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Health benefits of reducing NO_x emissions in the presence of epidemiological and atmospheric nonlinearities

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Abstract

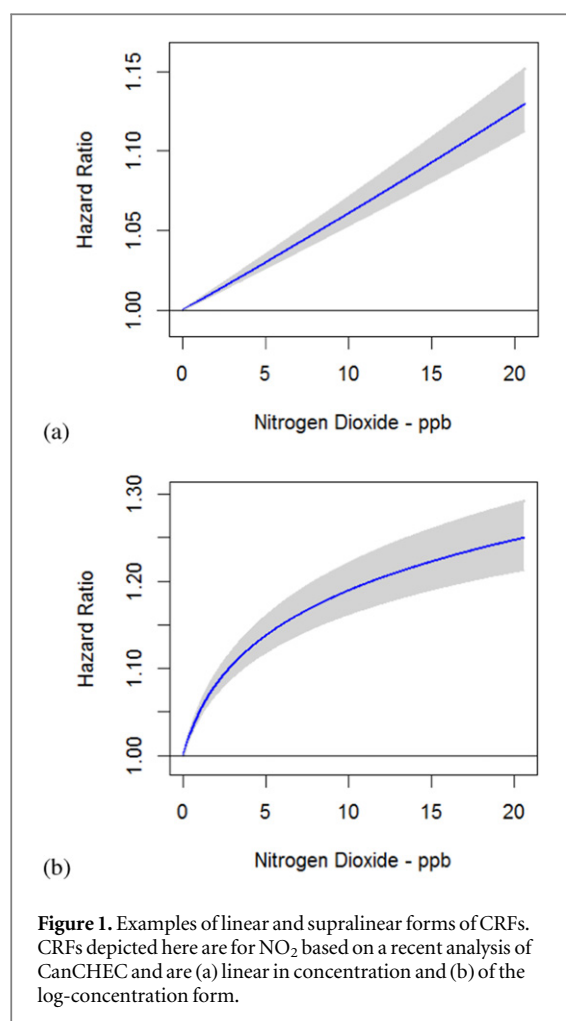
Recent epidemiological evidence suggests that the logarithm of concentration is a better predictor of mortality risk from long-term exposure to ambient PM_{2.5} and NO₂ than concentration itself. A log-concentration-response function (CRF) predicts a heightened excess risk per unit concentration at low levels of exposure that further increases as the air becomes less polluted. Using an adjoint air quality model, we estimate the public health benefits of reducing NO_x emissions, on a per-ton and source-by-source basis. Our estimates of benefits-per-ton assume linear in concentration and log-concentration CRFs for NO₂ and a CRF that is linear in concentration for O₃. We apply risk coefficients estimated using the Canadian Census Health and Environment Cohort. We find that a log-concentration CRF for NO₂ leads almost consistently to larger benefits-per-ton than a linear in concentration CRF (e.g., \$500 000 ton⁻¹ compared to \$270 000 ton⁻¹ for Ottawa). We observe that concentrations gradually decline due to widespread, progressive emissions abatement, entailing increasing health benefits as a result of (1) a log-concentration CRF for NO₂ and (2) the nonlinear response of O₃ to NO_x emissions. Our results indicate that NO_x abatement has the potential to incur substantial and increasing health benefits, by up to five times with 85% emission reductions, for Canada into the future.

1. Introduction

Managing the public health burden of ambient air pollution is a complex undertaking informed by atmospheric science and engineering, health, economic, and policy disciplines. Synthesis of information from these fields can yield insight into the public health impacts of air pollution, playing a critical role in science-based decision-making. Perhaps the most tangible form of quantitative assessments is one that links public health with sources of emissions themselves, yielding direct decision metrics. One useful metric in this context is benefit-per-ton (BPT; synonymous with marginal benefit/damage), which refers to the monetized health benefit of reducing 1 ton of emissions at their source. The BPT metric applies monetary value to aggregate health damages (such as mortality or morbidity counts) attributed to a 1 ton change in pollutant emissions. The utility of such a metric lies in its ability to be directly compared with

the cost-per-ton of emission reduction (emission abatement) in a benefit-cost assessment framework.

Numerous, complex atmospheric processes act on emitted species to transport and transform them into pollutants such as O₃, NO₂, and PM_{2.5} at the point of exposure. The potential for secondary pollutant formation often depends nonlinearly on the abundance of precursor species in ambient air. Nonlinearity in atmospheric processes implies that the same ton of emission control would yield different health impacts, and different BPT estimates, at different levels of emissions or emissions abatement. Such nonlinearity in BPT estimates with emissions abatement has been discussed in the literature for O₃ and its precursors (Repetto 1987, Hakami *et al* 2004, Pappin *et al* 2015). A recent study by Pappin *et al* (2015) found increasing BPT estimates for NO_x control in the US with widespread and progressive emissions abatement, due solely to nonlinear atmospheric chemistry and for acute O₃ exposure mortality using a single-pollutant



model. In other words, contrary to the commonly held view in environmental economics (Goodstein 1995, Hussen 2004), each additional ton of NO_x emission control makes future abatement efforts more rewarding. While sufficient evidence of compounding benefits with abatement exists for O_3 and NO_x , nonlinearity in the $\text{PM}_{2.5}$ response to precursor emissions has not been studied to the same extent. Collectively in the literature, there are indications that a similar case of compounding benefits may hold for $\text{PM}_{2.5}$ (Fann *et al* 2012, Zhang *et al* 2012, Holt *et al* 2015), though further research is needed to support this assertion.

In addition to a nonlinear atmospheric response, a second, and potentially significant, source of nonlinearity is that induced by concentration-response functions (CRFs). CRFs describe the relationship between pollutant concentrations and excess risk of a health endpoint. Traditionally, CRFs are modeled as linear functions, with log-transformed relative risk/hazard ratio (RR/HR) being linear with respect to concentration (figure 1(a)). A linear in concentration model choice implies a constant increased risk per unit change in exposure, independent of the level of exposure itself. An alternate form of CRF found in the epidemiological literature is a supralinear curve,

where the excess risk per unit concentration (i.e., the slope of the CRF) is highest at low levels of exposure, and gradually declines as the environment becomes more polluted (figure 1(b)). Henceforth, we refer to this form as a log-concentration CRF (log-transformed RR/HR is a linear function of log-transformed concentration). Such a supralinear CRF has recently been proposed as a more fitting model choice for $\text{PM}_{2.5}$ (Pope *et al* 2011, Burnett *et al* 2014, Crouse *et al* 2015) and NO_2 in Canada (Crouse *et al* 2015) for mortality and other endpoints. A log-concentration CRF implies an increased sensitivity of populations in clean environments to any changes in $\text{PM}_{2.5}$ or NO_2 exposure.

Complexities in atmospheric processes necessitate the use of sophisticated models that adequately describe the potentially nonlinear pathway from sources of emissions to exposed populations. A general lack of efficient modeling methods has prevented full characterization of BPTs in the literature for various sources across a range of abatement levels/scenarios. Further, no previous study has examined both the atmospheric response of ambient concentrations to emissions and the shape of the CRF as potential sources of nonlinearity in the health benefits of abatement (e.g., Pappin *et al* 2015, Pope *et al* 2015). We aim to examine the role of both factors in predicting the BPTs of NO_x emission control in Canada.

2. Methods

We investigate how different forms of the CRF influence the health benefits of emission control. We do so using an atmospheric chemical transport model (CTM) run for 2007 emissions and meteorological conditions in North America and various, hypothetical emissions control or abatement scenarios. We use exposure estimates from a forward CTM simulation, combined with linear in concentration and log-concentration CRFs, to inform a set of adjoint (reverse or backward) simulations. The utility of an adjoint model is in its ability to estimate BPTs of emission control on a source-by-source basis (Pappin and Hakami 2013). We account for the public health impacts of O_3 and NO_2 exposure in Canada, but not $\text{PM}_{2.5}$, as the adjoint model for PM processes is still under development. While the public health burden of $\text{PM}_{2.5}$ exceeds that of O_3 (Lim *et al* 2012), NO_x emissions have a significant impact on Canadian mortality due to O_3 and NO_2 exposure (Pappin and Hakami 2013). Furthermore, Crouse *et al* (2015) demonstrate that both O_3 and NO_2 contribute additional mortality risk to that predicted by $\text{PM}_{2.5}$ in a large, nationally representative Canadian cohort.

Our focus is on chronic exposure mortality, as recent epidemiological studies suggest that long-term exposure to criteria pollutants poses a substantially higher risk of mortality than short-term exposure (Jerrett *et al* 2009, Krewski *et al* 2009, Crouse *et al* 2012).

We recognize that evidence for a causal association between NO₂ exposure and mortality is an evolving area of research. A recent risk assessment for ambient NO₂ found the collective evidence to be suggestive of a causal association between NO₂ exposure in the long-term and mortality (Health Canada 2016). Two other analyses of CanCHEC (Crouse *et al* 2015) and the American Cancer Society Cancer Prevention Study II cohort (Turner *et al* 2016) found NO₂ to be an independent predictor of mortality, adding to the body of evidence supporting an NO₂ effect on mortality.

2.1. Adjoint formulation

Our estimates of abatement health benefits account for non-accidental mortality from long-term O₃ and NO₂ exposure in Canada. We apply both linear in concentration and log-concentration CRFs for non-accidental mortality based on, though not identical to, a recent analysis of CanCHEC (Crouse *et al* 2015). As mortality is the largest contributor to the total, monetized societal benefits of improving air quality (US EPA 1999, Hubbell *et al* 2005, Hubbell 2006), we consider our estimates to be largely representative of total health benefits.

CanCHEC is a population-based Canadian cohort consisting of approximately 3.6 million participants subjects >25 years of age who filed the long-form census in 1991. It has been linked to the Canadian Mortality Database from the time of enrollment through 2006. Participants' exposure estimates for summertime O₃ are from a combination of ground monitoring observations and atmospheric modeling predictions for 2002–2009 (Robichaud and Ménard 2014). Annual mean NO₂ concentrations are derived from a national land use regression model for 2006 (Hystad *et al* 2011). Exposures are assigned to each subject's postal code based on annual income tax data from 1984–2006 (Peters *et al* 2013). Several individual-level covariates are included in the Cox proportional hazards model that relates mortality to known risk factors such as income, education, and occupation, in addition to contextual risk factors representing both city and neighborhood characteristics. More detailed information about analysis of CanCHEC can be found elsewhere (Crouse *et al* 2012, Peters *et al* 2013, Crouse *et al* 2015).

Mathematically, BPTs estimated using adjoint sensitivity analysis are the derivatives of a cumulative health damage function with respect to emissions in each grid-cell location. In our case, this damage function, termed the adjoint cost function, is the monetized mortality count attributable to air pollution in Canada for a given exposure surface. For a linear in concentration CRF, the adjoint cost function is of the form in (1)

$$J = V_{SL} \sum_{\omega} M_{0,\omega} P_{\omega} (1 - e^{-\beta \bar{C}_{\omega}}). \quad (1)$$

For a log-concentration CRF, the adjoint cost function takes the form

$$J = V_{SL} \sum_{\omega} M_{0,\omega} P_{\omega} (1 - e^{-\beta \ln(\bar{C}_{\omega} + 1)}). \quad (2)$$

We note that equations (1) and (2) are written separately for O₃ and NO₂ using risk parameter estimates from the Cox proportional hazards model consisting of both pollutants together. In both cases, J is the monetized number of non-accidental mortalities attributable to long-term O₃ or NO₂ exposure, per year, in Canada. We apply a value of statistical life (V_{SL}) of 7.17 million dollars (2013 CAD) to each premature death in equations (1) and (2) based on that used in Health Canada's Air Quality Benefits Assessment Tool (AQBAT; Judek *et al* 2006). Above, $M_{0,\omega}$ is the non-accidental mortality rate for populations >25 years of age and P_{ω} is the population over 25 years of age, both for grid-cell location ω (derived from AQBAT). We apply risk coefficients, β , based on Cox proportional hazards models that imply linear regression for O₃ and linear or log-linear regression for NO₂ (table 1). In equations (1) and (2), \bar{C}_{ω} is the model-based, ground-level concentration of O₃ or NO₂ (in ppb) in grid-cell location ω , averaged over all simulation days. We use an 8 h averaging period for O₃ (daily maximum 8 h average; DM8A) and a 24 h (daily) averaging period for NO₂ for consistency with the exposure metrics used in CanCHEC.

Due to the computational cost of conducting full-year simulations, our CTM-based exposure estimates for O₃ and NO₂ are for the May–September period and relate to CRFs derived using summertime O₃ and annual average NO₂. Since we seek to attribute chronic exposure mortality to sources of emissions, we assume that our simulation period yields O₃ and NO₂ exposure levels typical of the May–September period in Canada, and would hence represent long-term exposure levels and source-receptor relationships.

Adjoint sensitivity analysis requires differentiation of the cost function (equations (1) and (2)) with respect to the local, hourly concentration. This differentiation yields the adjoint forcing term (φ), a key input parameter to an adjoint model used to calculate sensitivities (Hakami *et al* 2007). For a linear in concentration CRF, the adjoint forcing term is formulated as in (3)

$$\varphi_{\omega} = \frac{V_{SL} M_{0,\omega} P_{\omega} \beta e^{-\beta \bar{C}_{\omega}}}{\bar{t} n}. \quad (3)$$

The adjoint forcing term for a log-concentration CRF is formulated as

$$\varphi_{\omega} = \frac{V_{SL} M_{0,\omega} P_{\omega} \beta (\bar{C}_{\omega} + 1)^{-(\beta+1)}}{\bar{t} n}, \quad (4)$$

where all variables are as defined before, and \bar{t} is the number of hours in the daily exposure metric (8 for DM8A O₃ and 24 for daily average NO₂), and n is the number of simulation days (153 for the May–September O₃ season).

Table 1. Risk estimates and CRFs from the CanCHEC study used in estimating BPTs^a.

Two-pollutant model form	Cox proportional hazards model ^b
Linear in O ₃ Linear in NO ₂	$\ln HR = 0.0027\bar{C}_{O_3} + 0.0059\bar{C}_{NO_2} + \text{covariates}$
Linear in O ₃ ^c Log-NO ₂	$\ln HR = 0.0026\bar{C}_{O_3} + 0.0732 \ln(\bar{C}_{NO_2} + 1) + \text{covariates}$

^a Models for non-accidental mortality, 25–89 years of age.^b \bar{C}_{O_3} used in deriving the CRFs is the summertime average DM8A O₃ concentration; \bar{C}_{NO_2} is the annual average NO₂ concentration.^c The risk estimate of 0.0026 for O₃ from the two-pollutant log-NO₂ model is used for estimating BPTs.

2.2. Case study

We use the US EPA's Community Multi-scale Air Quality model (CMAQ; Byun and Schere 2006) and its gas-phase adjoint tool (CMAQ-adjoint, version 4.5.1; Hakami *et al* 2007) to conduct our analysis. Our O₃ and NO₂ exposure surfaces for Canada are spatially resolved to 36 km using forward CMAQ simulations that inform a set of reverse calculations in the adjoint model. Using the CMAQ-adjoint model, we estimate monetized O₃ and NO₂-related health benefits, attributed to an incremental (1 ton) reduction in emissions (BPTs; \$/ton). We report BPTs of NO_x control from sources in every grid-cell location, for various abatement scenarios. Our simulations are conducted over a domain spanning southern Canada, the continental US, and northern Mexico. We conduct our simulations over the O₃ season of 2007 (1 May–30 September).

To estimate nonlinearity in BPTs with abatement, we define scenarios of widespread reductions in emissions from anthropogenic sources, using 2007 as the baseline from which we reduce emissions. For each scenario, we use fixed-percentage abatement (0%, 25%, 50%, 70%, 85%) of all species emitted from anthropogenic sources within our domain. As biogenic emissions are mainly a function of meteorological conditions, we consider them to be constant in our analysis. We note that we do not perturb emissions outside of North America, as the inflow of pollution into our domain boundaries is constant from scenario to scenario.

3. Results and discussion

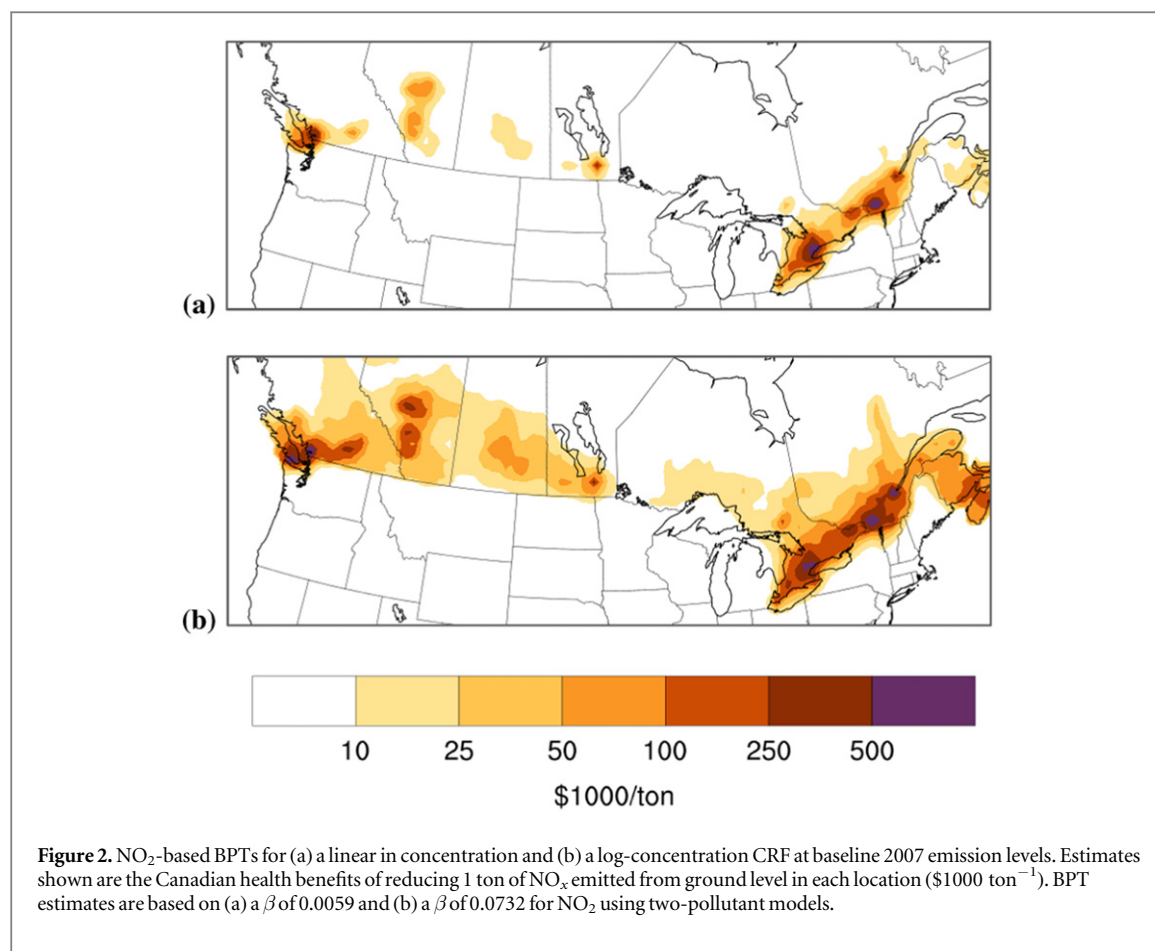
As we aim to examine the role of atmospheric chemistry and the shape of the CRF on BPT estimates, we examine O₃ and NO₂ health impacts separately. When isolating the impacts of NO_x emissions on either species, we apply the risk coefficient for only that species (table 1) in the adjoint cost function and forcing terms (equations (1)–(4)). We note that as the coefficients used in BPT calculations are from two-pollutant models with no interaction, our BPT estimates can be considered additive.

3.1. NO₂-based benefits-per-ton based on linear in concentration and log-concentration CRFs

At baseline, BPTs of NO_x control are the estimated Canadian public health benefits of reducing an additional ton of NO_x emitted from a specific source location (figure 2) in 2007. For example, an estimated value of \$50 000 ton^{−1} in figure 2 indicates that 1 ton of NO_x control in the specified location would yield estimated societal health benefits of \$50 000 ton^{−1} in Canada. We note that figure 2 depicts sources of influence, but does not provide information on the distribution of these health benefits within Canada.

We first present health benefits due to averted NO₂ chronic exposure mortality. BPTs based on a linear in concentration CRF for NO₂ (figure 2(a)) are localized around populous Canadian cities and surrounding suburban areas. BPT estimates are highly variable within Canada due to the short lifetime of NO₂ formed from emitted NO_x. BPTs for major cities in Canada vary with the size of populations susceptible to NO₂ exposure from emitted NO_x. For example, NO_x control in Vancouver incurs an estimated benefit of \$460 000 ton^{−1}, while NO_x control in Ottawa incurs a \$270 000 ton^{−1} benefit (based on a linear in concentration CRF; figure 2(a)).

In comparison to the traditional, linear in concentration form, BPTs based on a log-concentration CRF for NO₂ show greater spatial coverage (figure 2(b)). BPT estimates are almost consistently larger for the log-concentration CRF, particularly in cleaner or rural environments with low NO₂ exposure levels. For example, NO_x control in Vancouver and Ottawa incurs estimated benefits of \$510 000 and \$500 000 ton^{−1}, respectively, with a log-concentration CRF. These BPT estimates are higher than those based on a linear in concentration CRF. Of the two cities, Ottawa shows a stronger contrast between CRFs due to its low NO₂ concentrations. Low NO₂ exposure levels fall within the region of the log-concentration CRF that incurs a high incremental risk per ppb. This increased risk is larger than what would exist at the same exposure level in the linear in concentration CRF. Highly populous urban areas of Canada present an opposite case. One example is NO_x emitted in downtown Toronto, whose BPT is \$840 000 ton^{−1}.



based on the traditional linear form of CRF. Use of a log-concentration CRF for Toronto leads to a significantly lower estimate of \$650 000 ton⁻¹. Such lower BPTs based on a log-concentration CRF are due to high NO₂ concentrations in and around Toronto, where the increased risk per ppb is lower than in the linear form. NO_x emission control in Toronto translates into Canadian public health benefits both locally and downwind as NO₂ levels decline; impacts that are collectively captured in our estimates of BPTs.

The general finding of more widespread benefits of NO_x control for a log-concentration CRF indicates that the benefits of emission controls in both urban/suburban and rural areas should be considered as viable policy incentives. We note that while urban NO_x control results in lower BPT estimates under a log-concentration model in 2007, BPTs will eventually rise with continued reductions in NO₂ concentrations in the urban environment.

The choice of CRF is expected to play a more significant role in BPT estimates towards lower exposure levels or stricter emissions control policies than 2007. Dynamic changes in BPT estimates with abatement are therefore of interest, particularly as emissions in North America have been declining (Environment Canada 2014, US EPA 2015). In order to isolate the impact of nonlinearity in the CRF and/or atmospheric response on BPT estimation, we define various

abatement scenarios for which we assume unchanging population and mortality rates.

We find that BPTs are fairly constant, regardless of emission level, when a linear CRF for NO₂ is used. Constant BPTs imply that NO₂ concentrations change linearly with NO_x emissions. We therefore consider BPTs based on a linear in concentration CRF for NO₂ at the 2007 baseline (figure 2(a)) to be representative of BPTs at all abatement scenarios. As the atmospheric response of NO₂ to NO_x emissions is near linear, changes in BPT estimates across different abatement scenarios (figures 3 and 4(a)) can be attributed to non-linearity induced by the CRF. We find that a log-concentration CRF for NO₂ leads to increasing BPT estimates with more stringent abatement scenarios (figures 3 and 4(a)). This behavior exists due to the growing change in risk per unit concentration as NO₂ exposure levels decline with continued emissions abatement under the log-concentration form of CRF (figure 1(b)). For example, with large-scale, North American-wide emission reductions of 50%, the benefit of NO_x control in Toronto is estimated to be \$1250 000 ton⁻¹ (figure 3(c)); a two-fold increase from \$650 000 ton⁻¹ for the 2007 baseline scenario (figure 3(a)). At 25% abatement of 2007 emissions, Toronto's BPT lies at an estimated \$870 000 ton⁻¹ (figure 3(b)).

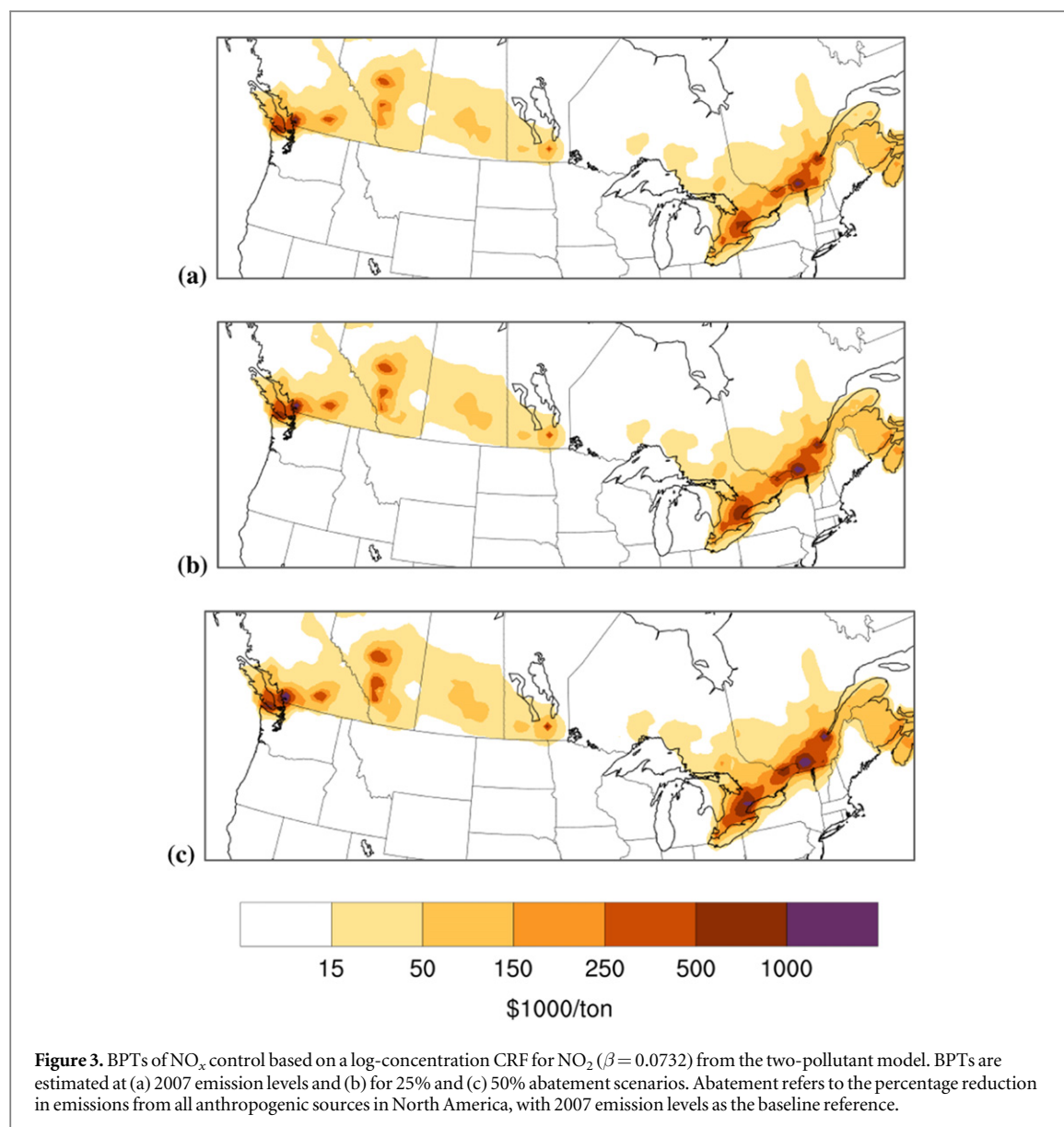


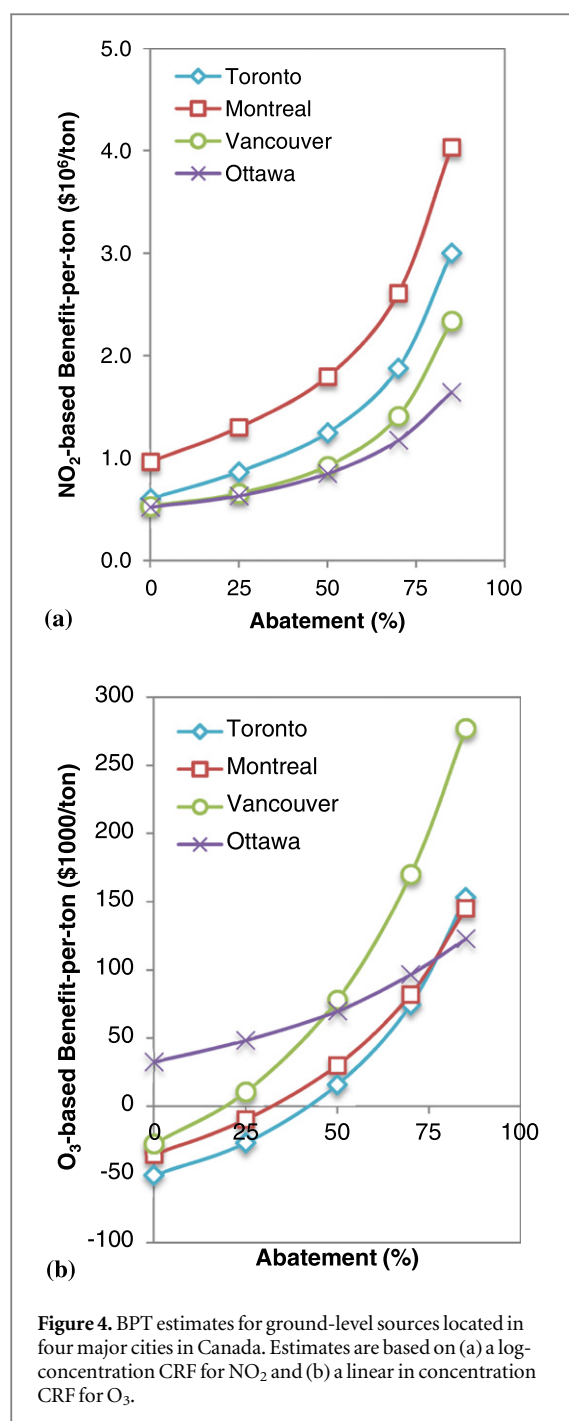
Figure 3. BPTs of NO_x control based on a log-concentration CRF for NO₂ ($\beta = 0.0732$) from the two-pollutant model. BPTs are estimated at (a) 2007 emission levels and (b) for 25% and (c) 50% abatement scenarios. Abatement refers to the percentage reduction in emissions from all anthropogenic sources in North America, with 2007 emission levels as the baseline reference.

A collocated, pair-wise comparison of BPT estimates based on linear in concentration and log-concentration CRFs for NO₂ yields insight into the predicted behavior among different statistical model choices (figure 5). At baseline 2007 emission levels, BPTs based on a log-concentration CRF for NO₂ are generally larger than those based on a linear in concentration CRF (figure 5(a)). Further examination by population density (denoted by the color of markers in figure 5) indicates that BPTs for NO_x emitted in high-population areas (yellow/orange) are comparable between the two models. On the other hand, BPTs in cleaner, low-population areas (dark blue) estimated with a log-concentration CRF are considerably larger than those based on the traditional, linear form. This trend arises because NO₂ exposure levels in these environments are on the low end of the spectrum of exposure levels in Canada, where the increased risk per ppb is heightened. This observed trend is similar among all levels of abatement, as populous areas

always have higher NO₂ concentrations than rural areas under blanket, fixed-percentage abatement scenarios. Going towards higher abatement levels (i.e., 85%; figure 5(b)), differences between the linear in concentration and log-concentration BPT estimates become more pronounced.

3.2. O₃-based benefits-per-ton based on a linear CRF

Up to this point, we have shown BPT estimates based exclusively on NO₂ chronic exposure mortality. As analysis of CanCHEC suggests that a linear in concentration CRF is the most appropriate model choice for O₃ (Crouse *et al* 2015), we apply a linear in concentration CRF to estimate O₃-based BPTs (figure 6). A noticeable feature of O₃-based BPT estimates at baseline 2007 emission levels (figure 6(a)) is their wide spatial coverage. Distant sources of influence exist for O₃, such as those in the northern US, due to its longer atmospheric lifetime and its ability to be transported over distances. At baseline, BPTs are significantly



smaller than those for NO₂ in figure 2 due to the smaller risk coefficient (table 1) and nature of NO₂ to be formed closer to populous receptor regions. In some major urban cores, O₃-based BPTs are negative (e.g., Vancouver, Montreal, and Toronto; figure 6(a)). Negative BPTs, or disbenefits, have been reported before (Pappin and Hakami 2013), and exist due to the nonlinear dependency of ground-level O₃ formation on emitted precursors (NO_x and volatile organic compounds (VOCs)). In environments with a large availability of NO_x compared to VOCs, O₃ production is suppressed. Most densely packed urban environments currently fall into this category, as their NO_x emissions exceed those of anthropogenic and biogenic

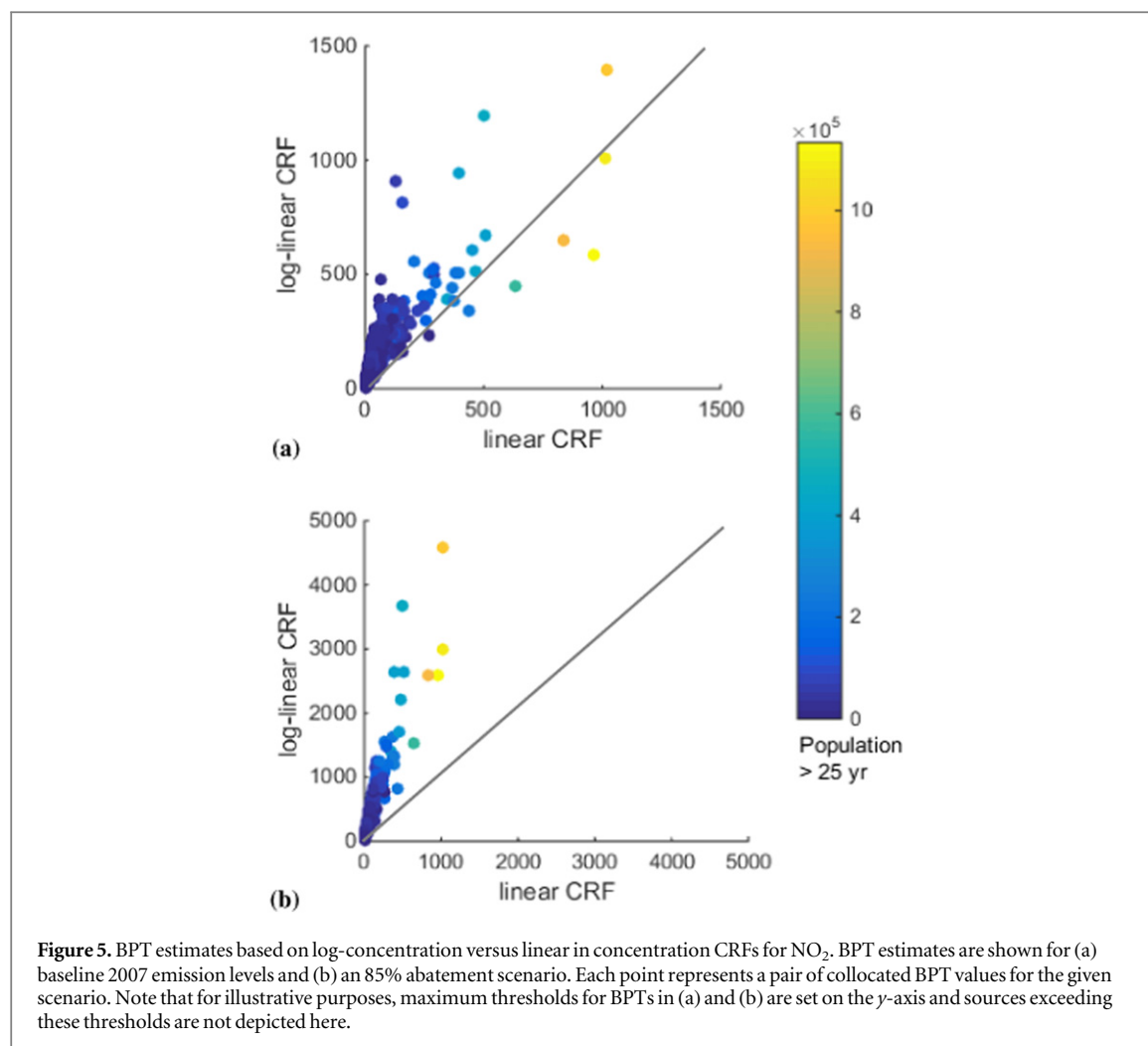
VOCs. A negative sensitivity in NO_x-inhibited environments indicates that any decrease in emitted NO_x would increase O₃ production by reducing titration of O₃ by NO. A reduction in NO_x makes available more free radicals that are necessary ingredients for producing O₃ and would otherwise have been scavenged by NO_x. One example of a negative response of O₃ to NO_x is the disbenefit in Toronto, whose BPT is $-\$50\,000\text{ ton}^{-1}$ at baseline (figure 6(a)).

As before, we depict BPT estimates for various large-scale, domain-wide abatement scenarios (25% and 50% abatement; figures 6(b) and (c) and figure 4(b)). The dominant feature of figure 6 is increasing BPTs towards higher abatement levels, as in figure 3 for NO₂. For example, Toronto's BPT that was initially estimated at $-\$50\,000\text{ ton}^{-1}$ for 2007 rises to $-\$27\,000\text{ ton}^{-1}$ with 25% abatement, and eventually becomes positive, to an estimated $\$16\,000\text{ ton}^{-1}$ with 50% abatement (figures 6 and 4(b)). Toronto's BPT estimates are one example of the widespread, increasing benefits with abatement observed across all source locations. As anthropogenic emissions are reduced, NO_x molecules are at a higher premium, and each ton of control has an increasingly important role in mitigating O₃ exposure. Similar behavior has been reported and discussed previously for the response of US acute O₃ exposure mortality to NO_x emissions (Pappin *et al* 2015).

NO₂ and O₃ present two cases of increasing BPTs with abatement for two different reasons. Compounding BPTs for NO₂ are incurred due to the shape of the log-concentration CRF. For O₃, increasing BPTs with abatement occur entirely due to atmospheric chemistry. Consideration of both NO₂ and O₃ together (i.e., the summation of figures 2 and 6(a)) would indicate compounding BPTs with continued abatement for two reasons. Regardless of the source of nonlinearity, increasing BPTs offer a new paradigm for long-term assessment of emissions abatement policies that is in contrast with the traditional view of diminishing benefits with abatement found in the environmental economics literature.

3.3. Other pollutants

While O₃ and NO₂ are major criteria pollutants in ambient air associated with chronic exposure mortality in Canada, NO_x emissions also contribute to formation of secondary inorganic PM. A more inclusive approach to estimating the BPTs of NO_x control would span over all pollutants impacted by emitted NO_x. Past studies have used various modeling approaches to estimate PM_{2.5}-based BPTs in the US. Fann *et al* (2012) and Holt *et al* (2015) found, using various applications of atmospheric CTMs, that such BPTs, or related sensitivities, increase from baseline to abatement scenarios. These findings do not exclusively apply to NO_x, and may extend to abatement of SO₂ and even primary PM emissions. Such findings may be



due to a nonlinear atmospheric response of PM to emitted precursors through aerosol thermodynamics and aqueous chemistry. With recent evidence that a supralinear CRF may be the most fitting model choice for PM_{2.5} (Pope *et al* 2009, Pope *et al* 2011, Crouse *et al* 2015), added nonlinearity in health benefits with abatement is expected (Goodkind *et al* 2014, Apte *et al* 2015, Pope *et al* 2015). Our findings for NO₂ and O₃ can therefore be cast in the light of compounding benefits of NO_x control that may remain, and even be amplified, with inclusion of PM_{2.5} (Pappin *et al* 2015).

4. Conclusions

Our results are affected by a number of uncertainties introduced when integrating epidemiological risk estimates, population and mortality data, and monetary valuation metrics with atmospheric CTMs. Our results are affected by uncertainties in risk estimates for various forms of CRFs. Changes in population characteristics and mortality rates from 2007 are not captured here and may affect BPT estimates into the future, particularly as pollution levels decline with continued abatement. We apply a uniform and constant value of a statistical life to mortality in

Canada, while recognizing that it may vary spatially and temporally as pollution levels decline. Uncertainty in emission inventories and in modeling complex atmospheric processes in CTMs introduces uncertainty into BPT estimates. Interpretation of our findings should consider these limitations and uncertainties of our analysis.

In estimating BPTs, we assume a causal relationship between O₃ and NO₂ exposure and mortality in the long-term. Our estimates of BPTs should be interpreted with a forward-looking lens and within the context of long-term public health benefits in Canada gained from emissions abatement. As exposure levels over long periods, rather than short periods, are most relevant to chronic health endpoints, the estimated benefits may take time to compound in the population. Further, our estimates are based on a reference year of 2007. Variability in BPTs is expected from year-to-year with changing emissions and meteorological conditions. We note that our BPT estimates for 2007 are not necessarily reflective of present-day BPTs due to the widespread and aggressive emissions abatement that has taken place since (National Emissions Inventory 2015). Our findings of rising BPTs with more aggressive emissions control suggest that

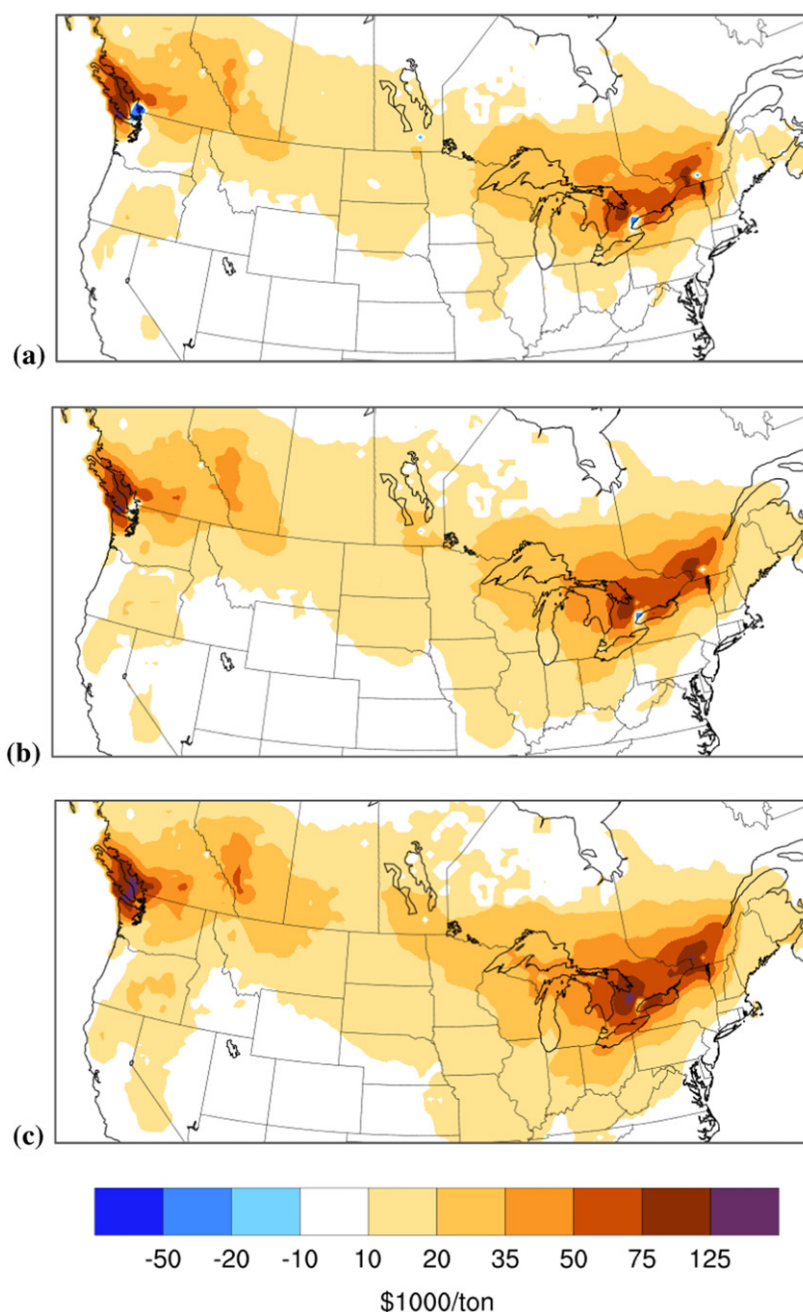


Figure 6. BPTs of NO_x control based on a linear in concentration CRF for O_3 ($\beta = 0.0026$) from the two-pollutant model. BPTs are estimated at (a) 2007 emission levels and for (b) 25% and (c) 50% abatement scenarios.

BPTs for present-day emission levels are likely to exceed those for 2007.

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