Rethinking Environmental Performance from a Public Health Perspective

A Comparative Industry Analysis

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Keywords

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Summary

To date the most common measures of environmental performance used to compare industries, and by extension firms or facilities, have been quantity of pollution emitted or hazardous waste generated. Discharge information, however, does not necessarily capture potential health effects. We propose an alternative environmental performance measure that includes the public health risks of toxic air emissions extended to industry supply chains using economic input-output life-cycle assessment. Cancer risk to the U.S. population was determined by applying a damage function to the Toxic Release Inventory (TRI) as modeled by CalTOX, a multimedia multipathway fate and exposure model. Risks were then translated into social costs using cancer willingness to pay. For a baseline emissions year of 1998, 260 excess cancer cases were calculated for 116 TRI chemicals, dominated by ingestion risk from polycyclic aromatic compounds and dioxins emitted by the primary aluminum and cement industries, respectively. The direct emissions of a small number of industry sectors account for most of the U.S. population cancer risk. For the majority of industry sectors, however, cancer risk per $1 million output is associated with supply chain upstream emissions. Ranking industries by total (direct + upstream) supply chain risk per economic output leads to different conclusions about the relative hazards associated with these industries than a conventional ranking based on emissions per economic output.

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Introduction

Over the past decade interest in measuring and monitoring private sector environmental performance has grown, particularly among third-party nongovernmental entities. In the United States most industries disclose levels of physical emissions from their facilities to the public for regulatory compliance purposes or in voluntary corporate reports (e.g., the U.S. Environmental Protection Agency's Toxic Release Inventory [U.S. EPA's TRI] or the global reporting initiative [GRI 2002]). (Refer to Appendix A for a complete list of commonly used acronyms in this article.) The TRI emissions database, covering approximately 650 toxic chemicals and chemical groups reported by U.S. manufacturing facilities (USEPA 2000d), is widely used to evaluate the environmental performance of firms and industries and the public health risks posed by facilities, and in social science research. As an alternative measure, the World Business Council for Sustainable Development has been promoting "eco-efficiency," which measures resource use or environmental effect relative to output across a product's life-cycle (Schaltegger and Sturm 1990; Schmidheiny 1992; DeSimone and Popoff 1997). What is lacking to date is a comprehensive measure that recognizes the interaction between industrial activity, ensuing emissions, and health impacts across industry supply chains. An environmental performance measure that can be financially quantified is helpful in many private and public sector decision-making settings, which rely on a weighting of the costs versus benefits of potential interventions on behalf of the environment. The systemic view of life-cycle assessment, in addition to techniques developed to financially valuate health impacts, presents useful methods to fill this gap.

In this article we propose a new environmental performance measure based on the public health risk per dollar unit of output from industries and their supply chains. Additionally, we provide an estimate of social cost associated with pollution that moves beyond prior estimates of the private costs of regulatory compliance, such as investments in pollution control technology and the associated managerial overhead (Ditz et al. 1995; Repetto and Austin 2000), property risks (Austin and Sauer 2002; Innovest 2003), or legal liability risks (ASTM 2003). Our goals are three fold: (i) to assess the U.S. population cancer risk associated with industrial toxic chemical air emissions drawn from the 1998 TRI over individual industry sector supply chains with economic input-output life-cycle assessment (EIO-LCA); (ii) to monetize these risks; and (iii) to compare industry rankings using a measure of aggregate cancer risk per unit of economic benefit. Our approach uses methods published in the peer-reviewed literature and publicly available data. Human exposure to each chemical in our sample is estimated with CalTOX, a multimedia multipathway fate and exposure model, and is denoted by the individual intake fraction, a ratio of potential human dose to total exposure relative to an emissions source (Bennett et al. 2001, 2002). Chemical-specific cancer potency factors are applied to intake fractions to derive cancer risk associated with TRI emissions. This analysis includes only carcinogenic effects of chemicals with information available on linear dose-response relationships (i.e., cancer potency). We use cancer willingness to pay in order to estimate social costs. Finally, we explore various sources of uncertainty.

Emissions Data Set

The emissions data set is drawn from the 1998 TRI (a mandatory self-reported EPA program), which includes releases of toxic chemicals to air, surface water, on-site land, and underground injection. The data are collected from facilities and can readily be evaluated at the facility or at the 4-digit Standard Industry Classification (SIC) industry sector level. Our sample covers emissions from 4,760 facilities in 420 4-digit SIC industry sectors drawn from the TRI database. In some cases, a single facility produces products that fall under multiple SIC codes, in which case emissions are divided equally among the sectors.[1] We focus on air emissions, because they are a major portion of TRI releases, compared to releases to water or soil. Furthermore, releases to water or soil require site-specific modeling, whereas air emissions, which can potentially travel farther from the release site, can be modeled on a non-site-specific basis. For this type of industry-level analysis, air emissions risks are less influenced by

aggregated large spatial analysis than risks due to water or soil discharges. Reported TRI stack and fugitive (escaping randomly from the production process) air emissions are combined.

Over the years the list of TRI chemicals has grown to comprise over 600 chemicals and chemical categories with various health and environmental impacts. We focus on air emissions for a subset of 116 designated carcinogenic TRI compounds and two chemical categories, polycyclic aromatic compounds and dioxins, for which cancer potency factors are available to permit an estimate of the number of associated cancer cases (see the table in the e-supplement at the *Journal*'s Web site). Although 1998 is selected as the baseline year (to match the most recent 1998 economic data), we include dioxin air emissions in 2000, the first year dioxin emissions were reported. We assume that dioxin emissions levels for 2000 are representative of those for 1998.

Measurement error in the TRI arouses general concern. Facilities rely primarily on published emissions factors or mass balance equations rather than measured data, which can result in large differences in reported releases from comparable facilities. According to a study by Williams and colleagues (2002), some industry sectors (e.g., the semiconductor industry) appear to underreport their TRI emissions. For our 1998 chemical sample over 50% of submitted records provide no indication of estimation method at all. In addition, changes on paper, such as redefinition of release characterization, can explain a major portion of year-to-year emissions reductions (Natan and Miller 1998). Such underreporting will introduce a downward bias into industry emissions coefficients in our analysis.[2]

Health Impacts

U.S. population cancer risk was estimated using cancer potency factors applied to calculated exposure for each compound. Mathematically, human cancer risk C is a function of emissions *S* (in milligrams per day [mg/day]), individual intake fraction (iIF), cancer potency factor (CPF) in $(\text{mg/kg-day})^{-1}$ for each chemical, the population exposed (Pop; United States: 280 million), multiplied by the ratio of the exposure duration ED in years (yr) to the 70-yr biological averaging time AT (yr), and divided by a 70-kg average body weight BW as follows:

$$C = \text{iIF} \times \text{CPF}_{(\text{kg-d/mg})} \times S_{(\text{mg/d})} \times \text{Pop} \times \text{ED/AT} \times 1/\text{BW}_{(\text{kg})} \quad (1)$$

For this analysis an industry's public health impact is the sum of estimated annual excess cancers associated with a single year of exposure (ED = 1998) to TRI chemicals emitted by that industry.

Prior research using the EIO-LCA methodology to estimate U.S. population toxic risks associated with TRI emissions has been based on permissible worker inhalation exposure, that is, threshold limit values (TLVs) (Horvath et al. 1995; Joshi 2000).[3] But this approach does not account for the movement of compounds through various environmental media with which a human can have contact. Some compounds are lipophilic and bioaccumulate, resulting in ingestion risks that are greater than inhalation risk, the basis of TLVs. In fact, the American Conference of Governmental Industrial Hygienists (ACGIH) does not recommend using TLVs for population risk assessment of continuous uninterrupted exposures (ACGIH 2003).

Multimedia fate and transport models have been proposed to estimate exposure to toxic chemicals (Grimstead et al. 1994; Jia and Guardo 1996). Hertwich and colleagues (1998) argue that CalTOX provides the best available method for estimating toxic human effects of TRI in LCA, and has been used to estimate human toxicity potentials (HTPs) (Hertwich 1999; Hertwich et al. 2001) as well as in the EPA's Tool for the Reduction and Assessment of Chemical Impacts (TRACI) (Bare et al. 2002). CalTOX is a physical-stochastic multimedia model (Mackay 1991; McKone 1993; Cowan et al. 1994) developed to assess time-varying concentrations of contaminants released to air, soil or water. It consists of two component models: (i) a multimedia fate and transport model based on both conservation of mass and chemical equilibrium determined using first-order transformations, and (ii) a multipathway human exposure model that provides estimates of ingestion intake, inhalation intake, and dermal uptake based on 23 exposure pathways. CalTOX yields intake fractions

for a specific chemical, which are a function of contaminant concentrations in the multimedia model compartment and rates of human contact (e.g., personal air, tap water, foods, household dusts, and soils) for a given population estimated assuming steady-state multimedia dispersion conditions and continuous emissions.[4]

Various other methods for weighting the TRI have been proposed in analyses not specific to LCA. These have been summarized by Toffel and Marshall (2004), who recommend using either TRACI-HTPs based on CalTOX or the U.S. EPA's Risk Screening Environmental Indicators (RSEI). We select CalTOX as our model to estimate cancer risk, because CalTOX is a true multimedia model and includes exposure through ingestion of produce and other agricultural products omitted in RSEI. These pathways are central to our risk assessment results. We do not adopt HTPs, which are simply intake fractions multiplied by the cancer potency of each compound relative to the same product for a reference compound (benzene for cancer risks and toluene for noncancer risks). Instead, we calculate number of cancer cases based on intake fractions, per equation (1), because this allows us to apply a financial valuation function to each cancer case. Intake fractions for the greater part of our TRI sample are based on prior estimates (Bennett et al. 2002).

This risk assessment is complicated by mixtures with varying cancer potency such as polycyclic aromatic compounds (PACs) and dioxins.[5] Commonly, compounds are grouped according to physical-chemical properties and toxicological potential, and individual chemical toxicity is denoted in toxic equivalency factors (TEFs) or potency equivalency factors (PEFs) relative to the most studied compound.[6] For dioxins this is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) and it is benzo(a)pyrene (BaP) for PACs. Use of equivalency factors is based upon the general finding that compounds within a group are thought to cause cancer by similar mechanisms. Thus, PACs interact with DNA and dioxins interact with the Ah (Aryl hydrocarbon) receptor (Birnbaum 1999; Warshawsky 1999). For PACs the additivity (i.e., no-interaction) assumption for toxic effects, critical for summing human toxicity over several hundreds of compounds in LCA, appears reasonable (Warshawsky 1999; Woodruff et al. 2000).

TRI PACs are a subset of the polycyclic aromatic hydrocarbon (PAH)-combustion-related chemical class. PAHs constitute the main component of polycyclic organic matter (POM), a hazardous air pollutant (HAP) listed in the 1990 Clean Air Act Amendments. In this article the PAC category refers specifically to the 21 PAH compounds listed in the TRI (table 1). Additional PAHs (anthracene, benzo(g,h,i,)perylene, naphthalene, and phenanthrene) that are considered International Agency for Research on Cancer (IARC) category 3 noncarcinogenic chemicals are listed individually in the TRI. Although our emphasis is on cancer risk estimation, we include these four to capture the entire PAH category in the TRI database.

In 1998 over 650,000 kg of TRI PAC was released into the air from industry sources. The contribution from individual PAC compounds needs to be estimated for risk assessment, because different constituents have varying levels of intake fraction and toxicity. Prior U.S. population risk assessments assumed (i) that high-potency compounds dominate stationary and mobile emissions, yielding a POM potency equivalent to 16% BaP, or (ii) that all compounds contribute equally, yielding a 6% BaP POM equivalency (Caldwell 1998; Caldwell et al. 1998; Woodruff et al. 2000). In contrast to these risk assessments, our focus is only on stationary sources reporting to the TRI. Therefore, we adopt a compound-specific PAC emissions distribution specific to major stationary sources and TRI SIC codes as available in the 1999 National Toxics Inventory (NTI) (table 1).

TRI PAC emissions are weighted by the most recent PEFs (OEHHA 1994; Collins et al. 1998), which allow several compounds to be more potent than BaP. Additionally, we use compound-specific CPFs determined under the California EPA expedited risk assessment process. Collins and colleagues argue for using PEFs only for chemicals definitively classified as carcinogens. Several TRI PAHs are not classified as carcinogens (table 1); but rather than exclude them, we have elected to follow the research community norm of treating PAHs as a group. We adjust for their inclusion by assigning a low 0.001 PEF

to the compounds not classified as carcinogens (Nisbet and LaGoy 1992).

Although it is preferable to apply PEFs specific to an exposure pathway for individual PACs (Schneider et al. 2002), this information is still lacking. We thus apply the same PEF to BaP potency for all exposure routes, but, we adopt an oral BaP CPF of 11.5 $(mg/kg\text{-}day)^{-1}$ and 3.9 $(mg/kg\text{-}day)^{-1}$ for inhalation risk for all PACs (CalEPA 2002), and weight these by percent of intake arising from each exposure pathway for each individual PAC, as determined in CalTOX.

The TRI dioxin category comprises 17 individual chemicals, commonly termed "congeners." For these the EPA requires facilities to report total media specific dioxin emissions (in grams [g]) and to break down these emissions by percent for each of the 17 congeners. But 30% of the 5218 grams of dioxin air emissions reported in 2000 were not speciated in such a manner. For these we either apply (i) industry averages where some facilities in that sector provide speciation data or (ii) the average congener distribution based on reported percentages for all dioxin data submitted to the EPA (table 1). Dioxin TEFs are based on the TEQ-WHO98 scheme (where TEQ stands for toxic equivalents and WHO stands for World Health Organization) (Berg et al. 1998) and are employed in the EPA September 2000 draft Dioxin Reassessment (hereinafter "EPA Reassessment").[7] We apply the most recent estimate for TCDD toxicity derived by the EPA, 1×10^6 per mg TCDD/kg-day, for both inhalation and ingestion pathways.

Intake fractions (IF) were estimated with CalTOX for all 42 PAH and dioxin congeners using physical-chemical properties obtained from the EPA Reassessment, the handbook by Mackay and colleagues (1992), and the Syracuse Research Corporation (SRC 2003). For several PAH compounds medium-specific half-lives and physical-chemical properties were derived based on molecular weight. For dioxins congener-group averages were assumed when specific data on physical-chemical properties were lacking.

To gain confidence in the model, estimated dioxin IFs were applied to 1995 dioxin emissions derived for the EPA Reassessment to yield a daily dioxin dose of 39.7 TEQ-WHO98 picograms/day (pg/dy) dominated by ingestion exposure (99.8%).[8] This daily dose estimate compares nicely with the EPA's estimated daily dose of 40.3 TEQ-WHO98 pg/day using measured food dioxin concentrations and human daily contact rates. The EPA Reassessment similarly finds that ingestion risk dominates human exposure to dioxin (97.5%).[9]

Cost of Cancer

With the growing importance of balancing costs and benefits in regulatory rulemaking, mandated under Executive Order 12291 (amended in Executive Order 12866), the U.S. EPA has invested considerable effort in assessing the social cost of pollution, including the cost of cancer. Other policy approaches include deriving national accounts based on a measure of welfare or net social impact (Jorgenson et al. 2005). A similar, though less quantitative, evaluation is common in many corporate decisions on how to allocate resources to an environmental strategy. Given these parallel developments, we assess the cost of cancer associated with our TRI sample emissions. The cost of cancer comprises a morbidity and a mortality component and varies by tumor site and latency period. We conduct a status quo estimate of social costs used in LCA practice (Hellweg et al. 2003) based on the EPA Cancer White Paper (USEPA 2000b), the corresponding comments of the EPA Science Advisory Board Environmental Economics Committee, EPA-SAB-EEAC-00-013 (SAB-EEAC 2000), and the EPA Arsenic Rule Benefits Analysis (USEPA 2000c).

The social costs of disease include direct costs of illness (COI) (e.g., physician time, hospitals, and drugs), indirect costs (cost of lost production and wages forfeited), and additional intangible costs (patient and family pain, grief, and suffering) (Torrance 1986). A controversial approach is to elicit revealed or stated preferences for small changes in health using a measure of willingness to pay (WTP). The central value of aggregate revealed (or stated) WTP is termed the value of a statistical life (VSL), and is defined as the marginal rate of substitution between the probability of dying and the utility of wealth conditional on surviving or dying (i.e., the value of risk reduction) in a specific time period (Hammitt

Table 1 Polycyclic aromatic compounds and dioxin emissions characteristics

Polycyclic aromatic compounds	*CAS number*[1]	*PEF*[2]	*IF(tot,air)*[3]	*NTI air emission (in %)*[4]	*Lifetime cancer risk*[5]
7,12-Dimethylbenz[a]anthracene	57-97-6	21.8	6.3E-05		
Dibenzo[a,h]pyrene	189-64-0	10	2.9E-04		
Dibenzo[a,i]pyrene	189-55-9	10	2.9E-04		
Dibenzo[a,l]pyrene	191-30-0	10	2.9E-04		
3-Methylcholanthrene	56-49-5	1.9	3.8E-05		
5-Methylchrysene	3697-24-3	1	1.6E-05		
7H-Dibenzo(c,g)carbazole	194-59-2	1	5.2E-05		
Benzo[a]pyrene	50-32-8	1	1.7E-04	0.066	3,521
Dibenzo[a,e]pyrene	192-65-4	1	2.9E-04		
Dibenzo[a,h]anthracene	53-70-3	0.4	1.6E-04	0.004	83
1-Nitropyrene	5522-43-0	0.1	4.8E-05		
Benzo[a]anthracene	56-55-3	0.1	2.0E-05	0.003	2
Benzo[b]fluoranthene	205-99-2	0.1	9.0E-05	0.003	7
Benzo[j]fluoranthene	205-82-3	0.1	8.1E-05		
Benzo[k]fluoranthene	207-08-9	0.1	3.0E-05	0.003	2
Dibenzo[a,h]acridine	226-36-8	0.1	4.0E-05		
Dibenzo[a,j]acridine	224-42-0	0.1	5.3E-05		
Indeno[1,2,3-c,d]pyrene	193-39-5	0.1	2.3E-04	0.004	30
Chrysene	218-01-9	0.01	2.5E-05	0.019	1
Fluoranthene	206-44-0	0.001	3.1E-06	0.898	0.8
Dibenzo(a,e)fluoranthene	5385-75-1				
Total				**1.00**	**3,647**
				(*in kg/yr*[6])	
Anthracene	120-12-7	0.001	1.8E-07	2,500	1.28E-04
Benzo[g,h,i,]Perylene	191-24-2	0.001	1.9E-04		
Naphthalene	91-20-3	0.001	5.3E-07	599,937	4.89E-02
Phenanthrene	85-01-8	0.001	1.3E-06	49,221	1.79E-02

Table I Continued

Dioxins	*CAS number*[1]	*TEQ-WHO98*[7]	*IF (tot, air)*[3]	*TRI air emission (in %)*	*Lifetime cancer risk*[5]
1,2,3,7,8-Pentachlorodibenzo-p-dioxin	40321-76-4	1	1.20E-03	0.012	2,885
2,3,7,8-Tetrachlorodibenzo-p-dioxin	1746-01-6	1	1.07E-03	0.007	1,584
2,3,4,7,8-Pentachlorodibenzofuran	57117-31-4	0.5	9.45E-04	0.028	2,689
2,3,7,8-Tetrachlorodibenzofuran	51207-31-9	0.1	3.06E-04	0.051	318
1,2,3,4,7,8-Hexachlorodibenzofuran	70648-26-9	0.1	1.92E-03	0.035	1,385
2,3,4,6,7,8-Hexachlorodibenzofuran	60851-34-5	0.1	1.94E-03	0.022	861
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	39227-28-6	0.1	2.10E-03	0.015	662
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	57653-85-7	0.1	2.24E-03	0.015	667
1,2,3,6,7,8-Hexachlorodibenzofuran	57117-44-9	0.1	1.95E-03	0.017	670
1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	19408-74-3	0.1	2.23E-03	0.012	563
1,2,3,7,8,9-Hexachlorodibenzofuran	72918-21-9	0.1	1.94E-03	0.007	275
1,2,3,7,8-Pentachlorodibenzofuran	57117-41-6	0.05	1.44E-03	0.021	305
1,2,3,4,6,7,8-Heptachlorodibenzofuran	67562-39-4	0.01	2.27E-03	0.097	449
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	35822-46-9	0.01	2.06E-03	0.067	284
1,2,3,4,7,8,9-Heptachorodibenzofuran	55673-89-7	0.01	2.29E-03	0.027	127
1,2,3,4,6,7,8,9-Octachlorodibenzofuran	39001-02-0	0.0001	1.72E-03	0.348	12
1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	3268-87-9	0.0001	1.85E-03	0.218	8
Total				**1.000**	**13,744**

[1]CAS Number refers to the Chemical Abstracts Service registry number for the substance.
[2]PEF is potency equivalency factor.
[3]U.S. population intake fraction (IF) for air emissions, assuming 280 million inhabitants. Intake fractions are calculated using CalTox PEF's taken from OEHHA (1994), Collins and colleagues (1998), and Nisbet and LaGoy (1992).
[4]Based on National Toxics Inventory (NTI) stationary point source air emissions, for TRI SICs (Toxic Release Inventory standard industry classification codes) only.
[5]US population *lifetime* cancer risk, calculated as CPF × IF(tot,air) × 1/BW × Emissions (mg/d), where CPF is cancer potency factor and assuming 70-yr lifetime and 70-kg body weight (BW); weighted by exposure pathway.
[6]PAH compounds reported separately to 1998 Toxic Release Inventory (TRI).
[7]TEQ-WHO98 values taken from Berg and colleagues (1998).
Note: One kilogram (kg, SI) ≈ 2.204 pounds (lbs).

2000). The most common sources of VSL are wage-risk assessments.

Scholars continue to debate the merits of adjusting such a wage-risk based VSL as applied to population risk assessment to reflect differences between populations in age, risk aversion, health status, and income (USEPA 2000a). As yet, the evidence is still considered too sparse to prompt such adjustments for cancer valuation (SAB-EEAC 2000). Thus, we employ the \$5.8 million wage-risk based VSL in 1997 U.S. dollars (hereafter dollars) (Viscusi 1993; USEPA 1997b, 1999, 2000a) or \$5.9 million adjusted for inflation to 1998 dollars with the Consumer Price Index.

At a minimum, nonfatal cancer WTP can be based on COI (Freeman 1993; USEPA 2000a). Scholars suggest that nonfatal cancer WTP should also include the costs of morbidity, such as foregone earnings, lost leisure time, preventive actions, and lifestyle changes (Tolley et al. 1994). Rowe and colleagues (1995) propose a weighted average WTP using COI for nonfatal cancers, adjusted by an arbitrary WTP/COI = 1.5 ratio, and VSL for fatal cancers.[10] Alternatively, the EPA (USEPA 2000c) recommends a more appropriate adjustment to VSL based on a measure of utility lost due to cancer morbidity as determined by Magat and colleagues (1996). They find a 58.3% utility loss associated with the morbidity consequences of terminal lymphoma, and a 41.7% utility loss due to death some time in the future.[11] We use the utility loss determined by Magat and colleagues, which is combined with average 5-yr survival rates for all cancer sites (to reflect uncertainty with respect to cancer type associated with our sample chemical emissions) in the following estimating equation:

$$\begin{aligned}\text{Cancer WTP} = {} & \text{Survival rate} \times (\$5.9\text{M} \times 0.583) \\ & \text{(Nonfatal cancers)} \\ & + (1 - \text{survival rate}) \times \$5.9\text{M} \\ & \text{(Fatal cancers)}\end{aligned} \tag{2}$$

Using the 62.4% average 5-yr age-adjusted U.S. 1992–1999 cancer survival rate (Ries et al. 2002), we estimate a per cancer WTP of \$4.4 million in 1998 dollars.

Recent research suggests that cancer WTP decreases over latency period to reflect discounting of a future change in the probability of getting cancer and dying relative to an immediate reduction in mortality risk (Revesz 1999; Hammitt and Liu 2003). Whereas initial cost-benefit analyses assumed no latency period between exposure and disease (USEPA 1997b), scholars recommend discounting the value of future statistical cancer cases over the mid-point of the expected latency period at market interest rates (Horowitz and Carson 1990; Cropper et al. 1994; USEPA 1999; SAB-EEAC 2000). Lacking information on actual occurrence of estimated cancers we assume a uniformly distributed annual cancer incidence and discount at 5% over an average 10-yr cancer latency period (Russell et al. 1996; SAB-EEAC 2000; USEPA 2000a, 2000b).

Supply Chain Impacts

Economic input-output life-cycle assessment (EIO-LCA) is used to estimate an industry's supply chain environmental impact, and is recommended as a way to avoid the boundary problem plaguing traditional process LCA (Lave et al. 1995; Matthews and Small 2001). EIO-LCA is based on the work of Wassily Leontief, who described total industry output X as a linear function of the inputs a purchased from other industries per unit output and final demand Y. In simplified form, final demand is a function of total output minus intermediate input $X - aX = Y$, or $(I - A)X = Y$, where I is the identity matrix and $A \equiv [a_{nn}]$, the $n \times n$ matrix of technical coefficients for n sectors. Per the Leontief inverse, $X = (I - A)^{-1}Y$, the output X from each industry sector can be estimated as a function of any level of final demand Y in an economy. EIO-LCA augments this framework with the addition of R, a $k \times n$ matrix composed of k environmental burdens generated per dollar's worth of n industry outputs. For this analysis let R be a $1 \times n$ vector for n industry sector cancer cases. The total environmental burden generated by final demand Y_n is $\boldsymbol{EB} = [\boldsymbol{R}] * [\boldsymbol{I} - \boldsymbol{A}]^{-1}\mathbf{Y}$, where EB is a $k \times 1$ vector of total (direct + indirect) economy-wide environmental burden, $[I - A]$ is an $n \times n$ matrix, and Y is an $n \times n$ identity matrix of industry final demand (Miller and Blair 1985).[12]

Comparative ranking of industries requires that we normalize human health impact by a measure of economic size, such as total output or value added (total output minus cost of material inputs). As a measure of gross domestic product (GDP), value added may more appropriately represent the "functional unit" of U.S. production in LCA.[13] Value added refers to the cost of *nonindustrial* (i.e., nonmaterial) inputs to production, and is composed of (i) employee compensation, (ii) indirect business tax and nontax liability, and (iii) other value added (e.g., corporate profits and consumption of fixed capital) (Lawson 1997). Labor income is about 2/3 and capital income 1/3 of U.S. GDP (Campbell 2000).

We use 1998 production and consumption data from the U.S. national accounts compiled by the U.S. Bureau of Economic Analysis (BEA) for 490 industry sectors.[14] Matching the TRI dataset, based on SIC codes, with the BEA industry sectors reduces the number of industry sectors to 219, because several SICs are consolidated under one BEA code or these sectors did not emit significant amounts of our sample compounds.

Results

For this sample of TRI compounds we estimate 260 excess annual cancer cases for the U.S. population associated with 54,456,471 kg of 1998 air emissions, of which 96% of the risk arises from dioxin and PAC emissions with primary exposure through ingestion. Specifically, the 5.2 kg of dioxin emissions contribute 76%, and the 650,000 kg of PAC emissions 20% of total cancer risk, respectively. The 53,806,466 kg emissions of the remaining 114 TRI chemicals contribute only 4% of cancer risk, due primarily to intake fractions at least one order of magnitude lower than for PAC and dioxin (see table 1; see also Appendix B, in the electronic supplement to this article that is available on the *Journal's* Web site). In comparison, the total estimated cancer incidence (i.e., newly diagnosed cancer cases) in the United States was 1,228,600 in 1998 (ACS 1998).

Approximately 45% (219 sectors) of our industry sample reported no direct emissions of our chemical sample to the TRI in 1998. This may arise because the industry (i) did not emit any of our sample carcinogens, (ii) emitted them, but did not report to TRI as required by law, or (iii) is not required to report emissions. Of those reporting sectors, 23 (i.e., 10%) account for 80% of emissions and over 60 emit PACs and/or dioxins. Six industry sectors (Iron and Steel Foundries, Secondary Nonferrous Metals, Manmade Organic Fibers, Electric Services/Utilities, Primary Aluminum, Cement) out of 219 sectors account for approximately 80% of direct cancer risks, whereas they contribute a mere 2% of 1998 GDP. The estimated cost of all 260 cancer cases is $1.1 billion in 1998 dollars, if cancer incidence is assumed simultaneous with exposure. Assuming an average 10-yr latency period, the present value of social cost discounted at 5% is $702 million in 1998 dollars. In comparison, in 1998, private industries generated $7.68 trillion of GDP (Lum and Moyer 2001). The estimated number of cancers and their social costs could increase, though, if some of the compounds currently untested for carcinogenicity were found to be carcinogens.

The top 20 industries ranked by direct kg emissions of our limited chemical sample are shown in table 2 and compared with top five rankings using a public health risk-based measure of environmental performance. Six of the top ten materials-intensive industries (kg per $1 million output) have a low risk ranking. High PAC emissions underlie the high risk ranking of the primary aluminum industry, whereas high dioxin emissions account for cement industry cancer risk. The impacts are highly skewed, because these two industries each contribute over 25% of direct cancer risk/output for the entire U.S. economy. The cost of the cancer externalities associated with 1998 emissions per $1 million value added (VA) reaches a maximum of $1,300 per $1 million VA for the primary aluminum industry (shown in the final column of table 2).[15] Table 2 indicates that a strategy of risk reduction based on mass kg emissions would target the chemical and pulp and paper industries; industries targeted because of high TRI emissions levels in prior research (Hamilton 1995; Khanna et al. 1998). This approach would completely omit the cement and aluminum industries, main sources of cancer risk (adjusted for economic size).

EIO-LCA results show that risks are highly concentrated in a few industries. The median

Table 2 Top 20 industries ranked by direct Toxic Release Inventory (TRI) emissions (kg)[1]

Industry name	*BEA Ind*	*Emissions (kg)*[2]	*Rank*	*Annual cancers*	*Rank*	*kg/output*	*Rank*	*Annual risk/output*	*Rank*	*Annual risk/VA*	*Rank*	*Cost $M/VA*
Miscellaneous plastics products	320400	9,814,166	***1***	0.777	*20*	82.0	*11*	6.5E-06	*67*	1.5E-05	*67*	9.1E-07
Inorganic and organic chemicals	270100	5,989,212	***2***	26.904	***4***	56.1	*20*	2.5E-04	*14*	6.6E-04	*14*	4.1E-05
Paper and paperboard mills	240800	4,663,528	***3***	2.671	*14*	80.3	*13*	4.6E-05	*28*	1.1E-04	*29*	6.6E-06
Plastics materials and resins	280100	2,778,570	***4***	2.210	*16*	62.0	*19*	4.9E-05	*26*	1.5E-04	*27*	9.5E-06
Pulp mills	240100	2,328,159	***5***	1.252	*18*	741.0	***1***	4.0E-04	*11*	1.0E-03	9	6.5E-05
Reconstituted wood products	200904	2,117,857	***6***	0.116	*48*	371.9	***2***	2.0E-05	*43*	4.3E-05	*45*	2.7E-06
Petroleum refining	310101	2,109,115	***7***	4.679	9	18.5	*56*	4.1E-05	*30*	2.4E-04	*22*	1.5E-05
Drugs	290100	1,990,404	***8***	0.293	*32*	20.6	*52*	3.0E-06	*83*	5.6E-06	88	3.5E-07
Wholesale trade	690100	1,187,507	***9***	3.213	*11*	1.5	*172*	4.0E-06	*78*	6.1E-06	*84*	3.8E-07
Photographic equipment and supplies	630300	884,795	***10***	0.098	*51*	43.7	*25*	4.9E-06	*74*	7.7E-06	*78*	4.8E-07
Mineral wool	362000	876,798	***11***	0.001	*163*	191.1	*5*	3.2E-07	*159*	5.9E-07	*166*	3.7E-08
Fabricated metal products	421100	757,154	***12***	0.067	*60*	75.2	*15*	6.6E-06	*66*	1.4E-05	*68*	8.9E-07
Nonwoven fabrics	171001	657,477	***13***	0.040	*71*	173.9	*6*	1.1E-05	*58*	2.8E-05	*56*	1.8E-06
Plating and polishing	420401	642,705	***14***	0.058	*63*	105.8	*8*	9.6E-06	*59*	1.5E-05	*65*	9.4E-07
Sugar	141900	631,413	***15***	0.066	*61*	84.8	*10*	8.9E-06	*62*	4.1E-05	*46*	2.6E-06
Chemicals and chemical preparations	270406	592,797	***16***	0.343	*31*	44.8	*23*	2.6E-05	*37*	7.1E-05	*35*	4.5E-06
Primary aluminum	380400	575,150	***17***	40.378	***3***	91.9	9	6.5E-03	***2***	2.1E-02	***1***	1.3E-03
Steel pipe and tubes	370105	535,922	***18***	0.035	*74*	74.4	*16*	4.9E-06	*73*	1.4E-05	*71*	8.6E-07
Motor vehicle parts and accessories	590302	505,805	***19***	0.038	*72*	4.2	*120*	3.1E-07	*161*	1.1E-06	*143*	7.0E-08
Pesticides and agricultural chemicals	270300	487,875	***20***	0.165	*40*	43.5	*26*	1.5E-05	*50*	2.9E-05	*55*	1.8E-06

:	:	:	:	:	:	:	:	:	:	:	:	:
Manmade organic fibers	280400	229,888	*40*	17.006	***5***	19.1	*55*	1.4E-03	***4***	3.9E-03	***4***	2.4E-04
Thread mills	160400	191,249	*45*	0.008	*114*	193.7	***4***	7.9E-06	*63*	2.4E-05	*61*	1.5E-06
Gum & wood chemicals	270401	162,443	*52*	0.525	*24*	198.0	***3***	6.4E-04	*7*	1.6E-03	*7*	9.9E-05
Utilities	680100	81,568	*72*	41.214	***2***	0.4	*213*	2.1E-04	*15*	3.0E-04	*19*	1.9E-05
Cement, hydraulic	360100	66,024	*78*	47.411	***1***	9.8	*78*	7.0E-03	***1***	1.2E-02	***3***	7.5E-04
Secondary nonferrous metals	380600	40,283	98	13.440	*7*	6.5	99	2.2E-03	***3***	1.3E-02	***2***	8.0E-04
Primary nonferrous metals	380501	39,304	*100*	3.069	*12*	11.8	*74*	9.2E-04	***5***	2.0E-03	***5***	1.3E-04

[1]Ranked highest to lowest. Top five industries ranked by measure of cancer risk and cost also included. Dots indicate gaps in the rankings for each measure of performance.
[2]Reallocated emissions.
Note: BEA Ind = U.S. Bureau of Economic Analysis industry code; VA = value added; $M = million U.S. dollars; one kilogram (kg, SI) ≈ 2.204 pounds (lbs). Industry sectors ranked according to emissions (kg) that also rank among the top five by other measures of environmental performance are in **bold**.

ratio of industry direct to total supply chain (direct + indirect) annual cancer risk per $1 million output for our sample is 2×10^{-4}, with a minimum of zero and a maximum of 0.96. The median ratio of direct to total kg emissions per $1 million output is 3.5×10^{-3}, ranging from zero to a maximum of 0.99. Figure 1 further amplifies this concentrated nature of the cancer risk we estimate.

Here we show industry rankings by the ratio of per industry sector direct to total impacts for a measure in kg/output and risk/output over the entire 490-industry sample. Although both kg and risks per $1 million output are highly concentrated in a few industries, risks/output are even more concentrated than kg/output, both because of to chemical specific emissions from a few industries and because approximately half of our sample does not report direct TRI emissions.

We provide further detail on direct versus upstream emissions for the top 20 industries ranked by direct risk/$1 million output and by total risk/$1 million output in figures 2 and 3, respectively. Although supply chain risks are more widely dispersed on average, we again note the relatively high concentration of direct cancer risks in a few PACs- and/or dioxin-emitting industries, notably the primary aluminum, cement, and manmade organic fiber industries (figure 2).

For these same industries, high direct risks overwhelm rankings by total risk, and with EIO-LCA we find that supply chains highly dependent on aluminum or cement inputs also rank in the top 20 (figure 3). For the entire industry sample, we find that the median percentage of upstream risk/$1 million output is close to 100%, with only minor impacts associated with direct emissions of the median industry. Clearly, this is driven by very small or zero levels of direct TRI emissions reported by a significant portion of our industry sample, though these industries contribute to U.S. population risk by triggering emissions in their supply chains. A test of validity based on epidemiology shows that a ranking based on emissions of toxic compounds per $1 million output has low sensitivity (15%), implying that only 15% of high-risk industry supply chains would be identified and the remaining 85% would not be correctly identified as risky.

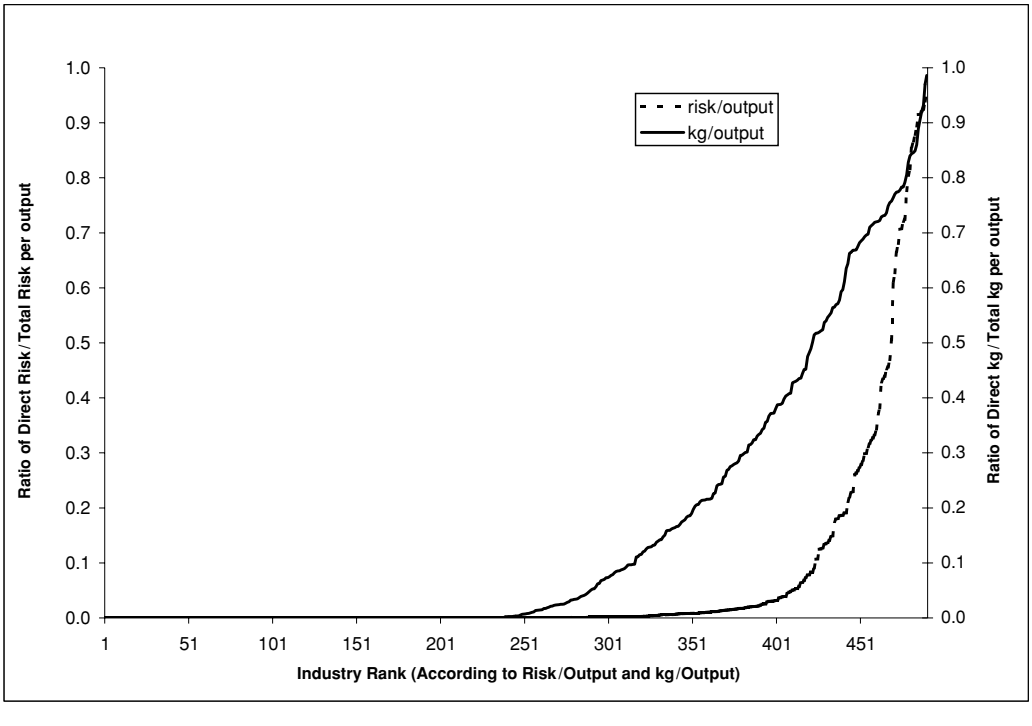

Figure 1 Ratio of per industry direct to total kilograms emissions and cancer risk, per $1 million U.S. dollars output.

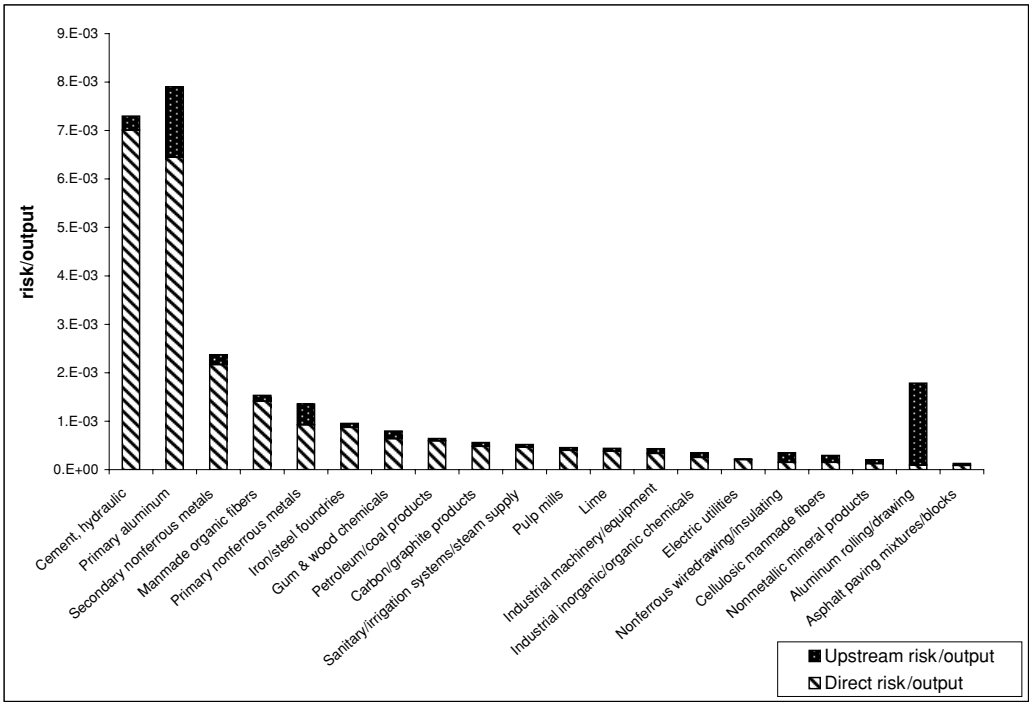

Figure 2 Ranking of top 20 industry sectors by direct cancer risk per $1 million U.S. dollars output.

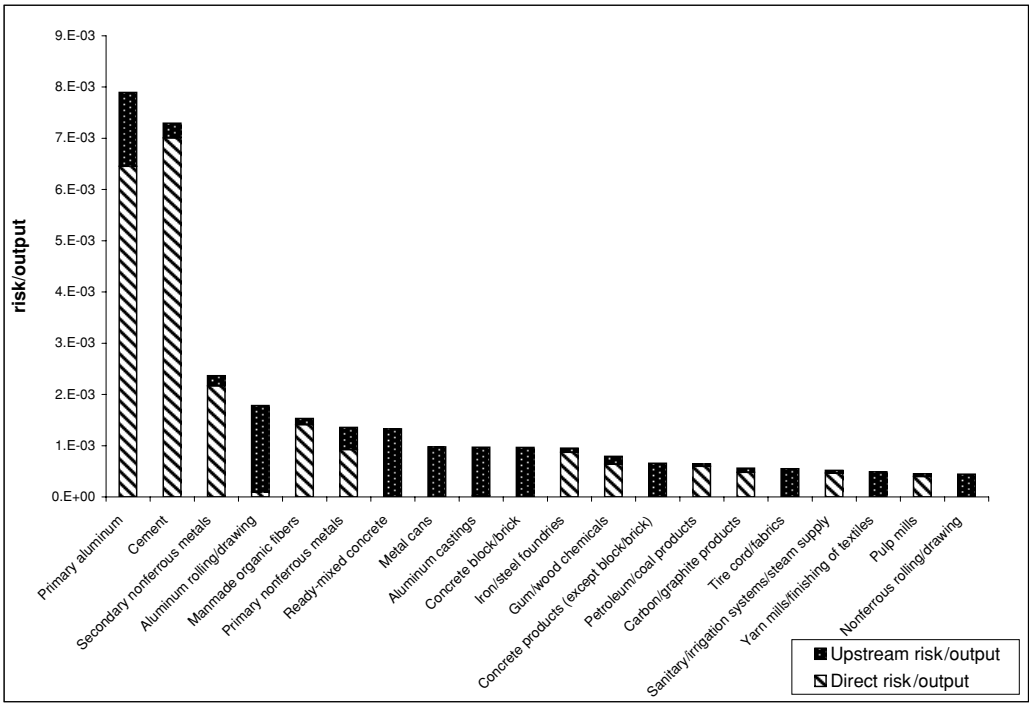

Figure 3 Ranking of top 20 industry sectors by total (direct + indirect) cancer risk per $1 million U.S. dollars output.

Sensitivity Analysis

As with all EIO-LCA, there is parametric and model uncertainty in this analysis. We evaluate a number of scenarios using alternative parameter values or model assumptions and determine the impact for each scenario. The scenarios include uncertainty on key parameters for PAC and dioxin emissions (e.g., cancer potency, half-life in air, and emissions speciation) and parameters related to cancer WTP. We evaluate the sensitivity using the ratios of annual cancer risk (including PAC and dioxins risks separately) and total cost determined for a given scenario to baseline conditions as well as changes in the top 10 industry rankings. Table 3 summarizes these results. In each scenario, we change only one parameter, leaving all others fixed at the baseline value. Two additional sources of uncertainty are discussed qualitatively with respect to their effect on industry sector rankings (e.g., allocation uncertainty in EIO-LCA and site-specific parameters). In this section, we do not address data input uncertainties related to TRI emission factors and economic input-output data or model uncertainties associated with the EIO-LCA and CalTOX models discussed elsewhere (Hertwich et al. 1999; Huijbregts et al. 2000).

PAC Emissions Potency

Prior research has shown that cancer risk assessment of hazardous air pollutant (HAP) emissions is sensitive to assumptions on polycyclic organic matter (POM) emissions speciation (Woodruff et al. 2000), of which the TRI PACs are a subgroup. Lacking measured data, the EPA assumed a baseline value of 16% BaP potency for POM emissions and a lower bound of 6% BaP potency (Caldwell 1998). Our baseline risk estimate uses a PAC emissions distribution from the NTI, yielding a PAC potency equivalent to 6.6% BaP. Applying the EPA's 16% BaP potency more than doubles PAC risk and yields only a 27% increase in total risk, because PAC

Table 3 Summary of sensitivity analysis (ratio of annual cancer risk with model perturbations to baseline annual cancer risk, total social cost, and supply chain rankings)

Parametric change scenarios	*PAC risk*	*Dioxin risk*	*Total risk (annual cancer cases in the U.S.)*	*Discounted social cost ($1M in 1998 U.S. dollars)*[1]	*Change in top ten supply chain rankings*
Baseline 6.6% BaP Potency	**1**	**1**	**260**	**$702**	**No**
1. 16% BaP (Caldwell et al.)	2.33	1	1.27	$882	Yes
2. 5% BaP (MACT inlet)	0.66	1	0.93	$646	No
3. 0.04% BaP (MACT outlet)	0.13	1	0.82	$571	Yes
4. CalTOX 1/2 life in air for PAC	0.30	1	0.86	$595	No
5. TCDD 1/2 life in air, 10th percentile value	1	0.97	0.98	$677	No
6. TCDD 1/2 life in air, 90th percentile value	1	1.01	1	$697	No
7. TCDD CPF (Cal EPA)	1	0.13	0.34	$233	Yes
8. Cancer WTP, 10th percentile value	1	1	1	$143	NA
9. Cancer WTP, 90th percentile value	1	1	1	$1,007	NA

[1] Assuming 10-yr latency period, 5% discount rate.

emissions account for 20% of total risk compared to 76% associated with dioxin emissions.

Alternatively, because 88% of TRI PACs are emitted by the primary aluminum industry (SIC 3334, BEA 380400), we apply sector-specific speciation data. Stack emissions (inlet and outlet) were measured in preparation for the 1997 primary aluminum Maximum Available Control Technology (MACT) POM standard for eight of 21 PACs (USEPA 1996b, 1997a).[16] Using the inlet distribution for SIC 3334 PAC emissions is equivalent to 5% BaP potency and reduces PAC risk by 24%, with a minimal effect on total risk. But if all facilities use control technology with 85–99% efficiency reflected in outlet data, this yields a 0.04% BaP potency and reduces PAC risk by 87% and total risk by 18% (table 3, scenario 3). Scenario 3 assumes that all SIC 3334 facilities are compliant with the 1997 MACT standard, an unlikely prospect in 1998. Basing our 1998 TRI PAC emissions speciation on NTI data thus appears a reasonable baseline.

Of these three parameter perturbations we find that scenarios 1 and 3 change the top 10 supply chain rankings, as these two scenarios reflect a more significant change in the potency of the emissions distribution. Specifically, ranking of primary aluminum as the highest supply chain risk, followed by the cement industry, is preserved under scenarios 1 and 3. Slight shifts occur, though, among other supply chains in the top 10 rankings as PAC risks increase or decrease to reflect changes in PAC emissions speciation.

Half-Life in Air

Prior research finds that intake fraction (IF) estimates are very sensitive to various physical-chemical properties (Hertwich et al. 1999), particularly medium-specific half-lives (Huijbregts 1999), which can generate uncertainties in risk estimates of up to six orders of magnitude (Huijbregts et al. 2000). Again focusing on PACs, Monte Carlo analysis in CalTOX shows that for BaP, the most studied PAC compound, IF is most sensitive to the half-life in air. Our baseline assessment used half-life in air for each PAC calculated with hydroxyl radical (OH) rate constants (Meylan and Howard 1993) based on quantitative structure-activity relationship (QSAR) methods developed by Atkinson (Atkinson 1989) and a 9.7×10^5 moles per milliliter (mol/mL) OH concentration (Prinn et al. 1995).[17] Mean half-lives in air were previously estimated in CalTOX v.4 for a subset of eight PACs using all available data (Chiao et al. 1995; CalTOX 2001) and are generally lower than those calculated for our baseline analysis. For example, our baseline value for BaP, 1.65×10^{-1} day, exceeds the earlier value (6.32×10^{-2} day) by approximately a factor of two. Using the lower half-life in air values from CalTOX v.4 reduces intake fractions for PACs, and hence PAC risk (70%) and total cancer risk by 14% (table 3, scenario 4). In comparison, sensitivity of dioxin risk to the half-life in air, based upon the 10th and 90th percentile values determined with Monte Carlo simulation for 2,3,7,8-TCDD, has a limited effect on total risk (scenarios 5 and 6). These changes did not impact the risk significantly enough to result in changes in the rankings.

Dioxin Toxicity

Although the toxicity of 2,3,7,8-TCDD, used to estimate dioxin cancer risk, has been extensively studied, a fully developed model of TCDD toxicity is still lacking, due primarily to animal-to-human dose extrapolation uncertainty. Thus, the TCDD cancer potency has been amended several times for the EPA Reassessment, with a most recent value of 1×10^6 $(\text{mg/kg-day})^{-1}$. Alternatively, the California EPA proposes a TCDD potency of 1.3×10^5 $(\text{mg/kg-day})^{-1}$ (CalEPA 2002). Using this lower CPF yields a significant reduction in dioxin risk by 87% and a 66% reduction in total cancer risk (scenario 7). This scenario reduces supply chain risks associated with dioxin exposure, and several supply chains that release PACs (e.g., primary aluminum, aluminum castings, and carbon and graphite products) move up in the rankings. Under this scenario primary aluminum is still the highest risk supply chain; however, the cement industry falls to third place after the aluminum rolling and drawing industry.

Cancer WTP

Baseline cancer WTP was $4.4 million in 1998 as discussed earlier. This estimate depends on the VSL, cancer site-specific survival rate, treatment cost and latency period, and the discount rate, each of which may introduce uncertainty in our WTP estimate. Equation (2) can be amended to reflect cancer-site specific morbidity costs with COI as a percentage of VSL as follows:

$$\begin{aligned}\text{Cancer WTP} = {} & \text{survival rate} \\ & \times(\text{VSL} \times \text{COI/VSL}) \\ & +(1 - \text{survival rate}) \times \text{VSL}\end{aligned} \tag{2a}$$

Distributions were developed for each parameter in equation (2a) and a distribution for cancer WTP was determined using a Monte Carlo simulation. We include the 10th and 90th percentiles of cancer WTP as it affects total costs (table 3, scenarios 8 and 9).

The difficulty of deriving adverse health effect indicators for LCA and associated cost estimation has been noted, particularly when the actual mechanism is not fully understood (Olsen et al. 2000). To explore sensitivity of cancer WTP, we derive measures of health effects from the literature, again focusing on PAC and dioxin cancer risk. BaP exposure may be associated with increased forestomach and lung tumors in mice (Gaylor et al. 2000; Armstrong et al. 2003), in addition to lung, skin, and bladder cancer for occupational exposure (Boffetta et al. 1997). Occupational dioxin exposure studies indicate an increased risk of lung cancer and for all cancer sites combined (Zober et al. 1990; Fingerhut et al. 1991; Manz et al. 1991; Aylward et al. 1996; Becher et al. 1998; Steenland et al. 1999). Site-specific 5-year survival rates range between 14.7% for lung cancer and 78.5% for skin cancer, whereas digestive system cancers (e.g., bladder and stomach) fall in the middle of this range at 43.4% (Ries et al. 2002). Latency periods range from 0 to 10, 15, and 20 yr in the above-cited dioxin studies or are assumed to be between 5, 10, and 20 yr (USEPA 2000c).

For Monte Carlo simulation we assume uniform distributions for the 5-year survival rates (14.7–78.5%), nonfatal cancer morbidity adjustments (0.028–0.583),[18] and latency period (0–20 yr). For the VSL the EPA-recommended Weibull distribution is assumed ($5.8 million U.S. dollars [M] mean, $4.15 M standard deviation in 1998 dollars) (USEPA 1999). We apply a triangular distribution centered on 5% for the discount rate, bounded by the social discount rate (3%) and the opportunity cost of capital (7%) (USEPA 1999). Cancer site-specific 10-year treatment costs for stomach, bladder, and lung cancers are drawn from the EPA Cost of Illness Handbook (USEPA 2001).

The discounted social costs calculated with the 10th and 90th percentile cancer WTP differ by one order of magnitude (table 3), which indicates the wide range of this uncertainty. Our baseline analysis yielded $702 million (1998 dollars) discounted at 5% over a 10-year latency period. Cancer WTP uncertainty is most sensitive to VSL (76.4%), followed by latency period (12%) and survival rate (8.4%), and is not very sensitive to morbidity adjustments and the discount rate in all scenarios, similar to prior research findings (USEPA 1997b; Levy 1999).

Allocation Uncertainty

Allocation uncertainty is one of several plausible sources of uncertainty specific to the EIO-LCA model (Lenzen 2001) discussed qualitatively here. Allocation uncertainty arises when a facility produces goods that fall under two or more SIC codes and therefore we do not know the proportion of emissions released for each SIC code. For these joint production facilities the researcher can inadvertently introduce an upward or downward bias to industry TRI emissions coefficients if releases are not properly allocated across SICs. This in turn can impact the matching of TRI and BEA databases in this analysis. We find a fairly high 1–1 correspondence for manufacturing industries, because both systems determine industry class based on the value of products produced/shipped or services provided.[19] Evidence of high intraindustry variation in levels of reported TRI emissions relative to inter-industry variation, particularly at higher levels of SIC aggregation (Streitwieser 1994), indicates the potential for heterogeneity at the four-digit SIC levels used in this analysis.

In 1998, 34% of our sample TRI chemical emissions were reported by 964 joint production facilities under up to six different SIC codes; the remaining emissions were reported under a single SIC code. To evaluate the sensitivity of our results to allocation uncertainty, we use a simple rule and allocate emissions equally across all listed SIC codes per multi-establishment facility and compare this to the traditional method of allocating all TRI emissions to the first-listed SIC code. Given the high concentration of cancer risk in industries that emit PACs and dioxin (e.g., primary aluminum and cement), we would expect our reallocation to change industry rankings when significant amounts of these compounds were emitted from joint production facilities. Significantly less than 1% of dioxins, though, are emitted from joint production facilities. The number is somewhat greater for PACs, being 25%, yet overall these joint production facilities do not affect the relative impact of direct versus upstream risks and produce no change in total risk or the top 10 industry risk rankings. Reallocating TRI emissions of our chemical sample over all listed SIC codes to account for joint production facilities does add several industry sectors to the analysis not included otherwise.

Site-Specific Modeling

Finally, uncertainty may arise due to the lack of site-specific modeling of fate, transport, and exposure. For our baseline analysis we define the continental United States as a single compartment in CalTOX. Ideally, life-cycle inventory analysis (LCIA) should reflect a compound's effective range of impact through its characteristic travel distance (Bennett et al. 1998) and, within that region, use site-specific inputs, such as population density (Spadaro and Rabl 1999; Crettaz 2000) and food production rates. It is difficult to complete this analysis on a site-specific basis, first, because true multimedia models accounting for spatial variation have only just become available. Research using a more site-specific multimedium multipathway fate and exposure model similar to CalTOX indicates that this would be a refinement of our approach (MacLeod et al. 2004). MacLeod and colleagues find that intake fractions across North American regions can vary by several orders of magnitude for inhalation-dominant compounds (e.g., benzene and carbon tetrachloride), which pose increased risks for releases in urban areas with higher population density. They find less variation for ingestion-dominant compounds, such as BaP and TCDD, with greater risks occurring for releases in areas with increased food production (MacLeod et al. 2004). Second, we have compared industries based on risk per economic output. Facility-level economic output data necessary for a site-specific assessment using EIO-LCA are not, however, easily accessed through public databases, including the closely guarded U.S. Census microdata.

Conclusions and Discussion

In this article we propose a method for understanding environmental performance through a measure of public health impacts of carcinogenic TRI air emissions. In this analysis we find that emissions of dioxin and PACs, which are ingestion-dominant, pose a greater cancer risk to the U.S. population than other compounds that are inhalation-dominant. We find that a significant portion of cancer risks arise from upstream economic activity. Ranking by TRI emissions identifies only four of the top ten high-risk supply chains identified using the EIO-LCA and toxicity weighting method shown here. Population risk estimates and industry rankings are very sensitive to the dioxin cancer potency factor, yielding a factor of ten difference in calculated dioxin cancer cases from the higher CPF used by the EPA and the one suggested by the California EPA. Analysis of the sensitivity of cancer WTP to latency, cancer specific survival rate, cost of morbidity, and discount rate revealed that these parameters were far less important than variations in VSL.

This risk-based approach implies a policy aimed at limiting U.S. population exposure via ingestion of highly toxic emissions, such as PACs and dioxins. Given the disproportionately higher cancer risks associated with emissions of these compounds, regulators should pay special attention to ensuring accurate reporting thereof and encouraging speciation of compound groups, such as PACs. Currently these and other persistent bioaccumulative toxic (PBTs) chemicals, which

have lower reporting thresholds, are analyzed separately in the EPA's TRI data releases. Future research using the approach proposed here should include more PBTs than our analysis, which has been limited due to data availability.

Our results also show that there are clear opportunities to intervene to reduce impacts across supply chains. This includes enforcement of existing industry-specific MACT standards aimed at primary aluminum industry PAC emissions and dioxin emissions from hazardous waste burning cement kilns. Alternatively, exerting demand side pressure either via policy initiatives or private sector efforts can reduce the adverse impact of upstream risks identified here. Ideally, corporate managers concerned with minimizing public health risks should focus on PAC and dioxin exposure throughout their supply chains and provide specific information on PBT releases in corporate reports to enable performance evaluations that are more directly related to health impacts. One manner in which this could be enabled would be for companies to (voluntarily) submit confidential data on their purchases by sector, possibly to the EPA. This information could then be used to compute upstream impacts using EIO-LCA, and the ensuing results could be provided to these companies to allow management to reduce purchases associated with high impacts in their supply chain. A more likely path would be for industry associations, such as the Global Environmental Management Initiative (GEMI) and international standards associations, such as drafters of the ISO 14001 environmental management standard (EMS), to reflect social impacts of emissions in their definition of "performance." Finally, socially responsible investors who analyze environmental and sustainability reports should note that aggregated mass emissions cannot adequately serve as a proxy for the social impacts of highly toxic compounds. A special focus on PBTs in environmental performance reviews would be highly beneficial from a public health perspective.

Taken at face value, our results imply that TRI health impacts are limited to PAC and dioxin emissions. But noncancer risks (e.g., hormonal/fertility changes, developmental, in-utero effects, and risks posed to infants by lactating mothers) are important intergenerational effects associated with toxic releases that should be considered, particularly from the sustainability standpoint. These effects may widen our analytical scope to include additional TRI chemicals of major concern that may not similarly pose a cancer risk. Ideally, a measure of environmental performance should reflect the combined impacts on human health and the ecosystem of various pollutant vectors, such as greenhouse gases, releases to water, toxic chemical emissions, and acid rain. In many cases, this will require future research relating these pollutant vectors to human health and ecosystem outcomes via a damage function.

Our approach has a limited scope for additional reasons. U.S. population cancer risk assessments based solely on the TRI database will generally underestimate potential exposures to toxic chemicals, because not all of the roughly 70,000 commercial chemicals in global circulation today are included. Furthermore, atmospheric transformation of more benign TRI chemicals, such as toluene and various PAHs, may yield more toxic chemicals not accounted for in this analysis (Dumbie et al. 1988; Kelly et al. 1994). The problems of measurement error and lack of reliable independent third-party verification of the TRI are notable.

A public health risk-based measure of environmental performance using EIO-LCA and the TRI cannot capture significant nonstationary source air emissions. Thus, with a limited chemical sample of stationary industry source emissions, our estimated 260 annual cases are lower than 800 excess annual cancer cases estimated for 1990 outdoor concentrations of 148 hazardous air pollutants associated with inhalation exposure to *both* stationary and nonstationary sources (Woodruff et al. 2000).[20] To illustrate, stationary sources account for approximately 10% of PAH air emissions (Yaffe et al. 2001), whereas vehicular combustion (i.e., product use) accounts for the majority of PAH air emissions (Harrison et al. 1996; Kavouras et al. 1999; Lim et al. 1999). Similarly, stationary sources account for only 3% of personal benzene exposure, whereas automobiles account for 82% of benzene emissions (Wallace 1990). Thus, unsurprisingly, we find a negligible population cancer risk associated with 1998 TRI benzene air emissions. These effects can be

evaluated with a complete hybrid LCA, combining the benefits of EIO-LCA for upstream analysis with traditional process LCA for both product use and end of life phases (Hendrickson et al. 1998).

Future revisions of this analysis should capture the cumulative lifetime cancer risk arising from TRI emission years prior or subsequent to 1998. An approach to modeling time series environmental burdens is the Computerized General Equilibrium (CGE) model, which accounts for important changes in economic relationships that drive emissions levels. Time series analyses also need to account for changes in TRI reporting requirements, gradual regulatory compliance (e.g., MACT), and outsourcing of major emissions sources. In addition, new scientific knowledge of the behavior and risks of various toxic chemicals will affect TRI cancer risk estimates. For example, future revision of the EPA Guidelines for Carcinogen Assessment to reflect dose response relationships specific to different modes of action and routes of exposure will impact this type of analysis (USEPA 1996a). Finally, although desirable, developing a firm level measure of environmental performance based on this method is complicated by the lack of publicly available firm level direct economic data to populate the input-output model. Firm-level analysis would be possible, assuming upstream interindustry transactions based on the data from the national accounts, but is potentially nonspecific. The analysis offered here can serve as an industry benchmark for future firm-level analyses and can help focus regulator and corporate policy on toxic chemical risk management.

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Notes

1. Several of these SICs are not required to report under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), yet appear in the dataset, because facilities include them in their reports if they are reporting under multiple SIC codes.
2. Another potential source of underreporting is levels at which facilities are required to report. Whereas, for most chemicals, reportable quantities start at 10,000 pounds (lb) of emissions per year, for persistent bioaccumulative toxic (PBT) chemicals the EPA has established lower reporting thresholds (one pound [lb] $\approx$ 0.4536 kilograms [kg, SI]). Thus for dioxin it is 0.1 g and for PACs 100 lb per year. Excluding emissions below reportable quantities would not greatly influence our analysis, because the majority of risk is associated with dioxin and PAC emissions, which have such low reporting thresholds.
3. The World Bank's ToxInt database similarly applies TLVs to 246 TRI chemicals.
4. CalTOX is a quasi-dynamic model that reaches steady state for all compounds within 30 yr.
5. CalTOX CPFs are drawn from the U.S. EPA's Integrated Risk Information System (IRIS), from the California Department of Toxic Substances Control, and from data tables used in work by Hertwich and colleagues (2001).
6. PEFs are based on cancer bioassay information and are specific to cancer risk estimation, whereas dioxin TEFs refer to multiple endpoints and are based on a combination of studies, including acute toxicity determination and limited cancer bioassay data (Collins et al. 1998).
7. TEQs are applied to dioxin emissions as follows: $TEQ \cong \Sigma(congener_1 \times TEF_1) + \ldots + (congener_j \times TEF_j)$.
8. One picogram (pg) $= 10^{-12}$ grams (g) $\approx 3.53 \times 10^{-14}$ ounces (oz).
9. We cannot compare measured PAC concentrations to modeled results, because a national PAH emissions inventory is not readily available. High BaP concentrations measured in various cereals and vegetables (Kazerouni et al. 2001) are predicted by CalTOX, though, increasing confidence in the model.
10. Others use quality adjusted life years (QALY) to monetize pain and suffering (Tolley et al. 1994) But QALY estimation relies on much stricter assumptions than WTP, generally ignores income, and has not been fully integrated with

welfare-based measures of value (Johannson 1995; USEPA 2000a).

11. These results hold only for lymphoma and may not necessarily be generalizable to other types of cancer.
12. Demand is set as the identity matrix such that an industry's environmental burden is a reflection of steady-state output to final demand for its product devoid of any output due to input requirements to fulfill final demand of *other* industry sector products.
13. Value added and output are highly correlated ($R^2 = 0.97$, $p \leq 0.001$) for U.S. industries, and thus normalizing cancer risk by value added to indicate the trade-off between jobs and human lives does not introduce additional bias.
14. Non-U.S.-based upstream production is captured under the assumption that overseas and U.S. production technologies are similar. For imports, domestic port value is assumed equivalent to the producers' price of comparable domestically produced commodities (Miller and Blair 1985; Lawson 1997).
15. Four industries (BEA codes 510104, 10302, 200400, and 210000) reporting negative VA in 1998 were excluded in the analysis. Of these only 510104 (computer peripheral equipment) and 200400 (special product sawmills) reported emissions of the TRI chemicals in our sample, ranking 206th and 2nd by direct kg emissions per output respectively. These two industries do not rank in the top 20 in terms of direct and total supply chain risk per output, and thus would not significantly affect our risk-based rankings.
16. Inlet test data are representative of precontrols, and outlet test data reflect environmental releases not captured or destroyed by air pollution control equipment with an 85–99% control efficiency range.
17. One mole (mol) of a substance $= 6.022 \times 10^{23}$ atoms, molecules, or other particles; the mass in grams of this amount of a substance is numerically equal to the molecular weight of the substance. One milliliter (mL) $= 10^{-3}$ liters (L) ≈ 0.034 fluid ounces.
18. Scholars recommend treating a COI-based cancer WTP for nonfatal cancers as the lower bound (here 2.8% VSL for stomach cancer) and the Magat and colleagues 58.3% adjustment to VSL as the upper bound of the estimation range. (SAB-EEAC 2000)
19. Initial matching of SIC codes with BEA industry codes leads to an 8% loss of data for our chemical sample. To minimize this we rematch SIC 7389 with BEA code 73.0109 and SIC 9711 with BEA industry 3.0001. SIC 2819, organic chemicals, emissions can be assigned either to BEA 27.01 or to BEA 38.04. As 94% of total sales are from 27.01, all 2819 emissions are allocated to this industry. SIC 4931 and 4933 emissions are assigned to 68.01 based on share of sales (96%) rather than 78.02.
20. Reductions in HAP emissions levels since 1990 will also explain this difference.

References

ACGIH (American Conference of Governmental Industrial Hygienists). 2003. *Threshold limit values (tlvs) and biological exposure indices (beis)*. Cincinnati, OH: ACGIH.

ACS (American Cancer Society). 1998. *Cancer facts and figures—1998*. Atlanta, Georgia: ACS.

Armstrong, B., E. Hutchinson, and T. Fletcher. 2003. *Cancer risk following exposure to polycyclic aromatic hydrocarbons (pahs): A meta-analysis*. London: London School of Hygiene and Tropical Medicine.

ASTM (American Society for Testing and Materials). 2003. *E2173-01 standard guide for disclosure of environmental liabilities*. West Conshohocken, PA: ASTM International.

Atkinson, R. 1989. *Kinetics and mechanisms of the gasphase reactions of the hydroxyl radical with organic compounds*. New York: American Institute of Physics and American Chemical Society.

Austin, D. and A. Sauer. 2002. *Changing oil: Emerging environmental risks and shareholder value in the oil and gas industry*. Washington, DC: WRI.

Aylward, L. L., S. Hays, and N. Karch. 1996. Relative susceptibility of animals and humans to the cancer hazard posed by 2,3,7,8-tetrachlorodibenzo-p-dioxin using internal measures of dose. *Environmental Science and Technology* 30: 3534–3543.

Bare, J. C., G. A. Norris, D. W. Pennington, and T. McKone. 2002. TRACI: The tool for the reduction and assessment of chemical and other environmental impacts. *Journal of Industrial Ecology* 6(3–4): 49–78.

Becher, H., K. Steindorf, and D. Flesch-Jnys. 1998. Quantitative cancer risk assessment for dioxins using an occupational cohort. *Environmental Health Perspectives* 106 (Suppl 2): 663–670.

Bennett, D., T. McKone, M. Matthies, and W. Kastenberg. 1998. General formulation of characteristic travel distance for semivolatile organic

chemicals in a multimedia environment. *Environmental Science and Technology* 32(24): 4023–4030.

Bennett, D. H., T. E. McKone, J. S. Evans, W. W. Nazaroff, M. Margni, O. Jolliet, and K. R. Smith. 2001. Defining intake fraction. *Environmental Science and Technology* 36: 206A–211A.

Bennett, D. H., M. D. Margni, T. E. McKone, and O. Jolliet. 2002. Intake fraction for multimedia pollutants: A tool for life cycle analysis and comparative risk assessment. *Risk Analysis* 22(5): 905–918.

Berg, M. V. d., L. Birnbaum, A. T. C. Bosveld, B. Brunstrom, P. Cook, M. Feeley, J. P. Giesy, A. Hanberg, R. Hasegawa, S. W. Kennedy, T. Kubiak, J. C. Larsen, F. X. R. v. Leeuwen, A. K. D. Liem, C. Nolt, R. E. Peterson, L. Poellinger, S. Safe, D. Schrenk, D. Tillitt, M. Tysklind, M. Younes, F. Waern, and T. Zacharewski. 1998. Toxic equivalency factors (tefs) for pcbs, pcdds, pcdfs for humans and wildlife. *Environmental Health Perspectives* 106(12): 775–792.

Birnbaum, L. S. 1999. TEFs: A practical approach to a real-world problem. *Human and Ecological Risk Assessment* 5(1): 13–24.

Boffetta, P., N. Jourenkova, and P. Gustavsson. 1997. Cancer risk from occupational and environmental exposure to polycyclic aromatic hydrocarbons. *Cancer Causes Control* 8(3): 444–472.

Caldwell, J. 1998. *Interim methodology to give estimates of potency for cumulative exposure project pom inventories*. Washington, DC: EPA.

Caldwell, J. C., T. J. Woodruff, R. Morello-Frosch, and D. A. Axelrad. 1998. Application of health information to hazardous air pollutants modeled in EPA's cumulative exposure project. *Toxicology and Industrial Health* 14(3): 429–454.

CalEPA (California Environmental Protection Agency). 2002. *Air toxics hot spots program risk assessment guidelines, part ii. Technical support document for describing available cancer potency factors*. OEHHA. <www.oehha.ca.gov/air/hot_spots/pdf/TSDlookup2002.pdf:>. Accessed August 2003.

Campbell, J. Y. 2000. Asset pricing at the millennium. *Journal of Finance* 55(4): 1515–1567.

Chiao, F. F., R. C. Currie, and T. E. McKone. 1995. *Intermedia transfer factors for contaminants found at hazardous waste sites, benzo(a)pyrene*. Davis, CA: Department of Environmental Toxicology, University of California.

Collins, J. F., J. P. Brown, G. V. Alexeeff, and A. G. Salmon. 1998. Potency equivalency factors for some polycyclic aromatic hydrocarbons and polycyclic aromatic hydrocarbon derivatives. *Regulatory Toxicology and Pharmacology* 28: 45–54.

Cowan, C., C. E. Mackay, T. C. J. Feijtel, D. v. d. Meent, A. D. Guardo, J. Davies, and N. Mackay. 1994. *The multi-media fate model: A vital tool for predicting the fate of chemicals*. Pensacola, FL: SETAC Press.

Crettaz, P. 2000. From toxic releases to damage on human health: A method for life cycle impact assessment, with a case study in domestic rainwater use. Lausanne, Switzerland: EPFL.

Cropper, M. L., S. K. Aydede, and P. R. Portney. 1994. Preferences for life-saving programs: How the public discounts time and age. *Journal of Risk and Uncertainty* 8: 243–265.

DeSimone, L. D. and F. Popoff. 1997. *Eco-efficiency, the business link to sustainable development*. Cambridge, MA: The MIT Press.

Ditz, D., J. Ranganathan, and R. D. Banks. 1995. *Green ledgers: Case studies in corporate environmental accounting*. Baltimore, MD: World Resources Institute.

Dumbie, B. E., D. V. Kenny, P. B. Shepson, T. E. Kleindienst, C. M. Nero, and L. T. Cupitt. 1988. Ms/ms analysis of the products of toluene photooxidation and measurements of their mutagenic activity. *Environmental Science and Technology* 22: 1493.

Fingerhut, M., W. Halperin, and D. Marlow. 1991. Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *New England Journal of Medicine* 324: 212–218.

Freeman, A. 1993. *The measurement of environmental and resource values: Theory and methods*. Washington, DC: Resources for the Future.

Gaylor, D. W., S. J. Culp, L. S. Goldstein, and F. A. Beland. 2000. Cancer risk estimation for mixtures of coal tars and benzo(a)pyrene. *Risk Analysis* 20(1): 81–85.

GRI (Global Reporting Initiative). 2002. *Sustainability reporting guidelines*. Boston: Global Reporting Initiative.

Grimstead, B., S. Schaltegger, C. Stinson, and C. S. Waldron. 1994. A multimedia assessment scheme to evaluate chemical effects on the environment and human health. *Pollution Prevention Review* Summer 1994: 259–268.

Hamilton, J. T. 1995. Pollution as news: Media and stock market reactions to the toxics release inventory data. *Journal of Environmental Economics and Management* 28: 98–113.

Hammitt, J. K. 2000. Valuing mortality risk: Theory and practice. *Environmental Science and Technology* 34: 1396–1400.

Hammitt, J. K. and J.-T. Liu. 2003. *Effects of disease type and latency on the value of mortality risk*.

Boston, MA: Harvard School of Public Health, manuscript.

Harrison, R. M., D. J. T. Smith, and L. Luhana. 1996. Source apportionment of atmospheric polycyclic aromatic hydrocarbons collected from an urban location in Birmingham, UK. *Environmental Science and Technology* 30(3): 825–832.

Hellweg, S., T. B. Hofstetter, and K. Hungerbuehler. 2002. Discounting and the environment: Should current impacts be weighted differently than impacts harming future generations. *International Journal of Life Cycle Assessment* 8(1): 8–18.

Hendrickson, C., A. Horvath, S. Joshi, and L. Lave. 1998. Economic input-output models for environmental life-cycle assessment. *Environmental Science & Technology* (April 1): 184A–191A.

Hertwich, E. 1999. Toxic equivalency: Addressing human health effects in life cycle impact assessment. Ph.D. dissertation, University of California, Berkeley, CA.

Hertwich, E., S. F. Mateles, W. S. Pease, and T. E. McKone. 2001. Human toxicity potential for life cycle analysis and toxics release inventory risk screening. *Environmental Toxicology and Chemistry* 20(4): 928–939.

Hertwich, E. G., T. E. McKone, and W. S. Pease. 1999. Parameter uncertainty and variability in evaluative fate and exposure models. *Risk Analysis* 19(6): 1193–1204.

Hertwich, E., W. Pease, and T. McKone. 1998. Evaluating toxic impact assessment methods: What works best? *Environmental Science and Technology* March 1: 138–144.

Horowitz, J. K. and R. T. Carson. 1990. Discounting statistical lives. *Journal of Risk and Uncertainty* 3: 402–413.

Horvath, A., C. T. Hendrickson, L. B. Lave, F. C. McMichael, and T.-S. Wu. 1995. Toxic emissions indices for green design and inventory. *Environmental Science and Technology* 29(2): 86A–90A.

Huijbregts, M. A. J. 1999. Priority assessment of toxic substances in the frame of LCA, development and application of the multi-media fate, exposure and effect model uses – LCA. Amsterdam: Faculty of Environmental Sciences, University of Amsterdam.

Huijbregts, M. A. J., U. Thissen, T. Jager, D. v. d. Meent, and A. M. J. Ragas. 2000. Priority assessment of toxic substances in LCA II: Assessing parameter uncertainty and human variability on the calculation of toxicity potentials. *Chemosphere* 41: 575–588.

Innovest. 2003. *Carbon disclosure project*. New York: Innovest Strategic Value Advisors.

Jia, C. and A. D. Guardo. 1996. Toxics release inventories: Opportunities for improved presentation and interpretation. *Environmental Science and Technology* 30(2): 86–91.

Johansson, P. O. 1995. *Evaluating health risks: An economic approach*. Cambridge, UK: Cambridge University Press.

Jorgenson, D. W., J. S. Landefeld, and W. D. Nordhaus. 2005. *A new architecture for the U.S. national accounts*. Chicago: The University of Chicago Press.

Joshi, S. 2000. Product environmental life-cycle assessment using input-output techniques. *Journal of Industrial Ecology* 3(2&3): 95–120.

Kavouras, I. G., J. Lawrence, P. Koutrakis, E. G. Stephanou, and P. Oyola. 1999. Measurement of particulate aliphtic and polynuclear aromatic hydrocarbons in Santiago de Chile: Source reconciliation and evaluation of sampling artifacts. *Atmospheric Environment* 33: 4977–4986.

Kazerouni, N., R. Sinha, C.-H. Hsu, A. Greenger, and N. Rothman. 2001. Analysis of 200 food items for benzo(a)pyrene and estimation of its intake in an epidemiologic study. *Food and Chemical Toxicology* 39: 423–436.

Kelly, T., R. Mukund, C. Spicer, and A. Polack. 1994. Concentrations and transformations of hazardous air pollutants. *Environmental Science and Technology* 28: 378A–387A.

Khanna, M., W. R. H. Quimio, and D. Bojilova. 1998. Toxics release information: A policy tool for environmental protection. *Journal of Environmental Economics and Management* 36: 243–266.

Lave, L. B., E. Cobas-Flores, C. T. Hendrickson, and F. V. McMichael. 1995. Using input-output analysis to estimate economy-wide discharges. *Environmental Science and Technology* 29(9): 420A–426A.

Lawson, A. M. 1997. Benchmark input-output accounts for the U.S. Economy, 1992: Make, use and supplementary tables. *Survey of Current Business* November 1997: 36–82.

Lenzen, M. 2001. Errors in conventional and input-output life-cycle inventories. *Journal of Industrial Ecology* 4(4): 127–148.

Levy, J. I. 1999. Environmental health effects of energy use: A damage function approach. Sc.D. dissertation, Boston: Department of Environmental Health and Health Policy and Management, Harvard School of Public Health.

Lim, L. H., R. M. Harrison, and S. Harrad. 1999. The contribution of traffic to atmospheric concentrations of polycyclic aromatic hydrocarbons. *Environmental Science and Technology* 33(20): 3538–3542.

Lum, S. K. S. and B. C. Moyer. 2001. Gross domestic product by industry for 1998–2000. *Survey of Current Business* November 2001.

MacLeod, M., D. H. Bennett, M. Perem, R. Maddalena, T. McKone, and D. Mackay. 2004. Dependence of intake fraction on release location in a multimedia framework: A case study of four contaminants in North America. *Journal of Industrial Ecology* 8(3): 89–102.

Mackay, D. 1991. *Multimedia environmental fate models: The fugacity approach*. Chelsea, MI: Lewis Publishers.

Mackay, D., W. Y. Shiu, and K. C. Ma. 1992. *Illustrated handbook of physical-chemical properties and environmental fate for organic chemicals, Vol. ii*. Chelsea, MI: Lewis Publishers.

Magat, W. A., W. K. Viscusi, and J. Huber. 1996. A reference lottery metric for valuing health. *Management Science* 42(8): 1118–1130.

Manz, A., J. Berger, and J. Dwyer. 1991. Cancer mortality among workers in chemical-plant contaminated with dioxins. *Lancet* 338: 959–964.

Matthews, H. S. and M. J. Small. 2001. Extending the boundaries of life-cycle assessment through environmental economic input-output models. *Journal of Industrial Ecology* 4(3): 7–10.

McKone, T. E. 1993. *Caltox, a multimedia total-exposure model for hazardous-wastes sites. Part ii: The dynamic multimedia transport and transformation model*. Livermore, CA: Lawrence Livermore National Laboratory.

Meylan, W. M. and P. H. Howard. 1993. Computer estimation of the atmospheric gas-phase reaction rage of organic compounds with hydroxyl radicals and ozone. *Chemosphere* 26: 2293–2299.

Miller, R. E. and P. D. Blair. 1985. *Input-output analysis: Foundations and extensions*. Englewood Cliffs, NJ: Prentice-Hall, Inc.

Natan, T. E. and C. G. Miller. 1998. Are toxic release inventory reductions real? *Environmental Science and Technology* 32(15): 368A–374A.

Nisbet, I. C. T. and P. K. LaGoy. 1992. Toxic equivalency factors (TEFs) for polycyclic aromatic hydrocarbons (PAHs). *Regulatory Toxicology and Phamacology* 16: 290–300.

OEHHA (Office of Environmental Health Hazard Assessment). 1994. *Benzo(a)pyrene as a toxic air contaminant*. California: Office of Environmental Health Hazard Assessment, Sacramento, CA: California EPA.

Olsen, S. I., W. Krewitt, P. Cretaz, D. Pennington, and S. Effting. 2000. *Indicators for human toxicity in life cycle impact assessment*. Working Paper for SETAC-Europe WIA2 Task Group on Human Toxicity.

Prinn, R., R. Weiss, B. Miller, J. Huang, F. Alyea, D. Cunnold, P. Fraser, D. Hartley, and P. Simmonds. 1995. Atmospheric trends and lifetime of CH_3 CCl_3 and global OH concentrations. *Science* 269(5221): 187–192.

Repetto, R. and D. Austin. 2000. *Pure profit, the financial implications of environmental performance*. Washington, DC: World Resources Institute.

Revesz, R. L. 1999. Environmental regulation, cost-benefit analysis, and the discounting of human lives. *Columbia Law Review* 99: 941–1017.

Ries, L. A. G., M. P. Eisner, C. L. Kosary, B. F. Hankey, B. A. Miller, L. Clegg, and B. K. E. (eds). 2002. *Seer cancer statistics review, 1973–1999*. <http://seer.cancer.gov/csr/1973_1999/>. Bethesda, MD: National Cancer Institute.

Rowe, R. D., C. M. Lang, L. G. Chestnut, D. A. Latimer, S. M. Bernow, and D. E. White. 1995. *The New York electricity externality study. Vol 1: Introduction and methods*. New York: Empire State Energy Research Corporation.

Russell, L. B., M. R. Gold, J. E. Siegel, N. Daniels, and M. C. Weinstein. 1996. Recommendations of the panel on cost-effectiveness in health and medicine. *JAMA* 276(15): 1253–1258.

SAB-EEAC (Science Advisory Board Environmental Economics Committee). 2000. *An SAB report on EPA's whitepaper valuing the benefits of fatal cancer risk reductions*. EPA-SAB-EEAC-00-013. Washington, DC: EPA Science Advisory Board.

Schaltegger, S. and A. Sturm. 1990. Oekologische rationalitaet. *Die Unternehmung* 4: 273–290.

Schmidheiny, S. 1992. *Changing course: A global business perspective on development and the environment*. Boston: MIT Press.

Schneider, K., M. Roller, F. Kalberlah, and U. Schuhmacher-Wolz. 2002. Cancer risk assessment for oral exposure to PAH mixtures. *Journal of Applied Toxicology* 22: 73–83.

Spadaro, J. V. and A. Rabl. 1999. Estimates of real damage from air pollution: Site dependence and simple impact indices. *International Journal of Life Cycle Assessment* 4: 229–243.

SRC (Syracuse Research Corporation). 2003. *Syracuse Research Corporation*. <esc.Syrres.Com/>.

Steenland, K., L. Piacitelli, and J. Deddens. 1999. Cancer, heart disease, and diabetes in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Journal of National Cancer Institute* 91: 779–786.

Streitwieser, M. L. 1994. *Cross sectional variation in toxic waste releases from the U.S. Chemical industry*. Washington, DC: Center for Economic Studies, U.S. Bureau of the Census.

Toffel, M. W. and J. D. Marshall. 2004. Improving environmental performance assessment. *Journal of Industrial Ecology* 8(1–2): 143–172.

Tolley, G., D. Kenkel, and R. Fabian. 1994. State-of-the-art health values. In *Valuing health for policy*, edited by G. Tolley, D. Kenkel, and R. Fabian. Chicago: University of Chicago Press.

Torrance, G. W. 1986. Measurement of health state utilities for economic appraisal: A review. *Journal of Health Economics* 5(4): 1–30.

USEPA. 1996a. *Proposed guidelines for carcinogen risk assessment*. EPA/600/P-92/003C. Washington, DC: US EPA.

USEPA. 1996b. *Primary aluminum industry: Technical support document for proposed mact standards*. Washington, DC: US EPA, Office of Air Quality Planning and Standards.

USEPA. 1997a. National emissions standards for hazardous air pollutants for primary aluminum reduction plants. *Federal Register* 62(194): 52383–52428.

USEPA. 1997b. *The benefits and costs of the Clean Air Act, 1970 to 1990*. Washington, DC: US EPA, Office of Air and Radiation.

USEPA. 1999. *The benefits and costs of the Clean Air Act 1990 to 2010*. EPA-410-R-99-001. Washington, DC: US EPA, Office of Air and Radiation, Office of Policy.

USEPA. 2000a. *Guidelines for preparing economic analyses*. Washington, DC: Office of the Administrator.

USEPA. 2000b. *Valuing fatal cancer risk reductions*. Washington, DC: US EPA.

USEPA. 2000c. *Arsenic in drinking water rule economic analysis*. EPA 815-R-00-026. Washington, DC: US EPA.

USEPA. 2000d. *Toxic chemical release inventory reporting forms and instructions*. Washington, DC: US EPA.

Viscusi, W. K. 1993. The value of risks to life and health. *Journal of Economic Literature* 31: 1912–1946.

Wallace, L. 1990. Major sources of exposure to benzene and other volatile organic chemicals. *Risk Analysis* 10(1): 59–64.

Warshawsky, D. 1999. Polycyclic aromatic hydrocarbons in carcinogenesis. *Environmental Health Perspectives* 107(4): 317–319.

Williams, E. D., R. U. Ayres, and M. Heller. 2002. The 1.7 kilogram microchip: Energy and material use in the production of semiconductor devices. *Environmental Science and Technology* 36: 5504–5510.

Woodruff, T. J., J. Caldwell, V. J. Cogliano, and D. A. Axelrad. 2000. Estimating cancer risk from outdoor concentrations of hazardous air pollutants in 1990. *Environmental Research* 82: 194–206.

Yaffe, D., Y. Cohen, J. Arey, and A. J. Grosovosky. 2001. Multimedia analysis of PAHs and nitro-PAHs daughter products in the Los Angeles basin. *Risk Analysis* 21(2): 275–294.

Zober, A., P. Messerer, and P. Huber. 1990. Thirty-four-year mortality follow-up of BASF employees exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) after a 1953 chemical reactor incident. *International Archives of Occupational Environmental Health* 62: 139–157.

Appendix A

Table 4 Listing of acronyms

BaP	benzo(a)pyrene
COI	direct costs of illness
CPF	cancer potency factor
EIO-LCA	economic input-output life-cycle assessment
GDP	gross domestic product
HAP	hazardous air pollutant
HTPs	human toxicity potentials
IF	intake fraction (U.S. population)
ilF	individual intake fraction
LCA	life-cycle assessment
MACT	maximum available control technology
NTI	National Toxics Inventory
OH	hydroxyl radical
PAC	polycyclic aromatic compound
PAH	polycyclic aromatic hydrocarbon
PBH	persistent bioaccumulative toxic
PEFs	potency equivalency factors
POM	polycyclic organic matter
RSEI	U.S. EPA's Risk Screening Environmental Indicators
SAB-EEAC	U.S. EPA Science Advisory Board Environmental Economics Committee
SIC	standard industry classification
TCDD	tetrachlorodibenzo-*p*-dioxin
TEFs	toxic equivalency factors
TEQ-WHO98	toxic equivalent scheme of the World Health Organization
TLVs	threshold limit values
TRI	U.S. EPA's Toxic Release Inventory
VA	value added
VSL	value of a statistical life
WTP	willingness to pay

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