poorly documented by state agencies. In California, only a few of the 12 Caltrans districts have retained full records of these changes. In Washington, full records have been retained for the speed limit changes on interstates, but not on other highways. Oregon did not change its speed limits until 2004, when 23 freeway miles went from 55 to 60 mph. I therefore restricted subsequent FOIA requests to the speed limits pertaining to the specific locations of the speed monitoring stations with data coverage around 1996.

Speed

While extensive real-time speed monitoring has led to unprecedented data availability for recent years, information on speed measurements before 2000 has mostly been purged from state databases. With generous help from the three Departments of Transportation, I have been able to recover detailed speed measurements by speed bin (in increments of 5 mph), by year-month-day-hour, by direction (north, south, east, west), by lane. For example, I observe that for the "Olympia" station in Washington, on the I-5 northbound, in the left lane, 366 vehicles traveled at a speed between 55 and 60 mph, on 2 January 1994, between 6-7 PM. This information is then used to compute average and 85th percentile speeds (by day or by hour), by assigning the midpoint of each speed bin to the number of vehicles within that bin. The speed variance can be computed under the same assumption, but is imprecise due to the truncation of the lowest speed bin (0-30 mph).

Speed data for California come from an archived digital database of summary reports, currently discontinued by Caltrans and replaced by the real-time speed monitoring network PeMS (http://pems.dot.ca.gov/). Similarly, WSDOT retrieved speed information from old speed monitoring reports. ODOT's old speed data is partly available in digital format (1988-1992) and partly in scanned paper format (1983-1987). Using optimal character recognition, I transformed the scanned reports to digital files, which were then cleaned by a data services company. For a few stations, ODOT measured speed by radar (as opposed to dual loop detectors below the highway surface) and reported them in tally sheets. I manually translated these tally sheets to match the format of the rest of the data set.

To control for weather conditions in the speed regressions, I constructed weather variables (maximum, minimum and average temperature, wind speed, and indicator variables for fog, rain, snow, hail, thunder and tornado) for each speed monitoring station. First, I geo-code the speed monitoring stations based on their location descriptions. Then, I calculate the distance between every speed monitoring station and every weather station from the National Climatic Data Center's "Global Summary of the Day". For each speed station, I use the observations from all weather stations within a 20 mile radius, and compute the inverse distance-weighted average.

Accidents

Oregon and Washington have retained a database with all major accidents since since 1985 (OR) and 1980 (WA). In Oregon, accidents are only included if at least one person involved suffered injury or death, or if the property damage exceeded $500 (up to 8/31/1997) or $1,000 (between 9/1/1997 and 12/31/2003). Washington also used a $500 threshold up to 2003, after which it increased to $700. The relevant variables for the accidents analysis are:

- date, time and day-of-week
- type (fatality, incapacitating injury, non-incapacitating injury, property damage)
- location (highway number, milepost, city, county)
- highway functional class (e.g., rural interstate, rural principal arterial, urban interstate, urban principal arterial, other urban freeway)
- indicator variable for urban or rural status
- road characteristics: indicator variables for intersection, straight road, lane transition, curve, grade, bridge, tunnel, type of divider, dry, wet, snow, ice, sand, oil, water, construction zone, off roadway crash
- weather: indicator variables for clouds, rain, sleet, fog, snow, dust, smoke, ash, wind
- daylight: indicator variables for daylight, dark and lit, dark and unlit, dawn, dusk
- indicator variables for alcohol involved, drugs involved

Urban interstates, urban principal arterials and other urban freeways are combined into one class "urban highways". Accidents on minor arterials, collectors and local roads are excluded. Using information on highway functional class and VMT data from the Federal Highway Statistics, I assign a year by class specific VMT to each accident. Finally, I match the accident location information with the data on speed limit changes to determine whether or not the speed limit on each location had changed. This allows me to assign each accident a treatment or control status.

To split the costs of accidents into private versus external (Section 10), I use the Fatal Accident Reporting System (1984-1990) to distinguish fatal accidents by number of vehicles involved, number of fatalities, and whether or not a pedestrian or cyclist died (Table 19). Single-vehicle accidents are defined as happening in isolation of other vehicles (crashes including "non-contact vehicles" that were not damaged but still involved are classified as multi-vehicle crashes). For non-fatal accidents, I use similar information from the Oregon and Washington accident databases.

Table 19: Fatal Accident Statistics by Number of Vehicles Involved

Number of vehicles involved	Percentage of fatal accidents	Fatalities per accident	Percentage pedestrian cyclist deaths
1	52.1%	1.07	36.0%
2	39.4%	1.19	2.9%
3	6.5%	1.23	2.9%
4 or more	2.0%	1.33	3.5%

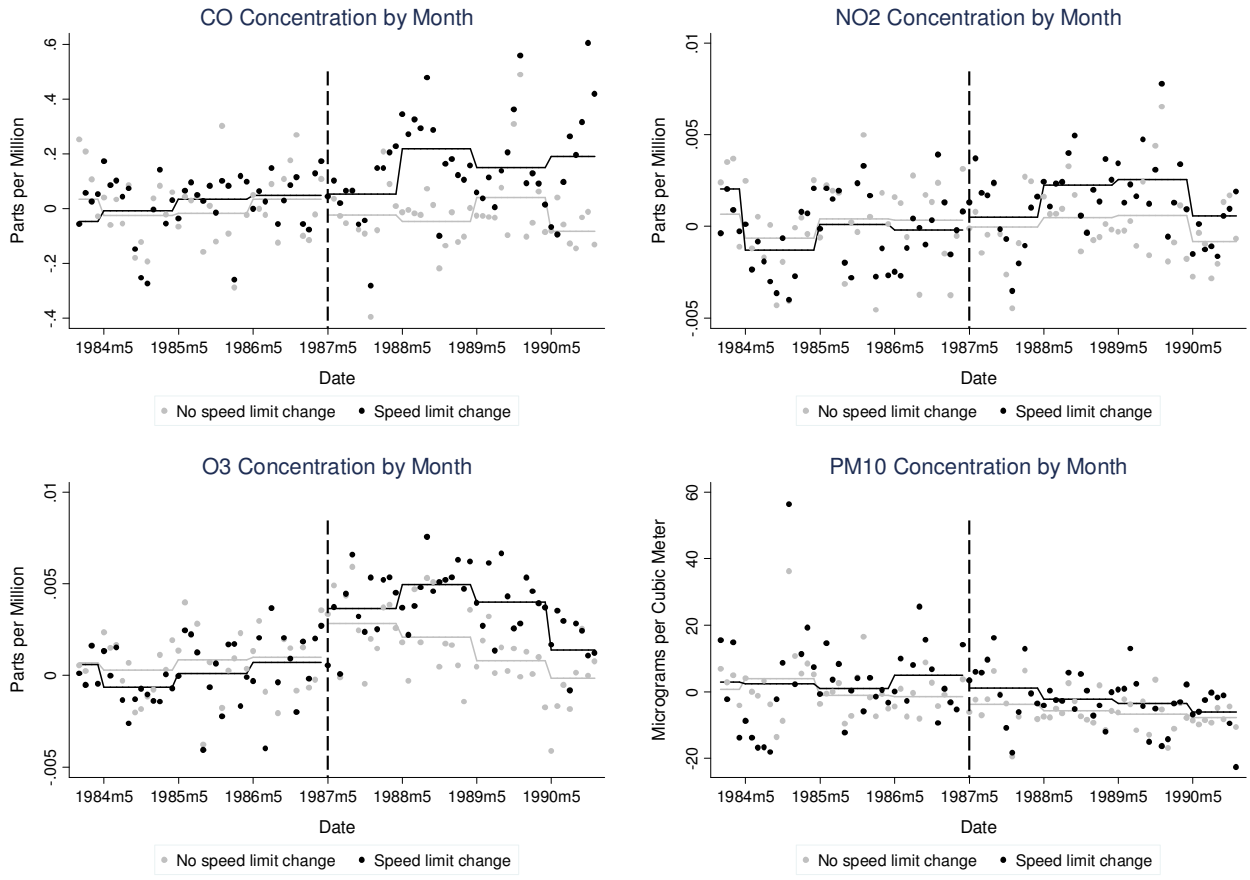
Notes: Source: Fatal Accident Reporting System, 1984-1990.

Air Quality

I augment the air quality data in two ways. First, I assign to each pollution monitoring station the inverse distance-weighted average of the weather variables, similar to those constructed for the speed stations. Second, I calculate the distance between each pollution station and the nearest point on any of the treatment highways. The distance is calculated in ArcGIS using the coordinates of the pollution stations and the geo-coded treatment highways. The pollution stations are then classified into treatment and control stations based on this distance.

Figure 12 shows that pre-existing trends in the various pollutant concentrations are similar for the treatment and control stations.

Figure 12: Trends in Air Pollution Before and After the Speed Limit Increases in California, Oregon and Washington (1987)



Notes: Gray dots indicate the monthly average air pollution concentration for control stations, black dots for treatment stations. Treatment stations are located less than 5 miles from a highway with a speed limit change. Concentrations are relative to the pre-treatment average concentration. All data are de-seasonalized at the monthly level. Solid lines are predicted values from regressions of pollutant concentrations on year dummies. Vertical dotted lines indicate the time of the speed limit change in California (1 May 1987).

Infant Health

The California birth cohort files contain information from all birth, infant death and fetal death records. The fetal death records are collected separately from live births on a fetal death certificate. Doctors are required to register fetal deaths of 20 weeks gestation or more. Very few fetal deaths are (voluntarily) reported prior to 20 weeks. Abortions are registered separately and not included in the fetal death data. In the infant health regressions, I use the following outcome variables for the period 1984-1990:

- low birth weight: birth weight less than 2,500 grams
- (extreme) premature birth: gestational age below 28 weeks (196 days)
- infant death (in the first year of life)
- fetal death in the second or third trimester

I use all potential control variables that are available for the entire period:

- infant characteristics: sex, race and Hispanic origin
- mother characteristics: race, Hispanic origin, age and age category (up to 18 years, 19-25 years, 26-30 years, 31-35 years, 36+ years)
- father characteristics: age, indicator variable for racial difference between parents
- pregnancy characteristics: month that prenatal care began, number of pregnancy complications, number of delivery complications, number of congenital anomalies; indicator variables for multiples, first trimester prenatal care, Cesarean section
- mother's medical history: total children born alive; indicator variables if the mother ever had a termination before 20 weeks, and if she ever had a termination after 20 weeks

In addition, the difference-in-differences estimation requires information on time and geography: date of birth, date of (fetal or infant) death, and the mother's residential zip code.

I use the mother's residential zip code in the birth cohort files to assign each record a distance to a treatment freeway, and to match it to the 1990 census variables. There is no exact match between the zip codes from these two sources, since several zip codes listed in the 1984-1990 birth cohort files had changed (or been re-numbered) compared to the 1990 census zip code list. Still, 96.0% of the birth records could be matched. I perform a manual matching procedure using old copies of the USPS's National Five-Digit ZIP Code & Post Office Directory to assign most of the remaining observations to a corresponding 1990 census zip code. The final match rate is 99.7%. The unmatched zip codes are likely typos on birth and death certificates.

I then calculate two sets of distance variables for each zip code in ArcGIS, using geo-coded census information on zip code boundaries (polygons) and census block populations (points). First, I use the population-weighted average centroid of each zip code to calculate the distance between these centroids and the closest highway segments with a speed limit change. Second, I use the within zip

code spatial information on census block populations to calculate the percentage of each zip code's population living within 3, 5 and 10 miles of the closest speed limit change.

Table 20 contains summary statistics for these variables and various definitions of treatment and control groups. The table contains additional variables to those listed above for three reasons. First, I match all birth and death records with zip code level demographic information from the 1990 census. Second, I show the means of variables in the California birth cohort files that are only available for 1989 and 1990. These variables cannot be used as control variables, since they are not available prior to the speed limit changes. However, they provide further evidence that treatment and control groups are similar on observables. Third, I collect data on respiratory and cardiovascular hospitalization rates by zip code from the California Office of Statewide Health Planning and Development's patient discharge data.

Table 21 tests for differential trends in control variables. The table reports the coefficients on the interaction term in equation (9) when the outcome variable is replaced by those control variables that are available from the California birth cohort files both before and after the speed limit changes. Figure 13 shows that there are no differential pre-existing trends in any of the health outcome variables.

Enforcement

I filed FOIA requests to obtain information on speeding tickets and highway trooper employment from the highway patrols in the three states, specifically asking if enforcement was deliberately adjusted in response to the speed limit changes. The California Highway Patrol and Oregon and Washington State Police confirmed that there were no orders to increase enforcement following the speed limit changes either through more citations or through increased trooper employment (CHP, OSP, WSP, personal communication, May-June 2011). Figure 14 plots the enforcement variables, which are unfortunately not available by highway type.

Changes in the statewide amount of speeding tickets following the speed limit changes in 1987 and 1996 are within the range of historical variability. There are trends in statewide trooper employment, especially in California. These fluctuations reflect state budget decisions to approve future academy classes and retirement. The pronounced dip in trooper employment in California is the result of the suspension of cadet classes in the police academy in West Sacramento between 7 August 1992 and 7 February 1994 and a hiring freeze during the same period (CHP, personal communication, 24 May 2011). In Oregon, trooper employment also falls after 1990 due to budget cuts (OSP, personal communication, 13 June 2011). Budget cuts in Washington did not happen until 1992. The temporary spike in trooper employment in August 1996 is due to two graduating academy classes that were out of sync with planned retirement. WSP attempts to run an academy class every nine months, if approved, to replace troopers retiring from the agency (WSP, personal communication, 23 May 2011).

Table 20: Summary Statistics for Infant Health Data, for Various Zip Code Distances (California)

	Up to 3 miles (1)	3-5 miles (2)	3-10 miles (3)	Up to 5 miles (4)	5-10 miles (5)	10-20 miles (6)
			Health outcomes			
Fetal deaths (per 1000)	6.6	6.7	6.8	6.7	6.9	6.6
Infant deaths (per 1000)	8.9	8.7	9.3	8.8	9.5	9.0
Birth weight (grams)	3,393	3,371	3,373	3,383	3,374	3,383
Low birth weight (per 1000)	56.6	60.0	60.7	58.0	60.9	60.0
Gestational age (days)	278.6	278.2	278.2	278.4	278.2	278.2
Premature birth (per 1000)	6.6	6.4	6.5	6.5	6.6	6.5
			Individual level variables			
Child male	51.2%	51.0%	51.1%	51.1%	51.1%	51.2%
Child Hispanic	33.3%	25.1%	28.4%	29.7%	29.7%	28.7%
Child black	4.9%	8.9%	9.2%	6.6%	9.3%	8.9%
Child Asian	3.6%	11.9%	7.9%	7.2%	6.3%	6.6%
Different race parents	10.2%	18.1%	14.5%	13.6%	13.1%	12.9%
Education mother (years)	11.7	11.7	11.8	11.7	11.8	12.1
Mother high school dropout	30.0%	28.3%	29.3%	29.3%	29.7%	27.3%
Mother high school graduate	39.0%	37.6%	37.1%	38.4%	36.9%	35.1%
Mother attended college	30.9%	34.2%	33.6%	32.4%	33.4%	37.6%
Age mother (years)	25.7	26.2	26.0	25.9	25.9	26.3
Age 18 or below	8.3%	7.4%	7.9%	7.9%	8.1%	7.1%
Age 19-25	43.1%	40.6%	41.0%	42.0%	41.2%	39.4%
Age 26-30	29.3%	30.0%	29.9%	29.6%	29.9%	30.7%
Age 31-35	14.5%	16.0%	15.7%	15.2%	15.6%	17.2%
Age 36 or above	4.8%	5.9%	5.5%	5.3%	5.3%	5.7%
Number of children born alive	2.2	2.2	2.2	2.2	2.2	2.1
Age father (years)	28.5	28.9	28.7	28.7	28.7	29.0
Multiples	2.1%	2.1%	2.1%	2.1%	2.1%	2.2%
Ever had termination	18.9%	17.4%	17.3%	18.2%	17.2%	17.9%
Pregnancy complications	25.9%	26.5%	25.0%	26.2%	24.5%	25.3%
Delivery complications	35.2%	34.3%	32.2%	34.8%	31.4%	32.5%
Congenital anomaly	1.4%	1.7%	1.5%	1.6%	1.4%	1.4%
Birth injury	0.2%	0.2%	0.3%	0.2%	0.3%	0.2%
Cesarean section	49.9%	49.4%	48.5%	49.7%	48.2%	48.7%
Month prenatal care begun	2.9	2.8	2.8	2.9	2.8	2.7
First trimester prenatal care	72.4%	74.2%	73.8%	73.2%	73.7%	76.3%
Number of prenatal care visits	10.2	10.3	10.3	10.2	10.3	10.5
Government insurance	41.2%	39.3%	40.8%	40.4%	41.3%	38.0%
			Zip code level variables			
Respiratory hosp. (per 1000)	9.6	7.5	8.4	8.6	8.8	8.4
Cardiovascular hosp. (per 1000)	13.5	12.4	12.8	13.0	13.0	12.1
Median family income ($1990)	35,193	36,803	35,468	35,893	34,969	38,590
Urban	79.3%	89.8%	89.6%	83.9%	89.5%	94.9%
Married (age 25-34)	84.1%	80.9%	79.4%	82.7%	78.8%	77.9%
Total children (age 25-34)	1.7	1.6	1.6	1.6	1.6	1.5
Unemployment	8.0%	7.1%	8.3%	7.6%	8.7%	7.4%
Poverty	13.2%	12.7%	14.5%	13.0%	15.2%	13.3%
Owner occupied housing	62.8%	60.2%	59.3%	61.7%	59.0%	57.3%
			Distance from highway variables			
Centroid distance	1.5	4.0	6.4	2.6	7.4	14.8
Population within 3 miles	81.4%	23.9%	7.2%	56.4%	0.9%	0.1%
Population within 5 miles	98.0%	76.8%	26.6%	88.8%	7.8%	0.2%
Population within 10 miles	99.6%	99.8%	94.1%	99.6%	92.0%	5.5%

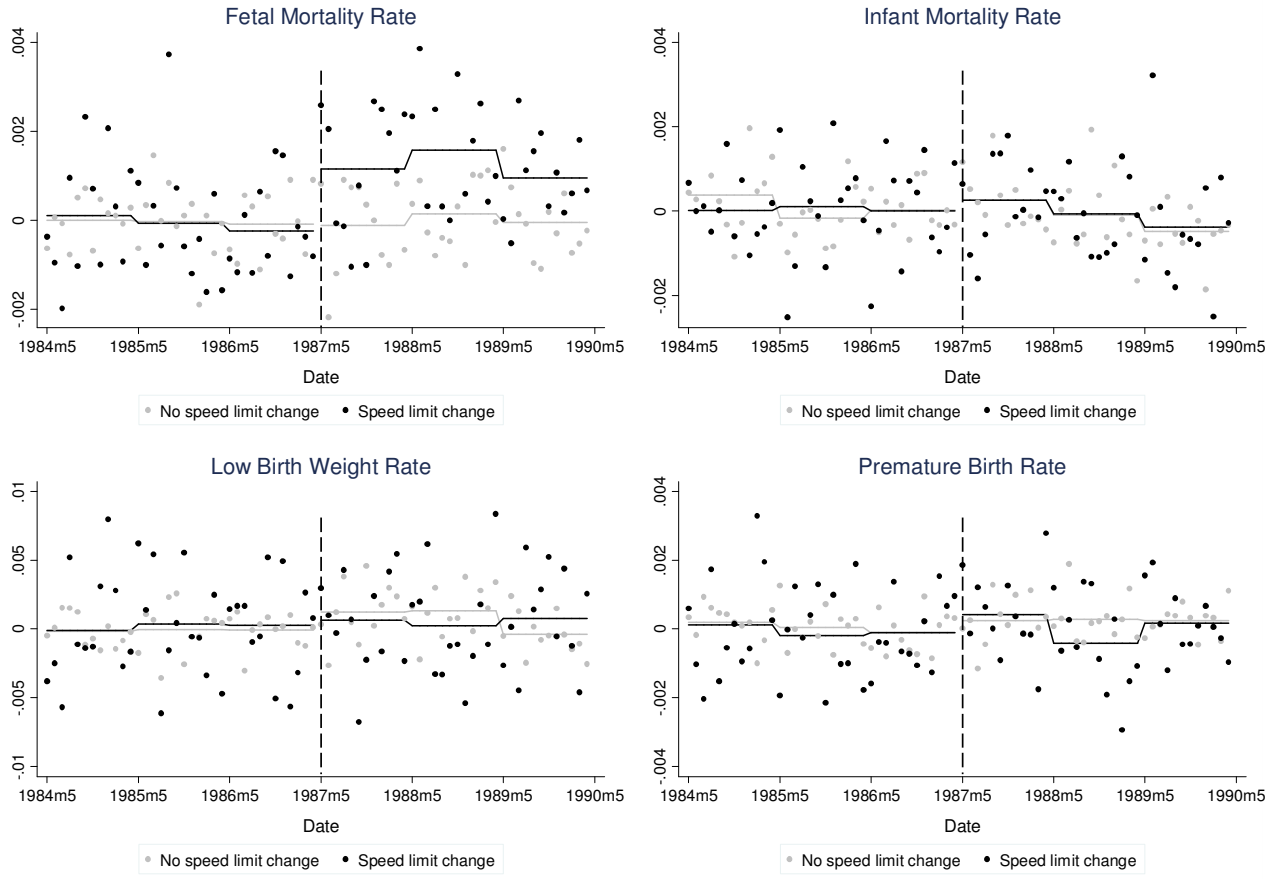
Notes: Means for the health related outcome variables, control variables and other descriptive variables in California. Means of variables from the birth cohort files (health outcomes and individual level variables) and patient discharge data (hospitalizations) are calculated for the period 1984-1986 (before the speed limit changes), or for 1989-1990 (if only available during that period). Other variables are births-weighted averages of the 1990 census variables at the zip code level (summary tape file 3b).

Table 21: Test for Differential Trends in Control Variables Before and After the 1987 Speed Limit Changes, for Various Buffer Distances (California)

	$(x, y) = (3, 3)$	$(x, y) = (3, 5)$	$(x, y) = (5, 5)$	$(x, y) = (5, 10)$
	(1)	(2)	(3)	(4)
Child male	-0.38%	-0.38%	-0.30%	-0.24%
	(0.34%)	(0.34)%	(0.26%)	(0.27%)
Child Hispanic	-2.21%***	-2.18%***	-1.03%*	-1.23%**
	(0.59%)	(0.59%)	(0.56%)	(0.59%)
Child black	-0.25%	-0.20%	0.16%	0.20%
	(0.35%)	(0.35%)	(0.35%)	(0.37%)
Child Asian	0.25%	0.14%	-0.48%	-0.42%
	(0.26%)	(0.25%)	(0.46%)	(0.47%)
Different race parents	-0.35%	-0.28%	0.17%	0.09%
	(0.31%)	(0.30%)	(0.30%)	(0.30%)
Age mother (years)	0.022	0.019	-0.041	0.002
	(0.039)	(0.039)	(0.031)	(0.032)
Age father (years)	-0.015	-0.014	-0.012	-0.021
	(0.030)	(0.030)	(0.026)	(0.028)
Number of children born alive	0.007	0.007	-0.001	-0.003
	0.015	(0.015)	(0.012)	(0.013)
Multiples	-0.01%	-0.01%	-0.03%	-0.04%
	(0.15%)	(0.15%)	(0.13%)	(0.13%)
Ever had termination	-0.52%	-0.48%	-0.03%	0.04%
	(0.42%)	(0.43%)	(0.36%)	(0.37%)
Number of pregnancy complications	-0.026	-0.020	0.015	0.018
	(0.029)	(0.029)	(0.031)	(0.033)
Number of delivery complications	0.040	0.036	-0.003	-0.018
	(0.033)	(0.033)	(0.028)	(0.028)
Number of congenital anomalies	0.006	0.004	-0.004	-0.001
	(0.008)	(0.008)	(0.009)	(0.009)
Cesarean section	0.14%	0.17%	0.40%	0.67%
	(0.93%)	(0.93%)	(0.77%)	(0.80%)
First trimester prenatal care	-1.97%*	-1.83%	-0.36%	-0.53%
	(1.15%)	(1.16%)	(0.85%)	(0.87%)
Month prenatal care begun	0.047	0.039	-0.019	-0.011
	(0.045)	(0.045)	(0.034)	(0.035)

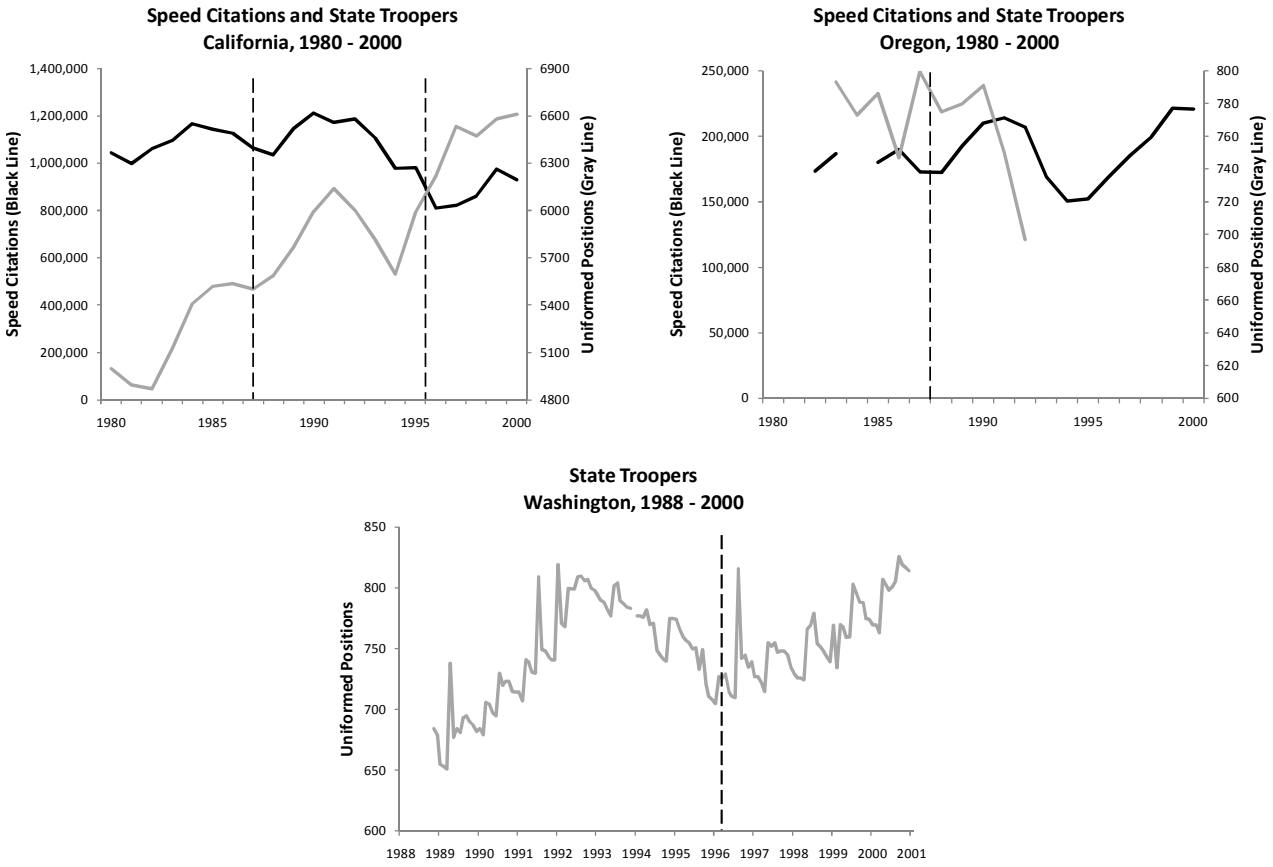
Notes: Coefficients on the interaction term in equation (9) when the outcome variable is replaced by those control variables that are available from the California birth cohort files both before and after the speed limit changes. Observations with a distance to the treatment highways exceeding 20 miles are dropped. Standard errors clustered at the zip code level in parentheses. Zip code, month-of-year and year fixed effects are included. Controls are included. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Figure 13: Trends in Health Outcome Variables Before and After the Speed Limit Increases in California (1987)



Notes: Gray dots indicate the monthly average health outcome for control zip codes, black dots for treatment zip codes. Treatment zip codes have a population-weighted average centroid less than 5 miles from a highway with a speed limit change. Zip codes with centroids more than 20 miles away are excluded. The various rates are relative to the pre-treatment average rate. All data are de-seasonalized at the monthly level. Solid lines are predicted values from regressions of health outcomes on year dummies. Vertical dotted lines indicate the time of the speed limit change in California (1 May 1987).

Figure 14: Statewide Trends in Speed Limit Enforcement: Speeding Tickets and Trooper Counts



Notes: The graphs plot speed citations (black lines) and state police trooper employment (gray lines). Annual data for California and Oregon; monthly data for Washington. Vertical dotted lines indicate speed limit changes.

Appendix B: Additional Robustness Checks

Robustness Checks for Speed Results

Table 22 shows the effect of the speed limit changes on traffic substitution towards highways with higher speed limits. The table reports estimates from specification (3) with the hourly number of vehicles as the dependent variable.

Robustness Checks for Accidents Results

Table 23 shows the accident rate specification using the Poisson instead of the negative binomial model. The LR test (footnote 18), calculated using the negative binomial regressions, rejects that there is no overdispersion for all but fatal accidents. Figure 15 confirms that the fit from the Poisson and negative binomial models is similar for fatal accidents, but the negative binomial model is a better approximation of the observed proportion of other types of accidents. The estimates from the two models are very similar.

Table 22: The Effect of Speed Limit Changes on Traffic Substitution Towards Treatment Highways

	California (1996)	Oregon (1987)	Washington (1996)
	(1)	(2)	(3)
Interaction (β_1)	0.0549	0.0002	0.0020
	(0.0342)	(0.1186)	(0.0870)
Observations	356,661	27,931	1,371,156
Fixed effects: station, direction, lane, year, month, day-of-week, plus:			
Station-direction	Y	N	Y
Hour-of-day	Y	Y	Y
Controls	All	All	All

Notes: The coefficient on the interaction term of 3 separate regressions is reported. The dependent variable is the hourly number of vehicles. Standard errors clustered at the station level in parentheses. The time window is 1994-1998 (CA), 1983-1992 (OR) and 1994-2001 (WA). ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Table 24 presents specifications in which the treatment effect can vary by season. The first two panels restrict the sample to summer and winter observations only. The final three panels combine heterogeneous season effects with more flexible control coefficients.

The treatment effect in winters is higher than in summers.[45] Allowing for differential control coefficients (panels 4 and 5) gives results similar to the restricted sample (panels 1 and 2). The joint estimation is more efficient. The seasonal difference in the treatment effect is only statistically significant in a few cases. Allowing for heterogeneous season effects without differential control coefficients (panel 3) introduces a larger gap between the summer and winter treatment effects. This estimation is likely too restrictive: for the estimation of heterogeneous treatment effects, equal control coefficients by highway type does seem to introduce a bias in the estimation.

Table 25 contains further robustness checks. First, speed may only be a secondary factor in alcohol and drugs related accidents. The first panel excludes these accidents. Second, the increase in accident rates may be attributed mostly to accidents involving heavy or light-duty trucks. About 10% of total accidents involves heavy trucks. The percentage of accidents that involves light-duty trucks is growing over time (from 32.3% to 44.6% over the study period). Since I do not observe VMT by vehicle and highway type, the treatment effect would be biased if rural interstates experienced a change in (light-duty) trucks relative to the control highways and if trucks have a different likelihood to be involved in accidents. I do observe the vehicle types involved in collisions in Washington. Panels 2 and 3 exclude heavy truck and light-duty truck related accidents, respectively. Third, analogous to the time discontinuity equation (4) for travel speed, I estimate a count data version of this equation with polynomials up to the tenth order. Panel 4 presents the average treatment coefficients and standard errors using time polynomials of order 0, 1, ..., 10.

Panel 1 shows that removing alcohol and drugs related accidents from the sample (instead of adding alcohol and drugs as control variables) hardly changes the coefficients compared to Table 7. Thus, the treatment effect is not driven by alcohol and drugs related accidents. Removing heavy

[45] It is possible to allow for more heterogeneity in treatment effects, for instance by adding weekend/weekday and daylight/dark dimensions. In most specifications, the treatment effects are largest in the winter, weekends and in the dark, but they are imprecisely estimated.

Table 23: Impact of 1987 Speed Limit Changes on Accident Rates: Poisson Model

	Fatal	Incapacitating	Non-incapacitating	Damage only	Total
	(1)	(2)	(3)	(4)	(5)
			Oregon & Washington combined		
Interaction (β_1)	0.365***	0.213***	0.143***	0.134***	0.142***
	(0.113)	(0.048)	(0.032)	(0.016)	(0.019)
Exact relative change	0.441	0.238	0.154	0.143	0.152
LR test p-value	0.500	0.000	0.000	0.000	0.000
			Oregon		
Interaction (β_1)	0.469***	0.169**	0.056	0.098	0.098*
	(0.163)	(0.084)	(0.069)	(0.066)	(0.058)
Exact relative change	0.598	0.185	0.057	0.103	0.103
LR test p-value	0.500	0.000	0.000	0.000	0.000
			Washington		
Interaction (β_1)	0.292***	0.215***	0.153***	0.114***	0.128***
	(0.092)	(0.056)	(0.056)	(0.036)	(0.037)
Exact relative change	0.339	0.240	0.165	0.121	0.137
LR test p-value	0.499	0.001	0.000	0.000	0.000
Observations	6,573	6,573	6,573	6,573	6,573

Notes: The coefficient on the interaction term of 15 separate regressions is reported. The dependent variable is the number of accidents per VMT per day. Highway type, year, month-of-year and day-of-week fixed effects are included. Controls are included. The exact relative change is calculated as $exp(\beta_1) - 1$. Standard errors clustered at the highway type by year level in parentheses. Observations are taken within a six year symmetric time window around the dates of the speed limit changes. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

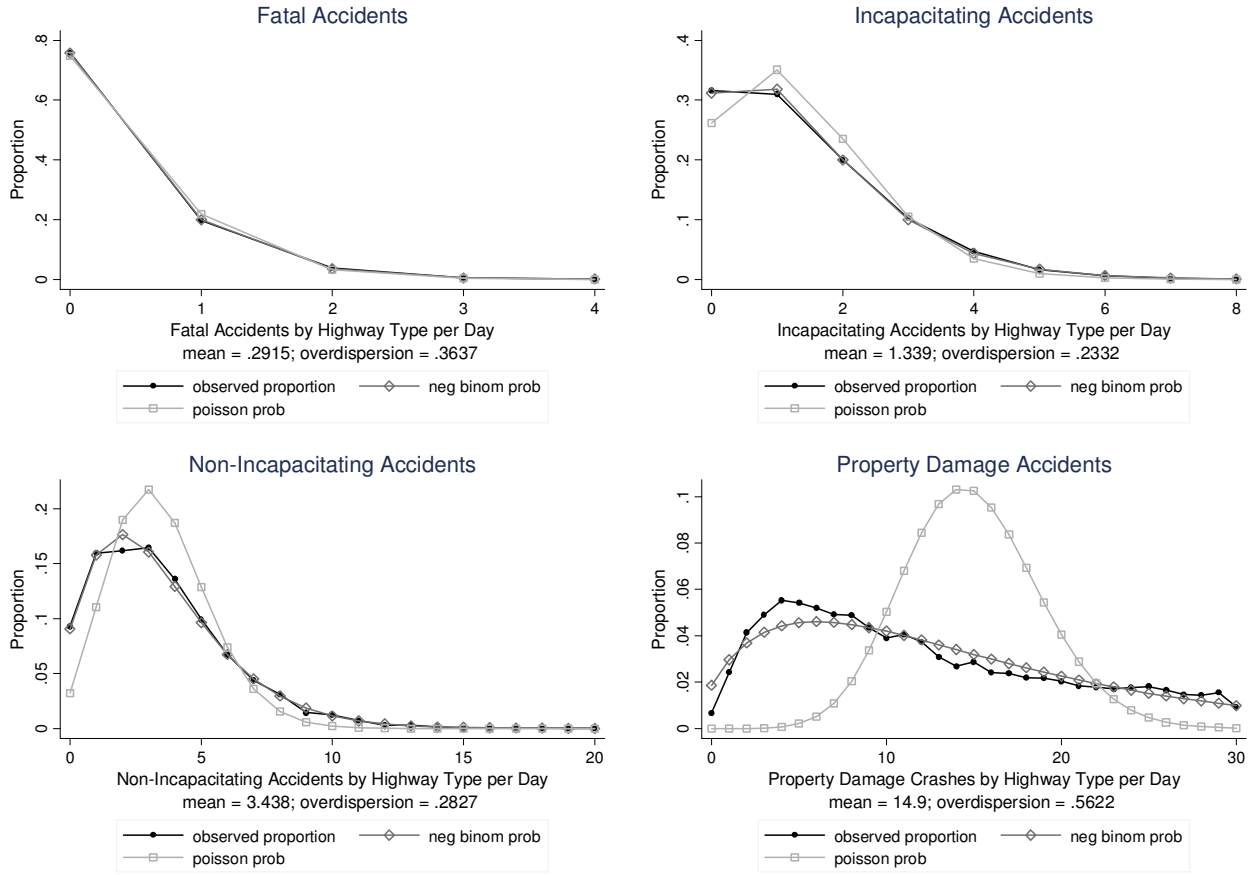
truck related accidents (panel 2) and light-duty truck related accidents (panel 3) gives similar but somewhat higher estimates of the treatment effects. This alleviates the concern that unobserved differential trends in truck miles traveled by highway type drive the results.

The average treatment effects from the time discontinuity regressions in panel 4 are somewhat different from the main specification in Table 7. The coefficient for non-incapacitating accidents is about twice as large. The time discontinuity estimates are noisy, and are sensitive to the order of the time polynomial. A time discontinuity specification is not preferred for two reasons. First, the discontinuity is not sharp since the official dates of the speed limit changes do not coincide with the (unknown) dates at which the new road signs were erected. Second, since fatal and incapacitating accidents are relatively low-frequency events, the number of observations close to the treatment date is limited. Therefore, longer term trends play an important role. This underscores the importance of differencing out such trends in accidents on control highways.

Finally, Figure 16 illustrates the placebo test results discussed in Section 7.2.3. The opportunity for conducting placebo tests is limited because the time window should not overlap or be too close to the date of the real treatment, and to the speed limit changes in 1996 (both on rural interstates and on other highways). In addition, since the 75 regressions are not performed on independent samples, this placebo test should not be interpreted as more than just suggestive supporting evidence.[46]

[46]This paper does not naturally lend itself for a series of placebo tests in the spirit of Conley and Taber (2011). They argue that many difference-in-differences models rely on a small number of groups that experience a policy change (in this case: rural interstates), while the usual asymptotic inference assumes that the number of groups

Figure 15: Fitting the Poisson and Negative Binomial Models (Oregon & Washington Combined)



Notes: The lines represent the observed proportions of accidents by highway type (black), and the fitted proportions for the Poisson model (light gray) and the negative binomial model (dark gray).

Robustness Checks for Infant Health Results

Table 26 presents the results from the infant health regressions using an alternative zip code distance classification based on the percentage of residents living within a particular distance from the highway (as opposed to the population-weighted average zip code centroid). The details are described in Appendix A. The results are similar to those in Table 14 in Section 9.

changing policy is large. They present an alternative inference approach for such cases, using information from a large sample of non-changing groups. Their alternative method consists of running a series of placebo tests in which a hypothetical "treatment" is assigned to groups that did not receive treatment in reality. With only two non-changing groups (rural principal arterials and urban highways), this placebo test is not suitable here.

Table 24: Alternative Specifications: Heterogeneous Season Effects

	Fatal	Incapacitating	Non-incapacitating	Damage only	Total
	(1)	(2)	(3)	(4)	(5)
			Summers only		
Interaction (β_1)	0.274*	0.197***	0.139***	0.092***	0.108***
	(0.152)	(0.049)	(0.034)	(0.025)	(0.018)
Exact relative change	0.315	0.218	0.150	0.097	0.114
			Winters only		
Interaction (β_1)	0.618***	0.248***	0.162**	0.175***	0.173***
	(0.128)	(0.072)	(0.068)	(0.032)	(0.034)
Exact relative change	0.855	0.282	0.175	0.191	0.189
			Heterogeneous season effects		
Interaction (β_1)	0.368***	0.187***	0.095***	0.013	0.046*
	(0.127)	(0.056)	(0.027)	(0.030)	(0.025)
Interaction (β_1) $*$ winter	0.021	0.077	0.118**	0.271***	0.222***
	(0.142)	(0.077)	(0.056)	(0.067)	(0.061)
Exact relative change (S)	0.444	0.205	0.099	0.013	0.047
Exact relative change (W)	0.474	0.302	0.237	0.329	0.307
			Highway type specific weather coefficients - heterogeneous season effects		
Interaction (β_1)	0.257**	0.154***	0.112***	0.079***	0.092***
	(0.132)	(0.045)	(0.032)	(0.022)	(0.019)
Interaction (β_1) $*$ winter	0.234	0.126	0.059	0.123**	0.110*
	(0.184)	(0.077)	(0.073)	(0.062)	(0.062)
Exact relative change (S)	0.294	0.167	0.118	0.082	0.096
Exact relative change (W)	0.635	0.323	0.187	0.223	0.223
			Highway type specific control coefficients - heterogeneous season effects		
Interaction (β_1)	0.290**	0.165***	0.145***	0.117***	0.128***
	(0.124)	(0.045)	(0.022)	(0.034)	(0.025)
Interaction (β_1) $*$ winter	0.179	0.148*	0.047	0.105	0.097
	(0.197)	(0.082)	(0.073)	(0.066)	(0.064)
Exact relative change (S)	0.337	0.180	0.156	0.124	0.136
Exact relative change (W)	0.599	0.368	0.212	0.249	0.252

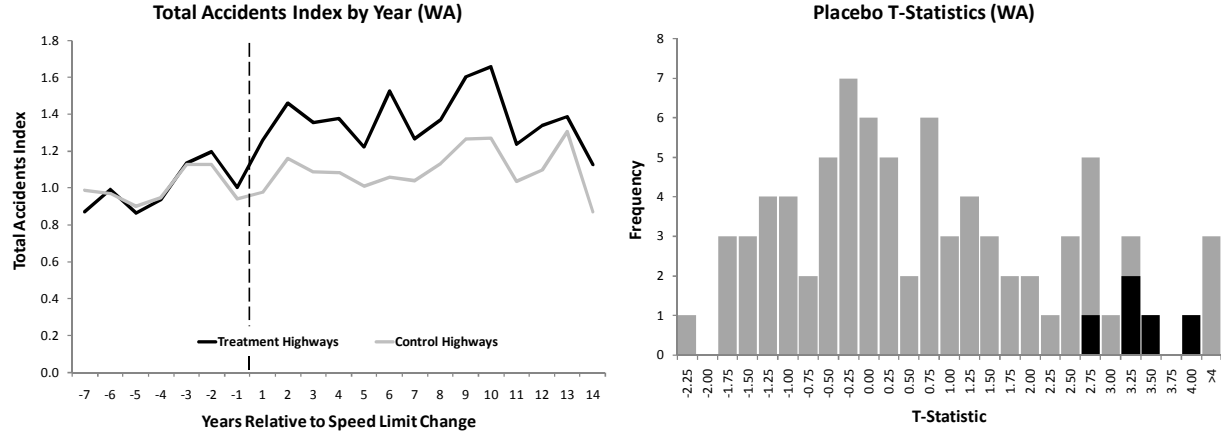
Notes: The coefficient on the interaction term of 25 separate negative binomial regressions using the combined Oregon and Washington sample is reported. The dependent variable is the number of accidents per VMT per day. Highway type, year, month-of-year and day-of-week fixed effects are included. Controls are included. The exact relative change is calculated as $exp(\beta_1) - 1$. Standard errors clustered at the highway type by year level in parentheses. Observations are taken within a six year symmetric time window around the dates of the speed limit changes (4,410 for summers only, 2,163 for winters only, 6,573 for all other regressions). ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Table 25: Alternative Specifications: Other Robustness Checks

	Fatal (1)	Incapacitating (2)	Non-incapacitating (3)	Damage only (4)	Total (5)
			Excluding alcohol and drugs related accidents		
Interaction (β_1)	0.355**	0.218***	0.139***	0.133***	0.135***
	(0.173)	(0.043)	(0.027)	(0.018)	(0.018)
Exact relative change	0.427	0.244	0.149	0.143	0.145
			Washington - excluding heavy truck related accidents		
Interaction (β_1)	0.343***	0.281***	0.223***	0.135***	0.158***
	(0.134)	(0.092)	(0.067)	(0.037)	(0.040)
Exact relative change	0.410	0.325	0.250	0.145	0.171
			Washington - excluding light-duty truck related accidents		
Interaction (β_1)	0.406***	0.296***	0.165***	0.135***	0.153***
	(0.095)	(0.078)	(0.062)	(0.043)	(0.043)
Exact relative change	0.501	0.344	0.179	0.145	0.165
			Time discontinuity specification		
Interaction (β_1)	0.269	0.226	0.280***	0.143***	0.180***
	(0.190)	(0.168)	(0.090)	(0.046)	(0.046)
Exact relative change	0.309	0.253	0.323	0.154	0.197

Notes: The coefficient on the interaction term of 15 separate negative binomial regressions is reported in panels 1-3. In panel 4, each reported coefficient is the average over 11 coefficients using time polynomials of order 0, 1, . . . , 10. The dependent variable is the number of accidents per VMT per day. Highway type, year, month-of-year and day-of-week fixed effects are included. Controls are included. The exact relative change is calculated as $exp(\beta_1) - 1$. Standard errors clustered at the highway type by year level in parentheses. The number of observations is 6,573. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Figure 16: Placebo Test for Accident Regressions (Washington)



Notes: The left panel plots the total accidents index for treatment and control roads in Washington, from 7 years before to 14 years after the speed limit change in 1987. The vertical dotted line indicates the time of the speed limit change (23 April 1987). Data from Washington are used because of the availability of a long window of pre- and post-treatment data. The graph demonstrates the absence of differential pre-existing trends. The right panel shows the t-statistics of the five treatment effect coefficients of the main specification in Table 7 (in black; one for each accident type), along with the t-statistics of 75 placebo regressions. In these regressions, a hypothetical speed limit change was introduced on rural interstates 6, 7, 8, 9 and 10 years after the real speed limit change. The symmetric window around the hypothetical speed limit changes varies from 6 to 8 to 10 years. The three high t-statistics in the placebo regressions correspond to a spike in non-incapacitating accidents on rural interstates in year 6.

Table 26: Regression Results for the Effect of the 1987 Speed Limit Changes on Infant Health, for Various Alternative Buffer Definitions (California)

	T: 90+% $\leq$ 3 miles C: 10-% $\leq$ 3 miles	T: 75+% $\leq$ 3 miles C: 10-% $\leq$ 3 miles	T: 90+% $\leq$ 5 miles C: 25-% $\leq$ 5 miles	T: 90+% $\leq$ 5 miles C: 50-% $\leq$ 5 miles	T: 75+% $\leq$ 3 miles C: 25-% $\leq$ 5 miles	T: 75+% $\leq$ 3 miles C: 50-% $\leq$ 5 miles
	(1)	(2)	(3)	(4)	(5)	(6)
Fetal death	0.00073	0.00075	0.00109**	0.00109**	0.00134***	0.00132***
(Gestational age $\geq$ 98)	(0.00059)	(0.00058)	(0.00052)	(0.00050)	(0.00050)	(0.00048)
Observations	276,416	301,630	337,246	362,638	354,229	379,621
Fetal death	0.00032	0.00039	0.00082*	0.00083*	0.00107**	0.00107***
(Gestational age $\geq$ 196)	(0.00049)	(0.00043)	(0.00044)	(0.00043)	(0.00043)	(0.00041)
Observations	274,121	299,143	334,485	359,684	351,322	376,521
Infant death	0.00004	-0.00032	-0.00037	-0.00046	-0.00032	-0.00041
(Gestational age $\geq$ 196)	(0.00088)	(0.00078)	(0.00064)	(0.00063)	(0.00064)	(0.00062)
Observations	272,838	297,760	332,923	358,013	349,691	374,781
Infant death	0.00027	0.00004	-0.00002	-0.00005	0.00016	0.00013
(Gestational age $\geq$ 259)	(0.00081)	(0.00069)	(0.00054)	(0.00054)	(0.00054)	(0.00054)
Observations	248,549	271,278	303,386	326,182	318,739	341,535
Low birth weight	-0.0066**	-0.0042*	-0.0019	-0.0026	-0.0017	-0.0024
(Gestational age $\geq$ 196)	(0.0030)	(0.0025)	(0.0022)	(0.0021)	(0.0021)	(0.0020)
Observations	272,838	297,760	332,923	358,013	349,691	374,781
Low birth weight	-0.0012	0.0012	0.0017	0.0013	0.0021	0.0017
(Gestational age $\geq$ 259)	(0.0020)	(0.0017)	(0.0013)	(0.0013)	(0.0013)	(0.0012)
Observations	248,549	271,278	303,386	326,182	318,739	341,535
Premature birth	-0.00011	-0.00034	-0.00039	-0.00046	-0.00044	-0.00052
(Gestational age $<$ 196	(0.00087)	(0.00071)	(0.00067)	(0.00064)	(0.00065)	(0.00063)
Observations	273,956	298,977	334,283	359,456	351,129	376,302

Notes: The coefficient on the interaction term of 42 separate regressions is reported. Treatment (T) and control (C) groups are defined in terms of the fraction of the population in a given zip code that lives within 3 or 5 miles from the treatment highways. In the column headings, x+% and y−% refer to "at least x%" and "at most y%". Standard errors clustered at the zip code level in parentheses. Zip code, month-of-year and year fixed effects are included. Controls are included. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Appendix C: Quantification of Health Effects

Table 27 classifies various costs and benefits by their inclusion in the analysis of this paper.

Table 27: Quantified and Unquantified Effects of Speed Limit Changes

Quantified effects	Unquantified effects
Time savings from traveling at higher speed	Pleasure of driving at higher speed
Fatal accidents	Non-respiratory related hospitalizations
Incapacitating accidents	Non-asthma related emergency room visits
Non-incapacitating accidents	Asthma exacerbations
Property damage only accidents	New cases of chronic asthma
Infant health/mortality	Increased airway responsiveness to stimuli
Fetal health/mortality	Chronic respiratory damage
Premature mortality for adults	Premature aging of the lungs
Respiratory related hospitalizations	Inflammation of the lungs
Asthma related emergency room visits	Increased susceptibility to respiratory infection
Outdoor worker productivity	Acute inflammation and respiratory cell damage
Increased gasoline consumption	Pulmonary function
Climate impacts	Minor restricted activity days
	School loss days
	UV-b exposure
	Impacts on agricultural yields
	Impacts on commercial forests
	Impacts on (commercial and recreational) freshwater fishing
	Watershed damages (e.g., water filtration, flood control)
	Impacts to recreation in estuarine ecosystems
	Reduced existence/option value for non-eutrophied ecosystems
	Coastal eutrophication from nitrogen deposition effects

Notes: Source: EPA (2002; 2011c).

I follow the EPA's approach to use concentration-response (C-R) functions to quantify premature mortality, respiratory hospitalizations, asthma related emergency room visits, and productivity of outdoor workers (EPA, 2010; 2011c). The EPA has collected a database of academic papers that estimate the relationship between a change in the concentration of a particular air pollutant and a specific health outcome. Using the functional form estimated in the corresponding paper, if available, or otherwise by assumption, the EPA assigns a functional form for the C-R function. In most cases, the C-R function is log-linear.[47]

Below I summarize the C-R functions employed in Section 10. Oftentimes, more than one paper is available to derive a particular C-R function. This leads to the construction of a low, central and high health impact scenario. For each health outcome, I select the papers that give rise to the lowest and highest health impact. If a third, central case, is available, I also select this C-R function. Otherwise, I averaged the health impacts from the low and the high C-R functions.

Log-linear C-R functions have the following shape:

$$\Delta health = health_0 * \left(1 - \frac{1}{exp(\beta \Delta p)}\right) \tag{10}$$

[47] These C-R functions are all incorporated into EPA's program to calculate health benefits, BenMAP. It is available to the public at http://www.epa.gov/air/benmap/index.html.

where *health* is the health outcome, p is the pollutant concentration and $health_0$ is the baseline incidence rate of the health outcome (before the change in pollution). Table 28 summarizes the values for β in the three health impact scenarios.

Table 28: Parameters of the Concentration-Response Functions for the Health Impact Scenarios

Scenario	Health outcome	Pollutant	Value of β	Source
Low	Premature mortality	O_3	0.000520	Bell *et al.* (2004)
Central	Premature mortality	O_3	0.000936	Samet *et al.* (1997)
High	Premature mortality	O_3	0.001750	Ito *et al.* (2005)
Low	Respiratory hospitalizations	O_3	0.002652	Schwartz (1995)
High	Respiratory hospitalizations	O_3	0.007147	Schwartz (1995)
Low	Respiratory hospitalizations	NO_2	0.003285	Fung *et al.* (2006)
High	Respiratory hospitalizations	NO_2	0.008759	Yang *et al.* (2003)
Low	Asthma emergency room visits	O_3	-0.001000	Wilson *et al.* (2005)
High	Asthma emergency room visits	O_3	0.003000	Wilson *et al.* (2005)
Low	Asthma emergency room visits	NO_2	0.002264	NYDOH (2006)
High	Asthma emergency room visits	NO_2	0.005460	Ito *et al.* (2007)
Central	Outdoor worker productivity	O_3	-0.142700*	Crocker and Horst (1981)

Notes: Source: EPA (2010; 2011c). *Crocker and Horst estimated the elasticity of outdoor worker income with respect to the ozone concentration.

The health impact scenarios also differ in their treatment of fetal deaths. The low and central health impact scenarios value fetal deaths at $0. The high health impact scenario values third trimester fetal deaths at the VSL, but second trimester fetal deaths at $0.