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LETTER

Impact of the Volkswagen emissions control defeat device on US public health

Steven R H Barrett[1], **Raymond L Speth**[1], **Sebastian D Eastham**[2], **Irene C Dedoussi**[1], **Akshay Ashok**[1], **Robert Malina**[1] **and David W Keith**[2]

[1] Massachusetts Institute of Technology, Cambridge MA 02139, USA
[2] Harvard University, Cambridge MA 02138, USA

E-mail: sbarrett@mit.edu

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Abstract

The US Environmental Protection Agency (EPA) has alleged that Volkswagen Group of America (VW) violated the Clean Air Act (CAA) by developing and installing emissions control system 'defeat devices' (software) in model year 2009–2015 vehicles with 2.0 litre diesel engines. VW has admitted the inclusion of defeat devices. On-road emissions testing suggests that in-use NO_x emissions for these vehicles are a factor of 10 to 40 above the EPA standard. In this paper we quantify the human health impacts and associated costs of the excess emissions. We propagate uncertainties throughout the analysis. A distribution function for excess emissions is estimated based on available in-use NO_x emissions measurements. We then use vehicle sales data and the STEP vehicle fleet model to estimate vehicle distance traveled per year for the fleet. The excess NO_x emissions are allocated on a 50 km grid using an EPA estimate of the light duty diesel vehicle NO_x emissions distribution. We apply a GEOS-Chem adjoint-based rapid air pollution exposure model to produce estimates of particulate matter and ozone exposure due to the spatially resolved excess NO_x emissions. A set of concentration-response functions is applied to estimate mortality and morbidity outcomes. Integrated over the sales period (2008–2015) we estimate that the excess emissions will cause 59 (95% CI: 10 to 150) early deaths in the US. When monetizing premature mortality using EPA-recommended data, we find a social cost of ~$450m over the sales period. For the current fleet, we estimate that a return to compliance for all affected vehicles by the end of 2016 will avert ~130 early deaths and avoid ~$840m in social costs compared to a counterfactual case without recall.

1. Introduction

Outdoor air pollution adversely affects human health (US EPA 2011, WHO 2006). The leading causes of outdoor air pollution-related premature mortality and morbidity outcomes are exposure to fine particulate matter ($PM_{2.5}$) (Dockery *et al* 1993, Pope *et al* 2002, WHO 2006) and ozone (Bell *et al* 2004, Jerrett *et al* 2009, WHO 2008). The US Environmental Protection Agency (EPA) estimated that in 2010 there were ~160 000 premature deaths in the US due to $PM_{2.5}$ exposure and ~4300 deaths related to ozone exposure (US EPA 2011). In the case of road transportation in the US, Caiazzo *et al* (2013) estimated that the sector's emissions caused 52 800 early deaths in 2005 (90% confidence interval (CI): 23 600 to 95 300) due to increased $PM_{2.5}$ exposure and 5250 early deaths (90% CI: −850 to 11 100) due to increased ozone exposure. An estimate by Dedoussi and Barrett (2014) using identical emissions and concentration-risk functions, but a different atmospheric chemistry-transport modeling approach, is 42% higher for $PM_{2.5}$-related early deaths. Fann *et al* (2013) find 16% lower total deaths than Caiazzo *et al* (2013) due to exposure to inorganic $PM_{2.5}$ for approximately comparable emissions. This variability across results from different health impact assessment approaches is indicative of the scientific uncertainty in exposure estimates

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associated with modeling atmospheric processes, in addition to uncertainty in concentration-response functions that relate exposure to health outcomes, and uncertainty in the quantity of emissions as well as their spatial and temporal distribution and chemical profile. Broadly the central estimates of early deaths due to emissions applying different approaches vary by a factor of two.

The US Clean Air Act (CAA) regulates atmospheric emissions from stationary and mobile sources for the purpose of protecting human health and the environment. The CAA and its implementing regulations include the setting of standards for motor vehicle NO_x (oxides of nitrogen, i.e. NO + NO_2) and other emissions (42 U.S.C. §§ 7401–7671q). Over time, the EPA has set increasingly stringent emissions regulations, including for light duty vehicle NO_x emissions (US EPA 2012). The EPA administers a program to ensure that vehicles sold in the US comply with the relevant emissions standards and issues certificates of conformity for the introduction of new vehicles. Emissions standards are set on the basis of particular drive cycles, against which new models are tested to demonstrate compliance. Regulations specifically prohibit 'defeat devices', which are auxiliary emissions control devices that '[reduce] the effectiveness of the emission control system under conditions which may reasonably be expected to be encountered in normal vehicle operation and use' (40 C.F.R. §86.1803-01) with certain exceptions. Defeat devices are defined to include software in the electronic control module (ECM). Violations related to both defeat devices and excess emissions can result in civil penalties (CAA § 205(a), 42 U.S.C. §7524(a), 40 C.F.R. §19.4), such as the case of excess greenhouse gas emissions from Hyundai and Kia light-duty vehicles (US EPA 2014b) and faulty emission control devices on Mercedes-Benz vehicles (US EPA 2006).

Diesel vehicles are a small but growing part of the US light duty vehicle fleet, with Volkswagen Group of America (VW) being one of the leading manufacturers, accounting for approximately 20% of new light duty diesel vehicle sales, and 70% of new diesel passenger car sales in 2014 (VW of America 2015). In 2015 VW admitted the use of defeat devices in certain 2009–2015 model year 2.0 litre light duty diesel vehicles (US EPA 2015a). This followed work by the West Virginia University (WVU) Center for Alternative Fuels, Engines & Emissions commissioned by the International Council on Clean Transportation, in which significantly higher in-use emissions were found for a 2012 Jetta and a 2013 Passat relative to emissions test values (Thompson *et al* 2014a). After a voluntary recall in December 2014 failed to resolve the excess emissions, and when EPA and the California Air Resources Board (CARB) would not approve VW 2016 model year diesel vehicles, VW admitted it had installed a software-based defeat device which detected if the vehicle was undergoing emissions testing and modified operation of the emission control system.

On 18 September 2015 the EPA, in coordination with CARB, issued a notice of violation to VW in which EPA alleged violations of the CAA and its implementing regulations. Specifically, EPA alleged that VW developed and installed emissions control system defeat devices in model year 2009–2015 2.0 litre diesel light duty vehicles, which resulted in real world NO_x emissions that are a factor of 10 to 40 above EPA the compliant levels. Possible penalties under the CAA include up to $37 500 for each noncompliant vehicle and $3750 per violation related to defeat devices (US EPA 2015a, ARB 2015), with a possible total penalty of approximately $20bn based on 482 000 affected vehicles sold. Initial estimates in the press of the impact already incurred on public health range from 16 to 106 premature mortalities (NYT 2015, VOX 2015, AP 2015), but there is yet to be a peer-reviewed study which addresses this question.

In this paper we assess the public health impacts of the excess emissions from VW 2.0 litre light duty vehicles in the US for the period 2008–2015. (Model year 2009 vehicles were sold from 2008.) Both mortality and morbidity outcomes associated with exposure to $PM_{2.5}$ and ozone are estimated. We also estimate the benefits of a return to compliance with the Clean Air Act and its implementing regulations for the existing in-use fleet. We assume the return to compliance occurs at a constant rate throughout 2016.

Beyond the specific case of the VW excess emissions, the methods used here demonstrate the growing ability to make spatially resolved probabilistic estimates of the health impacts of small changes in emissions using modern modeling tools, particularly linearized adjoints of best-in-class chemistry-transport models. The ability to make such estimates in near-real time may enable new approaches to the efficient and effective management of air pollution.

2. Methods

We conduct the analysis in a probabilistic framework given the significant uncertainties in estimating early deaths and associated cost. Here we describe our approach to uncertainty modeling, emissions estimation, annual average $PM_{2.5}$ and one-hour daily maximum ozone exposure estimation, mortality and morbidity outcomes estimation, and finally valuation of mortality impacts.

We first compute health impacts and costs from emissions in excess of VW reported values (US EPA 2015b) from 2008–2015. We then compute excess emissions, health impacts and costs if all vehicles are brought into compliance with EPA regulation by December 2016, starting in January 2016, and assuming a vehicle recall at a constant rate throughout

the year. This is representative of the VW plan at the time of writing (WSJ 2015). The benefit of the recall in terms of avoided health impacts and costs is calculated by subtracting the recall results from the counterfactual results that would occur if the vehicles affected remain in the US vehicle fleet until their retirement without being brought into EPA compliance.

2.1. Uncertainty

Uncertainty in input variables such as the total underreported NO_x is propagated throughout the calculation using Monte Carlo simulation. Ten thousand independent draws of each variable are performed, resulting in ten thousand independent estimates of the total mortality and morbidity impacts. The same draws are used to estimate the impacts in each year and under each scenario. Where not explicitly stated otherwise, reported results correspond to the median value of the the output distribution, i.e. there is equal probability that our central estimate is low or high. When monetizing mortality impacts, the economic value of statistical life is treated as an additional uncertain parameter with ten draws (i.e. 100 000 draws in total). Other sources of uncertainty, such as the shapes of exposure-response functions, are explored in sensitivity analyses which are discussed in the supplementary material.

We base our study on data from 2005 to 2015, using the most recent available data and correcting to the study period (from 2008) where necessary and possible. For example, gridded population data is for 2006, which is scaled for each year using changes in total US population. This introduces uncertainty in the population totals (which are forecasts from 2011 onwards), and also changes in the spatial distribution of population over time (which are not accounted for). Data sources and uncertainty associated with the different parts of our analysis are detailed in the following subsections. We further discuss sources of unquantified uncertainty in section 4.2, and where possible note the direction of the bias that may be introduced.

2.2. Vehicle fleet and activity

Total activity for the affected vehicles, expressed as vehicle-kilometers traveled (VKT), is determined using annual sales data for the affected vehicles and the Stochastic Transport Emissions Policy (STEP) light duty vehicle fleet model (Bastani *et al* 2012). The model uses assumed values for the average annual growth rate in kilometers traveled per vehicle and the decay rate with vehicle age. A logistic function is used to determine the vehicle retirement rate. The model uses triangular distributions to account for uncertainty in growth and decay rates and the median vehicle age at retirement. Parameters for each distribution are given in the supplementary material. Annual sales figures for affected VW vehicles between 2008 and August 2015 are obtained from WardsAuto Group (2015). These sales figures are scaled to match the total number of affected vehicles, 482 000, reported by the EPA which also includes affected Audi vehicles (US EPA, 2015c).

2.3. Excess emissions

The measurements of Thompson *et al* (2014a) are used to calculate NO_x emissions factors for affected vehicles. Thompson *et al* (2014a) tested two affected vehicles, a 2012 Jetta (vehicle A) and a 2013 Passat (vehicle B), on several drive cycles characteristic of highway and urban driving. Each drive cycle was driven one or two times with each vehicle. We use the individual drive cycle NO_x emissions factors for all urban drive cycles using either vehicle (9 samples) and compute the mean and standard deviation of a truncated normal distribution for the urban driving emissions factor. We do the same with the highway drive cycle (4 samples) for the highway driving emissions factor. The distribution parameters for the baseline emissions factor are computed from the weighted FTP-75 chassis dynamometer tests for each vehicle (Thompson *et al* 2014a).

Excess emissions are allocated to 10 road types in the US consisting of rural and urban interstate, arterial, collector and local roads. Emissions based on highway test data are allocated to rural interstate roads, while those calculated from city drive cycles are allocated to the other road types. This is done since the urban drive cycles included portions of highway driving. We use spatial surrogates for light duty diesel vehicle NO_x emissions from the 2005 National Emissions Inventory (US EPA 2008) to map roads to model grid cells.

Excess emissions from 2008 through 2015 are computed as the difference between the real driving emissions and the FTP-75 emissions, representing the difference between actual and expected emissions. Excess emissions from 2016 onward are calculated as the difference between the estimated real driving emissions and the EPA Tier 2 Bin 5 standard of 0.043 g km^{-1} (40 C.F.R. §86.1811–17), representing the difference in NO_x emissions that could be expected if these vehicles were brought into regulatory compliance.

2.4. Air quality modeling

Exposure to annual $PM_{2.5}$ and one-hour daily maximum ozone is estimated using updated versions of air quality sensitivity matrices developed as an air pollution policy assessment tool by Dedoussi and Barrett (2014). Sensitivity matrices derived from the GEOS-Chem adjoint have previously been used to assess the health impacts of emissions perturbations by Koo *et al* (2013) and Lee *et al* (2015). The sensitivity matrices map an arbitrary three-dimensional time-varying distribution of emissions to total US population exposure to inorganic $PM_{2.5}$ (on an annual average

basis) and ozone (based on a one-hour daily maximum value during the ozone season). The sensitivity matrices were computed from the adjoint of the GEOS-Chem chemistry-transport model applied at a ~50 km resolution to a domain encompassing the contiguous United States (CONUS). Further discussion of the air quality modeling approach relative to alternatives is provided in the supplementary material.

The GEOS-Chem adjoint model was developed by Henze *et al* (2007) and is based on the GEOS-Chem global tropospheric chemistry-transport model (Bey *et al* 2001). It calculates the sensitivities of quantities of interest (e.g. population exposure to $PM_{2.5}$ and ozone) with respect to various model parameters (e.g. emissions). GEOS-Chem performs transport, gas- and aerosol-phase chemistry and wet and dry deposition calculations. In the GEOS-Chem version that is applied in the present work the KPP chemical solver (Damian *et al* 2002) and the RPMARES aerosol equilibrium model (an implementation of the MARS-A scheme of Binkowski and Roselle (2003)) are used. GEOS5 meteorological data from the Global Modeling and Assimilation Office (GMAO) at the NASA Goddard Space Flight Centre for 2006 are used. We note that 2006 was a climatologically warm year in the US, with the annual average temperature 0.6 °C warmer than the 1995–2015 mean (NOAA 2015). The EPA National Emissions Inventory for 2005 is used for anthropogenic emissions in the US. We note that emissions have declined since, which will introduce an unquantified uncertainty to the size of sensitivities. In particular, decreasing SO_2 emissions will likely lead to a greater impact of NO_x emissions on ammonium nitrate particulate matter formation due to increased availability of free ammonia (Woody *et al* 2011). Decreasing NO_x emissions will likely lead to greater ozone sensitivity to NO_x emissions, as net ozone production is increased as a result of reduced titration of ozone at low NO_x concentrations (Seinfeld and Pandis 2006).

The GEOS-Chem adjoint simulations upon which the air quality sensitivity matrices are based are run for a 15-month period. The first 3 months of the (backward) simulation are used as the adjoint spin-up time, during which the model is run, but the outputs are not included in the analysis, to ensure that any initial conditions do not contribute significantly to the annual air quality impacts. We calculate the sensitivity of two objective functions (relevant to health impacts) with respect to NO_x emissions. The first objective function is defined as the annually averaged US population exposure to $PM_{2.5}$, and the second as the one-hour daily maximum US population exposure to ozone during the ozone season (May to September). We calculate exposure to one-hour daily maximum ozone given that this is the exposure metric linked to increased risk of premature mortality in the epidemiological work used in this study (Jerrett *et al* 2009). Morbidity outcomes, which are associated with the regulatory metric of eight-hour maxima, are calculated by assuming a constant correction factor between the one-hour and eight-hour maximum (Thurston and Ito 2001). The implications of this correction factor are discussed in the supplementary material. The population exposure impacts are quantified by performing an inner (Frobenius) product of the sensitivity matrix with the spatially resolved VW excess NO_x emissions map, obtained as described in section 2.3. This operation gives us the aggregate US air quality impacts change attributable to these emissions.

We estimate uncertainty in the $PM_{2.5}$ exposure calculations arising from the baseline atmospheric model (GEOS-Chem) as well as the uncertainty in applying the adjoint-based approach for capturing marginal impacts. Based on a comparison with annually averaged observational Federal Reference Method (FRM) $PM_{2.5}$ data from the EPA's Air Quality System (AQS) database from ~1000 locations in the CONUS, the AQS $PM_{2.5}$ values are on average ~40% higher than those predicted by GEOS-Chem (corrected to include particle bound water for comparison purposes using the Aerosol Inorganic Model by Clegg *et al* 1998). When comparing different atmospheric model approaches for capturing marginal impacts (Caiazzo *et al* (2013), Fann *et al* (2013) and Dedoussi and Barrett (2014) for the road transportation sector, correcting for the absence of secondary organic aerosols, and accounting for the different definitions of the mobile/road transportation sectors amongst studies), we find that these can be up to ~40% lower than the adjoint-based approach. Given the biases from these modeling and measurement-based comparisons, we apply a normally distributed multiplicative factor to the $PM_{2.5}$ sensitivity matrix calculated values with mean 1 and standard deviation 0.4. The bias of grid resolution is not captured, as it has been shown that CONUS $PM_{2.5}$ health impacts are largely unaffected by grid resolution in fine resolutions (Thompson *et al* 2014b, Arunachalam *et al* 2011). We note that model uncertainty may not be fully captured given the interacting issues of resolution, emissions, meteorology, and chemical processes, as well as the limited coverage of monitors, mismatch in scales, and potential for canceling errors in the model.

Uncertainty in the ozone estimates are estimated by comparing modeled and measured one-hour daily maximum ozone concentrations at monitoring locations across the US. Observational data are obtained for ~1200 sites from the EPA's AQS database. The normalized mean bias (NMB) from all locations is distributed with an average NMB of 25.3% and NMB standard deviation of 17.9%. We draw from this assuming a normal distribution and multiply the ozone impacts with its reciprocal as a corrective factor. This follows the uncertainty quantification done in Caiazzo *et al* (2013).

2.5. Health impacts

For a given estimate of total unreported NO_x emissions, the adjoint-based model described in section 2.4 is applied to determine the increase in total population exposure to both annual $PM_{2.5}$ and one-hour daily maximum ozone. Epidemiological concentration-response functions (CRFs) are applied to calculate the increase in premature mortality and morbidity (i.e. non-fatal) outcomes. Premature mortalities are calculated by assuming a log-linear CRF. Function parameters are derived from an American Cancer Society study (Krewski *et al* 2009, Jerrett *et al* 2009). Krewski *et al* (2009) correlated changes in annual average daily $PM_{2.5}$ exposure with increased premature mortality due to cardiopulmonary disease and lung cancer. Jerrett *et al* (2009) correlated increases in the daily one-hour maximum ozone exposure with premature mortality due to chronic obstructive pulmonary disease (COPD) and exacerbation of asthma. Alternative CRF shapes and parameters including the integrated exposure-response (IER) function applied in the 2010 Global Burden of Disease study (Burnett *et al* 2014, Lim *et al* 2012) are evaluated to determine the sensitivity of the result to the choice of CRF (presented in the supplementary material).

The rate of increase of relative risk with exposure is treated as two independent, uncertain variables for one-hour daily maximum ozone and annual average $PM_{2.5}$. We assume a triangular distribution with mode and 95% confidence interval taken from the epidemiological study results. Background levels of $PM_{2.5}$ and ozone are extracted from a previous forward $\sim$50 km resolution GEOS-Chem simulation of US air quality, with a population-weighted average background of 8.18 μgm^{-3} for $PM_{2.5}$ and 39.7 ppbv for ozone in 2006. The background distribution is taken from archived GEOS-Chem simulation data. Since the health impact functions are all assumed to be linear or log-linear with no lower threshold, increases in background exposure only have a significant effect at background levels higher than those in the US. Highly non-linear CRFs such as the integrated exposure response functions discussed in the supplementary material are more sensitive to background concentrations.

Morbidity impacts are estimated using linear CRFs from the 2005 update to the methodology for the European externalities of energy (ExternE) project (Bickel and Friedrich 2005). Morbidity outcomes include the increase in bronchodilator usage days, lower respiratory symptom days, minor restricted activity days (including work loss days), hospital admissions, and new cases of chronic bronchitis. Alternative response functions based on data from the EPA are also discussed in the supplementary material.

The gridded population distribution for the US applied in the GEOS-Chem adjoint-based model is taken from the Global Rural Urban Mapping Project (GRUMP) (Balk *et al* 2006) for 2006. This distribution is embedded in the adjoint sensitivity matrix, and the distribution is therefore assumed to be constant between target years. Estimated exposure is normalized by population to yield the population-weighted average change. Total population and population fractions by age are estimated for each year using data from UN forecasts (2013). Baseline mortality rates per 1000 people for each disease of interest are taken from the World Health Organization Global Burden of Disease report for 2012 (WHO 2014) and are assumed to remain constant.

2.6. Monetization

Increase in premature mortality is monetized using estimates of the monetary value of changes in mortality risk, expressed as the value of statistical life (VSL). Following EPA recommendations (US EPA 2014), VSL is treated as an uncertain variable. In the supplementary material, we also present results if only the mean VSL value is used for monetization. We estimate a VSL distribution for 2015 by adjusting the original study values from previous years used by EPA in terms of inflation and real income growth. Inflation is considered using the US Bureau of Economic Analysis' implicit price deflator for gross domestic product (GDP) (US Bureau of Economic Analysis 2015). Real income growth is considered through changes in the full-time employed median usual weekly earnings (US Bureau of Labor Statistics 2015) and assuming a VSL income elasticity of 0.4, consistent with the recommended central value in EPA's Benefits Mapping and Analysis Program-Community Edition (BenMAP-CE) model (RTI International 2015). A Weibull distribution is fitted to the augmented VSL data, yielding a shape factor of 1.5 and a scale factor of 8.9 for 2015. The resulting mean VSL is $8.1 million (2015 dollars). Alternative approaches for adjusting the EPA-recommended distribution of VSL values to 2015 include using the Consumer Price Index instead of the GDP deflator and using the high (1.0) and low (0.08) values of the EPA BenMap-CE VSL income elasticity recommendations. These alternative approaches yield mean 2015 VSLs within -1% to $+4\%$ of the mean value used in this study. The median VSL results of the alternative approaches are presented in the supplementary material.

For past years, the original VSL estimates considered by EPA are augmented by adjusting for income growth and expressing them in 2015 US dollars. For estimating the benefits associated with a return to compliance for the current fleet with the Clean Air Act after 2015, future year VSL distributions need to be estimated. We adjust 2015 VSL values by accounting for long-term annual real GDP growth in the US out to 2040 at a compound annual growth rate of 1.7% (OECD 2014) and assuming a VSL income elasticity of 0.4 as recommended in EPA BenMap-CE. The resulting VSL distributions for 2008–2040 are shown in the supplementary material.

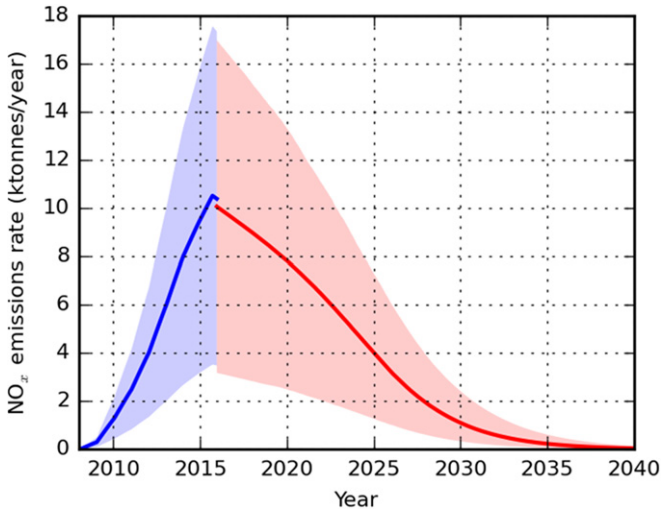

Figure 1. Annual excess VW light duty diesel vehicle NO_x emissions in kilotonnes (million kg). Results from 2008 through 2015 (blue) are estimates of actual excess emissions. Results from 2016 onward (red) are forecast based on the existing in-use vehicle fleet assuming no new sales of non-compliant vehicles from September 2015 and no retrofitting (used to calculate the benefit of a return to compliance). The shaded region indicates the 95% confidence interval. The discontinuity is due to the difference in the baseline for past (based on FTP-75 drive cycle measurements) and future (based on a return to regulatory limit) emissions.

Morbidity outcomes are not monetized. However, we expect mortality to capture >90% of monetized impacts (US EPA 2011). Social costs for each year are calculated by multiplying annual incidents of premature mortality with the VSL for the corresponding year, assuming the mortality lag structure recommended by the EPA for air-quality impacts. Total social costs for 2008 to 2015 from increased premature mortality are expressed in 2015 USD dollars using a social rate of time preference (discount rate) of 3% p.a. (US EPA 2014). Total social costs from additional increased premature mortality that occurs in future years if vehicles are not brought into compliance with EPA regulations are expressed in 2015 USD dollars using the same discount rate. The results for different discount rate choices are presented in the supplementary material.

3. Results

3.1. Emissions

Based on the drive cycle tests of Thompson *et al* (2014a), we estimate the NO_x emissions factors for urban driving for the affected vehicles to be 0.97 g km^{-1} (95% CI: 0.31 to 1.62) and for highway driving to be 0.50 g km^{-1} (95% CI: 0.06 to 0.94). The emissions factor based on the FTP-75 drive cycle is estimated to be 0.019 g km^{-1} (95% CI: 0.0085 to 0.0294). Based on the 2005 emissions inventory, we estimate 8.6% of VKT to be driven on rural interstates, for which we use the highway emissions factor, while the urban emissions factor is used for all other VKT. This gives a weighted average NO_x emission factor for affected vehicles across all roads of 0.93 g km^{-1} (95% CI: 0.33 to 1.53).

The excess NO_x emissions over time from the fleet of affected vehicles is shown in Figure 1. The total VKT by affected vehicles between 2008 and 2015 is estimated to be 40.5 billion km (95% CI: 39.4 to 41.6), corresponding to total excess NO_x emissions (above the emissions estimated based on the FTP-75 drive cycle) of 36.7 million kg (95% CI: 12.3 to 61.2). The total VKT expected for these vehicles from 2016 onward is estimated to be 93.4 billion km (95% CI: 80.2 to 109.9), corresponding to total excess NO_x emissions of 82.0 million kg (95% CI: 25.8 to 145.6). If these vehicles were brought into compliance with EPA regulations by the end of 2016 assuming a constant rate of repairs beginning in January 2016, we would expect the avoided VKT and NO_x emissions to be 87.3 billion km (95% CI: 74.4 to 103.6) and 76.6 million kg (95% CI: 24.1 to 136.6), respectively. Figure 2 shows the spatial distribution of excess VW light duty vehicle diesel NO_x emissions in the US, aggregated over 2008–2015, under the assumption that the excess NO_x emissions from the affected VW vehicles have the same spatial distribution as total NO_x emissions from light duty diesel vehicles.

3.2. Exposure and health impacts

The exposure and health impacts results (including the 95% CI) are shown in table 1, both integrated over the time period 2008–2015 and the forecast impacts taking a reference case of no recall. The total US population exposure of people over 30 years old to annual $PM_{2.5}$ and one-hour daily maximum ozone due to the VW excess NO_x emissions during the years 2008–2015 is calculated to be 0.78 million people-μg/m^3 (95% CI: 0.075–2.0) and 2.6 million people-ppbv (95% CI: 0.85–4.7), respectively. This results in a total of 59 (95% CI: 9.7–150) premature deaths, 87% of

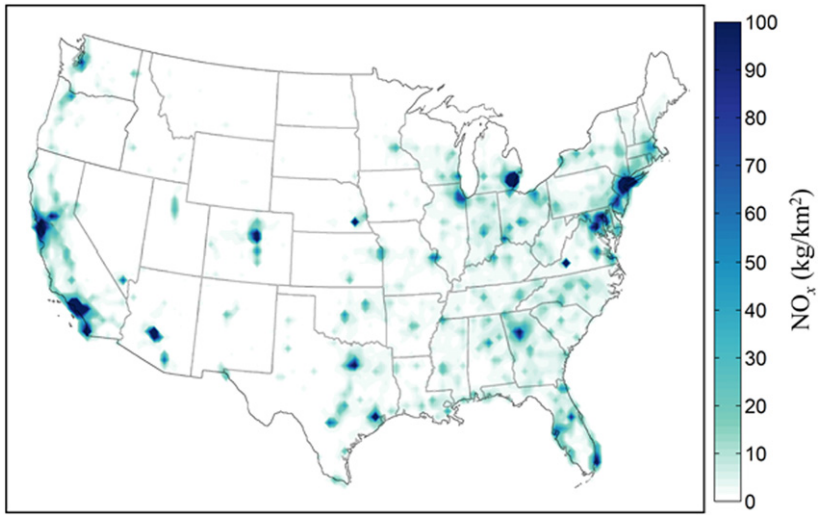

Figure 2. Distribution of estimated actual excess VW light duty diesel vehicle NO_x emissions summed over 2008 through 2015 (kg km^{-2}). The median value of emissions is used for each year. Emission density peaks at 446 kg km^{-2}.

Table 1. Estimated actual and forecast exposure and mortality impacts of excess VW light duty diesel vehicle NO_x emissions. Note that the median totals are not exactly equal to the sum of median values. Bracketed numbers are 95% confidence interval. Results after 2016 are forecast based on the existing in-use vehicle fleet assuming no new sales of non-compliant vehicles and no retrofitting (used to calculate the benefit of a return to compliance after 2015). Monetized values are adjusted to 2015 dollars. Values shown are rounded to 2 significant figures.

	Outcome	$PM_{2.5}$	Ozone	Total
2008-2015 estimated actual impacts	Exposure[a]	0.78 (0.075–2.0)	2.6 (0.85–4.7)	–
	Early deaths	51 (4.6–130)	7.7 (1.4–20)	59 (9.7–150)
	Mortality cost (million USD)	380 (34–1100)	62 (11–190)	450 (72–1200)
Forecast impacts from 2016 assuming no recall	Exposure[a]	1.9 (0.17–4.9)	6.3 (2.0–12)	–
	Early deaths	120 (10–330)	19 (3.3–49)	140 (23–370)
	Mortality cost (million USD)	770 (66–2200)	130 (22–370)	910 (140–2500)
Benefit of a recall	Avoided exposure[a]	1.8 (0.13–4.6)	5.9 (1.6–11)	–
	Avoided deaths	110 (8.5–320)	17 (2.8–47)	130 (18–350)
	Avoided cost (million USD)	710 (48–2100)	120 (18–350)	840 (110–2300)

[a] Exposure is in millions of people over 30, in μg m^{-3} or ppbv for $PM_{2.5}$ or ozone, respectively.

which are attributable to the $PM_{2.5}$ exposure and 13% to ozone exposure. Extended results are shown in the supplementary material.

In addition to increased premature mortalities, this increase in the population exposure to $PM_{2.5}$ and ozone is estimated to result in 31 (95% CI: −38 to 170) cases of chronic bronchitis and 34 (95% CI: −1.9 to 100) hospital admissions. The hospital admissions include respiratory and cardiac hospital admissions. We calculate that there will be ∼120 000 minor restricted activity days (including work loss days), ∼210 000 lower respiratory symptom days, and ∼33 000 additional bronchodilator usage days.

Assuming no new sales of the affected models from September 2015 and that no retrofitting of the affected VW vehicles occurs, we forecast the corresponding additional mortality and morbidity impacts until these vehicles are eventually disposed of. An additional 140 (95% CI: 23 to 370) premature deaths are forecast due to these NO_x emissions in excess of EPA compliant levels. In terms of the morbidity impacts we forecast 86 (95% CI: −91 to 460) additional chronic bronchitis cases and 87 (95% CI: −10 to 270) hospital admissions. Future minor restricted activity days are estimated to be increased by ∼270 000, lower respiratory symptom days by ∼490 000 and increased bronchodilator usage days by ∼77 000. However, we find that a full recall, beginning in January 2016 and ending in December 2016, would avoid 130 (95% CI: 18–350) of the 140 attributable future mortalities.

3.3. Social costs

Monetized mortality costs from 2008 until the end of 2015 due to the excess VW NO_x emissions are estimated at $450m (95% CI: $72m to $1.2 bn), while future costs if there is no recall (but no further sales from September 2015) are forecast to be $910m (95% CI: $140m to $2.5bn). The total cost that will occur without recall is therefore expected to be ∼$1.4bn in 2015 USD, or ∼$2800 per vehicle. However, assuming that vehicles are recalled at a constant rate from the start of 2016 and all devices replaced by the end of 2016, the total cost of future mortality impacts could

be reduced by 93% to $61m. This is equal to 62% of the total projected costs from 2008 to 2040.

4. Conclusions and discussion

4.1. Results and context

We estimate the public health impacts and associated costs of the alleged CAA violations by VW due to defeat devices being present in model year 2009–2015 light duty diesel vehicles with 2.0 litre engines. An estimated ~36.7 million kg of excess NO_x emissions occur from 2008 to 2015. Our computed excess NO_x emissions in 2015 are equivalent to ~1% of the total light duty vehicle emissions.

We estimate that ~59 early deaths will be caused by 2008–2015 excess emissions with a monetized cost of ~$450m. (Some of the estimated deaths caused by historical emissions have not yet occurred due to the cessation lag structure assumed.) Morbidity impacts include ~31 cases of chronic bronchitis, ~34 hospital admissions, ~120 000 minor restricted activity days, ~210 000 lower respiratory symptom days, and ~33 000 days of increased bronchodilator usage.

We compare our results to non-peer reviewed estimates that have appeared in the press in the month following EPA's Notice of Violation (NYT 2015, Vox 2015, AP 2015). These estimates consider excess NO_x emissions that have occurred from 2008 to 2015 only. Results from these studies, which consider only $PM_{2.5}$ exposure, range from 16 to 106 additional cases of premature mortality due to excess VW vehicle NO_x emissions, compared to our median result of 51, and 95% confidence interval of 4.6 to 130 cases for $PM_{2.5}$ due to NO_x only. For 2015 specifically, AP (2015) estimated 5 to 15 additional cases of premature mortality due to $PM_{2.5}$ compared to our median estimate of 14 cases in 2015.

The air pollution mortalities may also be compared to fatal accidents, another major adverse consequence of vehicle use. We estimate the vehicles of concern will have driven 40.5 billion km from 2008 to the end of 2015, which implies approximately 280 accident fatalities over this time period for a fleet of the size of the VW light duty diesel vehicles considered here when using average US fatality rates (ITF 2014, NHTSA 2015). Per kilometer driven, the air pollution death rate from the excess NO_x emissions is therefore ~20% of the accident fatality rate for an average US passenger car.

If no recall is made we estimate that the existing affected vehicles will cause ~140 early deaths from 2016, with a monetized cost of ~$910m. However, if the vehicles are recalled and brought into compliance by the end of 2016 then 93% of these deaths and 92% of the costs can be avoided. Including the early deaths that have occurred or are already bound to occur, this means that bringing the vehicles into compliance in 2016 can avert 66% of total early deaths and 62% of monetized costs.

4.2. Limitations and uncertainties

We now note unquantified uncertainties in this study, some of which it may be possible to quantify with further research. The fleet model uses US average car purchasing, use, and disposal trends. These may vary for the specific VW application. Any effect of the December 2014 voluntary recall has not been factored in to damage estimates for 2015 due to lack of data. The air quality sensitivity matrices are based on 2005–2006 data, but may have changed over time since then. Toxicity of different PM species is assumed equal, consistent with EPA practice. However, as NO_x emissions impact ammonium nitrate most strongly, this would amplify the impact of any differential toxicity relative to the basket of urban PM for which CRFs are derived. Secondary organic aerosol impacts have not been included in the health impact assessment. The time after exposure changes when changes in risk occurs is an uncertainty, where we have applied a cessation lag structure to $PM_{2.5}$ mortality impacts consistent with EPA practice. This lag structure reduces valuations by <10%, suggesting it is not a major contributor to uncertainty. The basis upon which to estimate excess emissions is limited to the number of available measurements. In terms of monetizing changes in premature mortality, social cost uncertainty exists beyond the EPA-recommended uncertainty quantification applied in this study. Additional uncertainty may stem from unknown heterogeneity in the valuation of mortality risk changes by age cohort and health status, and from an unknown degree of bias from benefits transfer.

Finally, we note that there may have been environmental benefits of the defeat device that we have not computed. For example, the reduced use of diesel exhaust fluid in selective catalytic reduction may potentially have also reduced ammonia slip, and therefore associated $PM_{2.5}$ exposure and health impacts. In particular, Dedoussi and Barrett (2014) find that total $PM_{2.5}$ exposure attributable to road transportation is caused approximately equally by NO_x and NH_3 emissions.

4.3. Policy implications

In the 18 September 2015 EPA letter to VW, EPA alleged that VW violated section 203 (a)(3)(B) of the CAA, 42 U.S.C. § 7522(a)(3)(B), because of the 'defeat devices' (ECM software). VW admitted the presence of defeat devices. This can incur a civil penalty of up to $3750 (or $2750 for violations prior to 13 January 2009) for each occurrence (CAA § 205(a), 42 U.S.C §7524(a), 40 C.F.R. §19.4). We refer to this as the 'defeat devices fine'. EPA also alleged that VW violated section 203(a)(1) of the CAA, 42 U.S.C. § 7522(a)(1) because the defeat devices mean that the vehicles 'do

not conform in all material respects to the vehicle specifications described in the applications for the certificates of conformity that purportedly cover them'. Violation of section 203(a)(1) of the CAA, 42 U.S.C. § 7522(a)(1) can result in a civil penalty of up to $37 500 (or $32 500 for violations prior to 13 January 2009) for each occurrence (CAA § 205(a), 42 U.S.C §7524(a), 40 C.F.R. §19.4). We refer to this latter as the 'COC fine'.

Here we do not consider the legal strength of EPA's argument, but comment on the public policy aspects. For a policy that aims to increase social welfare, the level of fines for a given violation should be considerably higher than the marginal social cost of that violation (Polinsky and Shavell 1992). Our estimates suggest that the median monetized mortality cost of the alleged violations over the lifetime of a vehicle is ~$2800, and may be as high as $7500. This suggests that the maximum defeat device fine alone would not be significantly higher than the social cost of the violation. However, if the COC fine can be assessed, then total maximum fines of $41 250 would be at least five times higher than the social costs of the fine, and likely 15 times higher. This suggests that the CAA and its implementing regulations provide sufficient scope to assess a fine that exceeds the social costs, potentially by a significant factor. We note that this is based on the expected full social costs from those vehicles already sold if the issue were not corrected. If instead only actual damages to be incurred from excess emissions until the end of 2015 are considered, then the social cost per vehicle is estimated to be ~$940, or at most $2600. In this case the same broad conclusions would apply—that the defeat devices fine alone could not significantly exceed the social costs, but that the defeat devices and COC fines in combination do provide such scope.

Finally, we note that while the 18 September 2015 EPA letter to VW cites ozone exposure resulting from the excess NO_x emissions as a concern, we find that 87% of deaths are due to fine particulate matter exposure, with 13% due to ozone.

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References

AP 2015 AP analysis: Dozens of deaths likely from VW pollution dodge *Associated Press* 3 October 2015 edition (http://bigstory.ap.org/article/a6925f0af82e44aaa1a1ed4b55d030f6/ap-analysis-dozens-deaths-likely-vw-pollution-dodge)

ARB 2015 California Air Resources Board letter (18 September 2015) to Volkswagen Group of America, Inc. (http://arb.ca.gov/newsrel/in_use_compliance_letter.htm)

Arunachalam S, Wang B, Davis N, Baek B H and Levy J I 2011 Effect of chemistry-transport model scale and resolution on population exposure to $PM_{2.5}$ from aircraft emissions during landing and takeoff *Atmos. Environ.* **45** 3294–300

Balk D L, Deichmann U, Yetman G, Pozzi F, Hay S I and Nelson A 2006 Determining global population distribution: methods, applications and data *Adv. Parasitol.* **62** 119–56

Bastani P, Heywood J B and Hope C 2012 The effect of uncertainty on US transport-related GHG emissions and fuel consumption out to 2050 *Transport. Res. A-Pol* **46** 517–48

Bell M L, McDermott A, Zeger S L, Samet J M and Dominici F 2004 Ozone and short-term mortality in 95 US urban communities, 1987–2000 *J. Am. Med. Assoc.* **292** 2372–8

Bey I, Jacob D J, Yantosca R M, Logan J A, Field B D, Fiore A M, Li Q, Liu H Y, Mickley L J and Schultz M G 2001 Global modeling of tropospheric chemistry with assimilated meteorology: Model description and evaluation *J. Geophys. Res.* **106** 23073–95

Bickel P and Friedrich R (ed) 2005 *ExternE—Externalities of Energy: Methodology 2005 Update* (Luxembourg: European Commission)

Binkowski F S and Roselle S J 2003 Models-3 community multiscale air quality (CMAQ) model aerosol component 1. Model description *J. Geophys. Res. Atmos.* **108** 4183

Burnett R T *et al* 2014 An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure *Environ. Health Perspect.* **122** 397–403

Caiazzo F, Ashok A, Waitz I A, Yim S H L and Barrett S R H 2013 Air pollution and early deaths in the United States. Part I: Quantifying the impact of major sectors in 2005 *Atmos. Environ.* **99** 610–7

Clegg S L, Brimblecombe P and Wexler A S 1998 A thermodynamic model of the system H^+—NH_4^+—SO_4^{2-}—NO_3^-—H_2O at tropospheric temperatures *J. Phys. Chem.* **102A** 2137–54

Damian V, Sandu A, Damian M, Potra F and Carmichael G R 2002 The kinetic preprocessor KPP-a software environment for solving chemical kinetics *Comput. Chem. Eng.* **26** 1567–79

Dedoussi I C and Barrett S R H 2014 Air pollution and early deaths in the United States. Part II: Attribution of $PM_{2.5}$ exposure to emissions species, time, location and sector *Atmos. Environ.* **79** 198–208

Dockery D W, Pope C A, Xu X, Spengler J D, Ware J H, Fay M E, Ferris B G and Speizer F E 1993 An association between air pollution and mortality in six US Cities *New Engl. J. Med.* **329** 1753–9

Fann N, Fulcher C M and Baker K 2013 The recent and future health burden of air pollution apportioned across US Sectors *Environ. Sci. Technol.* **47** 3580–9

Henze D K, Hakami A and Seinfeld J H 2007 Development of the adjoint of GEOS-Chem *Atmos. Chem. Phys.* **7** 2413–33

ITF 2014 *International Transport Forum Road Safety Annual Report 2014* (http://internationaltransportforum.org/Pub/pdf/14IrtadReport.pdf)

Jerrett M, Burnett R T, Pope C A, Ito K, Thurston G, Krewski D, Shi Y, Calle E and Thun M 2009 Long-term ozone exposure and mortality *New Engl. J. Med.* **360** 1085–95

Koo J, Wang Q, Henze D K, Waitz I A and Barrett S R H 2013 Spatial sensitivities of human health risk to intercontinental and high-altitude pollution *Atmos. Environ.* **71** 140–7

Krewski D *et al* 2009 Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality *HEI Research Report* 140 (Boston, MA: Health Effects Institute)

Lee C J, Martin R V, Henze D K, Brauer M, Cohen A and van Donkelaar A 2015 Response of global particulate-matter-related mortality to changes in local precursor emissions *Environ. Sci. Technol.* **49** 4335–44

Lim S S *et al* 2012 A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010 *Lancet* **380** 2224–60

NHTSA 2015 Fatality analysis reporting system (FARS) encyclopedia (http://www-fars.nhtsa.dot.gov/Main/index.aspx)

NOAA 2015 *Climate at a Glance* National Centers for Environmental Information (NCEI) (https://ncdc.noaa.gov/cag/time-series/us/110/0/tavg/ytd/12/1995-2006?base_prd=true&firstbaseyear=1995&lastbaseyear=2005)

NYT 2015 How many deaths did Volkswagen's deception cause in the US? *New York Times* 18 September 2015 (http://nytimes.com/2015/09/29/upshot/how-many-deaths-did-volkswagens-deception-cause-in-us.html?_r=0)

OECD 2014 Economic Outlook No 95—May 2014—Long-term baseline projections. OECD.Stat (http://stats.oecd.org/Index.aspx?DataSetCode=EO95_LTB)

Polinsky M A and Shavell S 1992 Enforcement costs and the optimal magnitude and probability of fines *J. Law and Econ.* **35** 133–48

Pope C III, Burnett R T, Thun M J *et al* 2002 Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution *J. Am. Med. Assoc.* **287** 1132–41

RTI International 2015 BenMAP-CE Environmental Benefits Mapping and Analysis Program—Community Edition User's Manual. Prepared for Office of Air Quality Planning and Standards US Environmental Protection Agency (http://www2.epa.gov/benmap/manual-and-appendices-benmap-ce)

Seinfeld J H and Pandis S N 2006 *Atmospheric chemistry and physics: from air pollution to climate change* (Hoboken, NJ: Wiley)

Thompson G J, Carder D K, Besch M C, Thiruvengadam A and Kappanna H K 2014a In-use emissions testing of light-duty diesel vehicles in the United States *Final Report* Center for Alternative Fuels, Engines & Emissions, West Virginia University (www.theicct.org/sites/default/files/publications/WVU_LDDV_in-use_ICCT_Report_Final_may2014.pdf)

Thompson T M, Saari R K and Selin N E 2014b Air quality resolution for health impact assessment: influence of regional characteristics *Atmos. Chem. Phys.* **14** 969–78

Thurston G D and Ito K 2001 Epidemiological studies of acute ozone exposures and mortality *J. Expo. Anal. Env. Epidemiol* **11** 286–94

US Bureau of Economic Analysis 2015 NIPA table 1.1.9 Implicit Price Deflators for Gross Domestic Product. September 25, 2015 (http://bea.gov/faq/index.cfm?faq_id=513)

US Bureau of Labor Statistics 2015 Table 1: median usual weekly earnings of full-time wage and salary workers by sex, quarterly averages, seasonally adjusted (http://bls.gov/news.release/wkyeng.t01.htm)

US EPA 2006 *Mercedes-Benz USA, LLC and DaimlerChrysler AG Clean Air Act Settlement* (http://www2.epa.gov/enforcement/mercedes-benz-usa-llc-and-daimlerchrysler-ag-clean-air-act-settlement)

US EPA 2008 Documentation for the 2005 Mobile National Emissions Inventory Version 2 (http://epa.gov/ttn/chief/net/2005inventory.html)

US EPA 2011 *The Benefits and Costs of the Clean Air Act: 1990 to 2020* Final report of US Environmental Protection Agency Office of Air and Radiation

US EPA 2012 Milestones in mobile source air pollution control and regulations (http://www3.epa.gov/otaq/consumer/milestones.htm)

US EPA 2014 *Guidelines for Preparing Economic Analysis* National Center for Environmental Economics Office of Policy (http://yosemite.epa.gov/ee/epa/eed.nsf/webpages/Guidelines.html)

US EPA 2015a Notice of violation (18 September 2015) sent by EPA to Volkswagen Group of America, Inc. (http://www3.epa.gov/otaq/cert/documents/vw-nov-caa-09-18-15.pdf)

US EPA 2015b Annual certification test results & data (http://www3.epa.gov/otaq/crttst.htm)

US EPA 2015c EPA, California Notify Volkswagen of Clean Air Act Violations / Carmaker allegedly used software that circumvents emissions testing for certain air pollutants (http://yosemite.epa.gov/opa/admpress.nsf/6424ac1caa800aab85257359003f5337/dfc8e33b5ab162b985257ec40057813b!OpenDocument)

Vox 2015 How many deaths did Volkswagen's pollution scandal cause? *Vox* 24 September 2015 (http://vox.com/2015/9/23/9383641/volkswagen-scandal-pollution)

VW of America 2015 *TDI: U.S. Market Success* Diesel Technology Forum March 2015

WardsAuto Group 2015 *US Sales of Selected VW Models by Powertrain, CY2008-Present* WardsAuto InfoBank

WHO 2006 *Health Risks of Particulate Matter from Long-range Transbonudary Air Pollution* (www.euro.who.int/__data/assets/pdf_file/0006/78657/E88189.pdf)

WHO 2008 *Health Risks of Particulate Matter from Long-range Transboundary Air Pollution* (www.euro.who.int/__data/assets/pdf_file/0005/78647/E91843.pdf)

WHO 2014 *Disease and Injury Country Mortality Estimates for 2012* (World Health Organization)

Woody M, Baek B H, Adelman Z, Omary M, Lam Y F, West J J and Arunachalam S 2011 An assessment of Aviation's contribution to current and future fine particulate matter in the United States *Atmos. Environ.* **45** 3424–33

WSJ 2015 Volkswagen to launch diesel-car recall in January *The Wall Street Journal* 7 October 2015 (http://wsj.com/articles/volkswagen-to-launch-massive-vehicle-recall-in-january-1444213610)