Additional File 2. Augmented Concentration Details

We fit concentration distributions to the strongest secondary sources we could locate (from the year 2010 and in Ontario, if possible), including data from governmental websites and the peer-reviewed literature. The availability of environmental concentration data varied by carcinogen and environmental source. Data from monitoring campaigns or studies with larger sample sizes, robust sampling protocols, and Ontario-specific information were preferred.

Limit of detection

For environmental data, a common approach to treat concentration estimates below the limit of detection (LOD), also known as non-detects, is substitution (e.g., by replacing the non-detects by concentrations of 0, the LOD/2, the LOD/$\sqrt{2}$, or the LOD). Substituting values below the LOD is consistent with exposure assessment practices elsewhere (e.g., Health Canada, US Centers for Disease Control and Prevention) and was employed in our analyses.

We use the following guidelines when we are calculating summary parameters *from raw data*:

1. We ascertained the LOD of the provided data (inquiring about the LOD if it was not stated in the documentation accompanying the data).
2. If the entity providing the data (i.e., the data steward) reported a result, we used it, even if the result was below the stated LOD.
3. If the entity providing the data reported a result as "<LOD" or "<DL" or "ND" or "<MRL", we substituted this value with the LOD/$\sqrt{2}$ (approximately 0.7071×LOD).

Where we obtained concentration information from journal articles, we ascertained how the authors treated samples below the LOD based on information provided in the manuscript and supplementary material (if applicable).

The LOD levels are presented in Table 1

Table 1. Limit of detection (LOD) information by carcinogen and environmental source

Carcinogen	Environmental Source	LOD	Units	Sample Size	Percent reported (%)^	Percent below LOD (%)^
Combustion by-products						
Outdoor air pollution ($PM_{2.5}$)	Outdoor Air	NR	µg/m^3	12122	100%	
2,3,7,8-Tetrachlorodibenzo-para-dioxin (TCDD)	Outdoor Air	NR	pg of TEQ/m^3	79	100%	
	Food and Beverages[x]	NR	pg of TEQ/kg-day			
Polycyclic Aromatic Hydrocarbons (PAHs)	Outdoor Air	NR	ng/m^3	198	100%	0%
	Indoor Air					
	Indoor Dust	0.051	µg/g		100%	
	Drinking Water	1	ng/L	68	100%	
Metals and metalloids						
Arsenic	Outdoor Air	0.02-0.06	ng/m^3	384	100%	2%
	Indoor Air	NR	ng/m^3	502	99%	1%
	Indoor Dust	0.1	µg/g	1025	100%	
	Drinking Water	1	µg/L	277	100%	92%
Cadmium	Outdoor Air	0.02-0.06	ng/m^3	384	100%	6%
	Indoor Air	NR	ng/m^3	502	74%	26%
	Indoor Dust	0.1	µg/g	1025	100%	
	Drinking Water	0.5	µg/L	277	100%	100%
Chromium (VI)	Outdoor Air	0.19-0.34	ng/m^3	384	100%	21%
	Indoor Air	NR	ng/m^3	502	57%	43%
	Indoor Dust	0.5	µg/g	1025	100%	
	Drinking Water	5	µg/L	277	100%	100%
Nickel	Outdoor Air	0.09-0.24	ng/m^3	384	100%	21%
	Indoor Air	NR	ng/m^3	502	48%	52%
	Indoor Dust	0.5	µg/g	1025	100%	
	Drinking Water	2	µg/L	277	100%	95%
Volatile organic compounds (VOCs)						
1,2-Dichloropropane	Outdoor Air	NR	ng/m^3	661	100%	100%
	Indoor Air	0.02	ng/m^3	3857	4%	96%
	Drinking Water	0.05	µg/L	342	100%	100%
1,3-Butadiene	Outdoor Air	NR	ng/m^3	1076	100%	100%
	Indoor Air	0.043-0.055	ng/m^3	884	100%	7%
Alpha-	Outdoor Air	NR	ng/m^3	283	100%	0%

Carcinogen	Environmental Source	LOD	Units	Sample Size	Percent reported (%)^	Percent below LOD (%)^
chlorinated toluenes	Indoor Air	0.018-0.050	ng/m^3	845	100%	97%
Benzene	Outdoor Air	NR	µg/m^3	1174	100%	0%
	Indoor Air	0.07	µg/m^3	3857	100%	0%
	Drinking Water	0.05	µg/L	342	100%	100%
Dichloromethane	Outdoor Air	NR	µg/m^3	1122	100%	0%
	Indoor Air	0.081-0.089	µg/m^3	884	100%	0%
	Drinking Water	0.2	µg/L	342	100%	100%
Formaldehyde	Outdoor Air	NR	µg/m^3	164	100%	0%
	Indoor Air	NR	µg/m^3	215	100%	
Tetrachloroethylene (PCE)	Outdoor Air	NR	µg/m^3	1174	100%	0%
	Indoor Air	0.01	µg/m^3	3857	99%	1%
	Drinking Water	0.05	µg/L	342	100%	0%
Trichloroethylene (TCE)	Outdoor Air	NR	µg/m^3	1161	100%	0%
	Indoor Air	0.01	µg/m^3	3857	75%	25%
	Drinking Water	0.05	µg/L	342	100%	0%
Vinyl chloride (chloroethene)	Outdoor Air	NR	µg/m^3	844	100%	0%
	Indoor Air	0.110-0.115	µg/m^3	884	100%	
	Drinking Water	0.05	µg/L	342	100%	100%
Other						
Acrylamide	Food and Beverages	10	µg/kg of food			
Asbestos	Outdoor Air	0	f/mL	1678	100%	
	Indoor Air	0	f/mL	3979	100%	
Polychlorinated biphenyls (PCBs)	Outdoor Air	NR	pg TEQ/m^3	78	100%	
	Indoor Air	NR	pg ΣPCB/m^3	10	100%	
	Indoor Dust	NR	ng ΣPCB/g	10	100%	

LOD: limit of detection; NR: not reported.

Note: Except for acrylamide, no LOD information was provided for the food intakes.

^The percent reported refers to the fraction of samples where a value was provided by the data steward. Percent below limit of detection (LOD) refers to the percent of samples that were below the stated LOD. In our analysis, we used all values provided by the data steward (even if they were below the stated LOD). When the data steward listed a value as below the LOD, we performed substitution

[x] Except for acrylamide, no LOD information was provided for the food intakes.

Food intake details

The food intakes (in units of µg/kg-d) were obtained from the Total Diet Study as mean values for approximately 11 age bins. When male and female mean intakes were provided separately for each age bin, we averaged them. In an effort to attempt to characterize variability associated with the food intake estimates, we used the spread of measures from the ten age bins, noting this will underestimate true variability. For PAH, we obtained an estimate of intake (in ng/d) converted it to intake units of ng/kg-d by dividing by bodyweight.

Table 2. Food intake discrete probability distributions for dioxin, acrylamide, arsenic, PCBs, and PAHs

2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD)		Acrylamide		Arsenic		Polychlorinated biphenyls (PCBs)		Polycyclic aromatic hydrocarbons (PAHs)	
Exposure (pg of TEQ/kg-day)	Probability	Exposure (µg/kg-day)	Probability	Exposure (µg/kg-day)	Probability	Exposure (ng/kg-day)	Probability	Exposure (ng/d)	Probability
0.440	0.188	0.157	0.113	0.365	0.188	1.625	0.188	10.000	0.030
0.535	0.313	0.187	0.250	0.420	0.002	1.950	0.313	30.050	0.265
0.710	0.250	0.211	0.013	0.440	0.002	2.545	0.250	50.050	0.310
0.890	0.100	0.248	0.250	0.490	0.003	2.920	0.100	70.050	0.265
1.520	0.088	0.288	0.150	0.530	0.250	4.820	0.088	90.050	0.080
1.880	0.002	0.356	0.063	0.545	0.100	5.180	0.002	110.050	0.040
1.930	0.002	0.442	0.063	0.575	0.313	5.240	0.002	130.050	0.000
2.100	0.003	0.597	0.063	0.630	0.003	5.500	0.003	150.050	0.010
2.390	0.050	0.609	0.038	0.830	0.050	7.160	0.003		
2.450	0.002			0.940	0.088	7.410	0.050		
2.710	0.003			2.920	0.002	7.940	0.002		

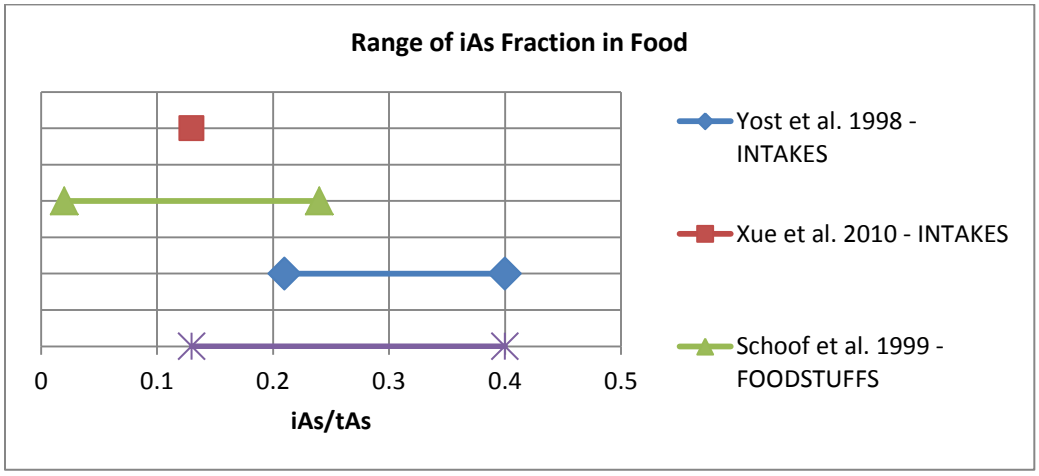
Carcinogen-specific information

We compiled additional information related to the concentration estimation for several of the carcinogens, as listed below.

Arsenic

For food ingestion, we model the fraction of food that is inorganic As versus organic by a uniform distribution, with range of 0.13 and 0.40 based on three studies (Schoof et al., 1999; Xue et al., 2010; Yost et al., 1998) summarized in Figure 1.

Figure 1. Range of arsenic in food that is inorganic



Chromium

For Cr, we applied the fraction that is carcinogenic – that is the Cr(VI) fraction. We modelled this using a uniform distribution with a range of 0.1 to 0.5. We developed this range by evaluating information from a number of studies (Bell and Hipfner, 1997; Government of Canada, Health Canada, Environment Canada, 1994; Krystek and Ritsema, 2007; Mentze et al., 2004; Swietlik et al., 2011; Talebi, 2003; World Health Organization, 2003).

Cadmium

While Cal EPA OEHHA lists a slope factor for cadmium ingestion, it is based on the inhalation unit risk. The WHO and USEPA do not classify cadmium as a carcinogen by the ingestion route of exposure. While IARC does not make a determination on the route of exposure, their association of cadmium with lung and prostate cancer is based on occupational studies where the primary route of exposure is inhalation. For this study, we estimated the cadmium cancer burden by inhalation and not ingestion.

Diesel PM

No exposure estimates for diesel PM exist for Ontario. Such estimates are difficult to obtain for any region because of the measurement and modeling challenges. Instead, we derived a diesel PM distribution using data from a California Air Resources Board (CARB) report on identifying diesel exhaust as a toxic air contaminant (California Environmental Protection Agency Air Resources Board, 1998). For the year 1990, the CARB report estimated a statewide California population-weighted diesel PM_{10} concentration of 3.0 $\mu g/m^3$, and projected this to decline to 1.7 $\mu g/m^3$ by the year 2010 (see Figure V-2 from CARB report). In other words, the 2010 diesel PM_{10} level was 0.5667 times the 1990 level. In Table V-2 of the same CARB report, there are 1990 diesel PM_{10} levels for 15 counties in California. We effectively converted these 1990 diesel PM_{10} estimates to 2010 estimates by applying the 0.5667 factor. Next, we applied a diesel $PM_{2.5}/PM_{10}$ ratio of 0.94 (taken from another CARB report on the same issue, "The Report on Diesel Exhaust, Findings of the Scientific Review Panel On The Report on Diesel Exhaust") to effectively convert the diesel PM_{10} estimates to diesel $PM_{2.5}$. Finally, we estimated an outdoor 2010 diesel $PM_{2.5}$ GM of 0.68 $\mu g/m^3$ (GSD of 2.35 $\mu g/m^3$) using the information above as applied to the 15 counties (Table 3).

Another approach to estimating the diesel PM levels would be to determine the fraction of $PM_{2.5}$ levels in Ontario that are of diesel exhaust origin. We did not locate any published values for this for Ontario. However, for six air basins in California, we have the 2010 diesel $PM_{2.5}$ estimates, along with monitored values for $PM_{2.5}$. For these six air basins, the fraction of $PM_{2.5}$ that was diesel ranged from 8% to 14%, with a mean value of 11% (median 11%; see Table 3). Applying 11% to the mean $PM_{2.5}$ level in Ontario (5.7 $\mu g/m^3$) gives an estimate of 0.57 $\mu g/m^3$, which is close to the modeled mean we employed of 0.67 $\mu g/m^3$. CAREX Canada has previously applied an estimate of 12% of $PM_{2.5}$ that is diesel $PM_{2.5}$, which is in line with our calculated estimate of 11%.

Furthermore, since the California Air Resources Board (CARB) report also stated that indoor levels of diesel are ⅔ of outdoor levels, we calculated an indoor level of 0.46 $\mu g/m^3$ (GSD of 2.35 $\mu g/m^3$) for the RA model.

Table 3. Lognormal 2010 diesel concentration distribution, fit to levels from 15 air basins

Air Basin	Estimated Outdoor Diesel $PM_{2.5}$ ($\mu g/m^3$)	Measured Outdoor $PM_{2.5}$ ($\mu g/m^3$)	Diesel fraction (%)
Great Basin Valley	0.11		
Lake County	0.16		
Lake Tahoe	0.53		
Mojave Desert	0.43		
Mountain Counties	0.32		
North Central Coast	0.75		
North Coast	0.64		
Northeast Plateau	0.59		
Sacramento Valley	1.33	10.9	12%
Salton Sea	1.38		

San Diego	1.54	10.8	14%
San Francisco Bay Area	1.33	10.6	13%
San Joaquin Valley	1.38	17.1	8%
South Central Coast	0.96	10.0	10%
South Coast	1.92	17.9	11%
GM ($\mu g/m^3$)	0.68	AM	11%
GSD ($\mu g/m^3$)	2.35	Range	8% - 14%

AM: arithmetic mean; GM: geometric mean; GSD: geometric standard deviation

Nickel

While we were able to calculate exposure concentrations from Ni in food, drinking water, and dust, there was no existing OSF. As such, we were unable to estimate the cancer burden by Ni ingestion. (We do estimate the cancer burden by Ni inhalation.)

PAHs

PAHs represent a class of compounds. We used benzo[a]pyrene as a surrogate for total PAH exposure. While there are many other PAHs, benzo[a]pyrene is the most toxic. Health Canada, US EPA, and Cal EPA developed OSF and IUR estimates for benzo[a]pyrene, which we applied in our analysis.

PCBs

PCBs are a class of compounds, consisting of many different congeners. There are two ways to treat this class of compound. One is to sum the individual PCB measurements and apply the PCB slope factor to this sum. Another is to weight the PCBs by their toxicities, summing the weighted values to obtain a toxic equivalency, or TEQ, then apply the dioxin slope factor to the TEQ. We applied the TEQ approach in our analysis.

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