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Effects of heat recovery for district heating on waste incineration health impact: A simulation study in Northern Italy

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HIGHLIGHTS

- We estimated the health effect caused by the emission of PM_{10}, PCDD/F, PAH, Cd and Hg from a new waste incinerator.
- We considered the compensation in PM_{10} emission obtained through the activation of a district heating network.
- We found a moderate impact of the incinerator on human health for the exposed population.
- The switch-off of domestic boilers compensates for health impacts caused by PM_{10} emission from the incinerator.
- The degree of self-subsistence of the diet and the consumption habits contributes substantially to risk determination.

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ABSTRACT

The construction of waste incinerators in populated areas always causes substantial public concern. Since the heat from waste combustion can be recovered to power district heating networks and allows for the switch-off of domestic boilers in urbanized areas, predictive models for health assessment should also take into account the potential benefits of abating an important source of diffuse emission.
In this work, we simulated the dispersion of atmospheric pollutants from a waste incinerator under construction in Parma (Italy) into different environmental compartments and estimated the potential health effect of both criteria- (PM_{10}) and micro-pollutants (PCDD/F, PAH, Cd, Hg). We analyzed two emission scenarios, one considering only the new incinerator, and the other accounting for the potential decrease in pollutant concentrations due to the activation of a district heating network. We estimated the effect of uncertainty in parameter estimation on health risk through Monte Carlo simulations. In addition, we analyzed the robustness of health risk to alternative assumptions on: a) the geographical origins of the potentially contaminated food, and b) the dietary habits of the exposed population.
Our analysis showed that under the specific set of assumptions and emission scenarios explored in the present work: (i) the proposed waste incinerator plant appears to cause negligible harm to the resident population; (ii) despite the net increase in PM_{10} mass balance, ground-level concentration of fine particulate matter may be curbed by the activation of an extensive district heating system powered through waste combustion heat recovery and the concurrent switch-off of domestic/industrial heating boilers. In addition, our study showed that the health risk caused by waste incineration emissions is sensitive to assumptions about the typical diet of the resident population, and the geographical origins of food production.

1. Introduction

The estimation of health effects caused by the anthropogenic emission of atmospheric pollutants is a key issue in the debate on the sustainability of human activities. Human exposure to atmospheric pollution may occur not only through air inhalation, but also indirectly through ingestion of contaminated water, soil or food (Abrahams, 2002; Lippmann, 2009; Sweetman et al., 2000; WHO Europe, 2000).

A number of methodologies for assessing the direct and indirect effects on health caused by air pollution have been proposed by public health institutions, international research organizations and environmental agencies (Fryer et al., 2006; Rovira et al., 2012). The general approach, inspired by the scheme proposed by the National Academy of Sciences (NAS, 1983), is to track the diffusion of Chemicals Of Potential Concern (COPC) through different environmental media according to the so called "source–pathway–receptor" model (Fig. 1).

Widespread public concern is associated with the atmospheric emission of pollutants from municipal solid waste (MSW) incinerators. The epidemiologic literature on the health effects caused by the activity

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List of acronyms

COPC	Chemicals Of Potential Concern
CSF	Cancer Slope Factor
Db	domestic boilers
ERF	Exposure–Response Function
EIA	Environmental Impact Assessment
ES1/ES2	first/second emission scenario for PM10
ESM	Electronic Supplementary Material
FHItD	Full Home-grown Italian Diet
FHNAD	Full Home-grown North American Diet
FMItD	Full Mix Italian Diet
HHRAP	Human Health Risk Assessment Protocol
HI	Hazard Index
HMs	heavy metals
Hp	existing heating plant
HQ	Hazard Quotient
HRA	Health Risk Assessment
In	Incinerator
LADD	Lifetime Average Daily Dose
LCA	Life Cycle Assessment
MSW	Municipal Solid Waste
PHItD	Partial Home-grown Italian Diet
RfD	Reference Dose
RR	Relative Risk
Sb	supplementary gas boiler
TCR	Total Cancer Risk
TDI	Tolerable Daily Intake

Model parameters cited in the text

EF	exposure frequency
AT	averaging time
tD	lifetime of the incinerator
BvFOR	air–plant biotransfer factor for forage
BrAG	soil–plant biotransfer factor for aboveground vegetables
CrMILKagr	milk and dairy products consumption rate
CrAGPurb	aboveground protected vegetables consumption rate
FlocVEGurb	fraction of home-grown vegetable consumption, urban scenario
QpSILmilk	quantity of silage ingested by cows
BD	soil bulk density
ZsURB	soil accumulation depth

of MSW incinerators is extensive and well reviewed (Franchini et al., 2004; Porta et al., 2009; UK DEFRA, 2004; WHO Europe, 2007). An association between health risk and proximity to incinerators was documented in a number of epidemiological studies (Cordier et al., 2010; Ranzi et al., 2011; Viel et al., 2008). Nevertheless, while very informative, the results of these ex-post epidemiological studies cannot be easily extrapolated to estimate ex-ante the future health effects of alternative emission scenarios from proposed projects of modern waste incinerators. On the contrary, simulation studies are able to provide ex-ante estimates of the potential health effects of a proposed waste incinerator by: (i) linking stack emissions to the projected level of exposure of the resident population, and then (ii) by using pollutant-specific Exposure–Response Functions to quantitatively estimate the potential risk for human health.

Several simulation studies have recently estimated the health effects of pollutants from MSWI incinerators and other industrial plants (Cangialosi et al., 2008; Kim et al., 2011; Lonati and Zanoni, 2012; Morra et al., 2009; Roberts and Chen, 2006; Rovira et al., 2010). Nevertheless, while HRAs have been routinely performed in the last twenty years, there are still a number of issues that have not been thoroughly investigated in the literature.

First, it is not clear whether the potential health impacts caused by incinerator stack emissions can be partially compensated for by the reduction in atmospheric emissions achievable through district heating and the switch-off of a substantial number of domestic boilers (Rezaie and Rosen, 2012). It is critical to determine whether the choice to locate a waste incineration facility near a densely populated area may be justified by the benefits derived from heat recovery from waste incineration and the activation of extensive district heating.

Second, in standard HRAs it is often assumed that only food produced at the exposure location (i.e., home-grown at the residence address) is potentially contaminated by stack emissions (EPA, 2005a), while food of animal origin consumed by residents in urban areas is commonly assumed to be produced elsewhere and, as such, considered to be uncontaminated. Although this might be true in large urban settings, in many semi-urbanized areas “farmers' markets” selling local food products are becoming increasingly popular. As a consequence, it is important to assess whether people living in small- and medium-sized urban settings could be potentially affected by stack emissions by regularly eating food produced in the nearby countryside that may be potentially contaminated by waste incineration.

Third, previous risk assessment studies performed in Italy used the typical diet of North American (Cangialosi et al., 2008; Morra et al., 2006) or Spanish citizens (Lonati and Zanoni, 2012). However, the typical Italian diet is substantially different from the North American one (Da Silva et al., 2009), and therefore it is relevant to assess whether assuming the consumption of one diet or the other may change the outcome of HRA studies.

Here, we present the results of a preliminary analysis of a multi-compartment model to estimate the potential long-term consequences on human health of the operations of a MSW incinerator plant currently under construction in the city of Parma (Italy). The analysis was carried out specifically to investigate the effect on HRA of: (i) the activation of district heating fuelled by the MSW incinerator and the switch-off of domestic boilers, and (ii) different assumptions on dietary habits and geographical origin of production of food consumed by the resident population.

2. Materials and methods

HRA studies of incinerators emissions typically focus on two COPC classes (Roberts and Chen, 2006; Schuhmacher et al., 2004): (i) criteria pollutants (i.e., particulate matter, nitrogen oxides, etc.) and (ii) micropollutants (i.e., dioxins, heavy metals, etc.).

Different approaches have been developed to analyze these two categories of pollutants (Fig. 1), as described below. Here, we focused on health effects due to chronic exposure under the assumption that the waste incinerator plant under study will be well managed (and thus emissions will never exceed legal limits) and, consequently, the probability of acute short-term exposure to high level of pollutants will be negligible.

2.1. Health risk assessment for criteria pollutants

Criteria pollutants have short residence times in the atmosphere due to degradation reactions, and do not usually show bioaccumulation properties. Direct inhalation through contaminated air is usually considered the main pathway of exposure (WHO Europe, 2000, 2006).

The number E of new cases per unit time (case year^{-1}) caused by a projected increase in ground concentration of atmospheric pollutants (or the number of cases avoided due to a reduction in ground concentration of pollutants) was estimated by means of Exposure–Response Functions (ERFs), as described in previous studies (Forastiere et al., 2011; Kunzli et al., 2000; Martuzzi et al., 2006). Appendix D in the

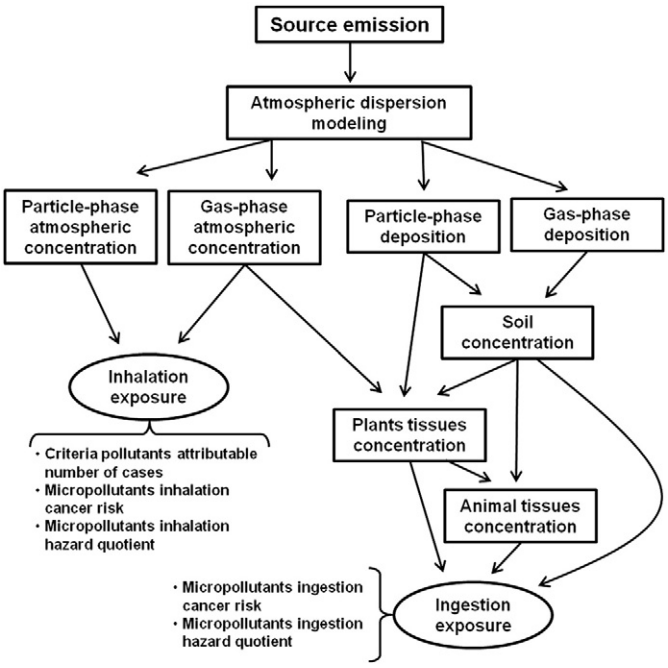

Fig. 1. Source–pathway–receptor model for the diffusion of Chemicals Of Potential Concern (COPC) through different environmental media.

Electronic Supplementary Material (ESM) reports in detail the methodology used in the present study.

2.2. Health risk assessment for micropollutants

Micropollutants generally have high persistence in the environment and may exhibit bioaccumulation properties. The most relevant exposure pathway for humans is ingestion through diet (Fries, 1995; Linares et al., 2010; Llobet et al. 2003; Sweetman et al., 2000), even if inhalation and other ways of exposure, like ingestion of contaminated soil and water or dermal contact, may be of interest in particular situations (EPA, 2005a; WHO Europe, 2000).

For this group of pollutants, we applied the model proposed by the United States Environmental Protection Agency (EPA) in the *Human Health Risk Assessment Protocol* (HHRAP) (EPA, 2005a). More details about the equations used for health risk calculation are reported in ESM Appendix D.

For carcinogen pollutants, such as dioxins (PCDD/F) polycyclic aromatic hydrocarbons (PAH), polychlorinated biphenyl (PCB) and some heavy metals (HMs), the EPA model (2005a) assumes there is no safe threshold dose below which there is no health risk. The health risk, measured as the probability of developing cancer during the entire lifetime (assumed to be 70 years), is estimated by multiplying the exposure dose by a Cancer Slope Factor (CSF), i.e. the estimate of the carcinogenic potency of the chemical. As a screening procedure, risks caused by different exposure pathways and carcinogen pollutants can be summed up to obtain the Total Cancer Risk (TCR).

For non-carcinogenic pollutants, a threshold dose is assumed to exist below which no appreciable health effects are expected. For each contaminant, the risk is computed in terms of Hazard Quotient (HQ), i.e. the ratio between the estimated daily dose and the reference dose (RfD). Accordingly, HQ > 1 implies that the reference dose is exceeded for a specific contaminant. HQs due to exposure to different toxic pollutants can be then combined to determine the overall Hazard Index (HI).

2.2.1. Diet composition and exposure scenarios

Health risk caused by micropollutants is strongly influenced by the extent to which the food consumed by the resident population is exposed to contaminants produced by the waste incinerator facility under investigation. While EPA (2005a, Section 4.2) lists a variety of exposure scenarios, here we focused our investigation effort on only two scenarios assuming that they characterize the exposure of the large majority of the resident population i.e.: (i) the "rural exposure" scenario, applied to people living in a rural area and (ii) the "urban exposure" scenario, applied to people living in urban areas.

The typical assumption in HRA studies is that people living in a specific location can only be exposed to micropollutants through the fraction of their food that is grown at home and that can be thus contaminated by stack emissions depending upon the distance from the MSW facility (Cangialosi et al., 2008; Lonati and Zanoni, 2012; Schuhmacher et al., 2004). The remaining fraction, i.e. the part that is not home grown, is assumed to be uncontaminated (Lonati and Zanoni, 2012; Schuhmacher et al., 2001). This assumption may underestimate the risk for people living in areas of low fall-out when part of their diet is supported by food produced in high fall-out areas. Bias in exposure estimations might also occur for farmed animals, as a considerable fraction of their forage, silage and grain may not be grown around the farm but in areas still potentially contaminated by waste incineration emissions.

To correct for this potential bias, in each cell in which the study area is discretized we proposed to explicitly account for: (i) the fraction *Floc* of residents' diet supported by home-grown food and (ii) the fraction *Favg* of diet supported by food that is not produced in the cell of residence but in other rural cells still under the influence of waste incineration emissions. We subsequently modified the equations for computing the daily intakes of micropollutants reported in the original EPA model (2005a), as described in detail in ESM Appendix D.

Note that $Floc + Favg \leq 1$, as a fraction of the food consumed locally could also be produced outside the study area, and therefore assumed to be uncontaminated by the emission source.

3. Case study

The HRA model was used to estimate the potential health effects of the activation of a new MSW incinerator in the city of Parma. This incinerator is currently under construction, and it is expected to be ready to operate in early 2013. The Environmental Impact Assessment (EIA) procedure was carried out in 2008 according to the European legislation (EU Directives 85/337/CEE and 97/11/CE), but no quantitative health risk assessment was ever performed. The emission data presented in the Environmental Impact Statement were used in our study to conduct the HRA. ArcView® 3.2 and ArcGIS® 9.1 were used for spatial elaborations, while the health risk assessment, as well as the Monte Carlo analyses, were run in MS Excel® 2007 through Visual Basic for Application programming.

3.1. The study area

The study area was a 16.75×20 km^2 rectangle centered on the city of Parma (Fig. 2), divided in a 250×250 m^2 regular cell grid, for a total of 5360 cells. For each cell, we determined the prevalent land use (i.e., agricultural, urban and surface water), the farming (ha cell^{-1}) and breeding (tons cell^{-1}) intensity and the number of residents using available information from the Cartographic Database of the Emilia Romagna Region (RER, 2012). For the very few cells in which the prevalent soil use was "surface water", we did not calculate the indirect risk for ingestion of contaminated soil or home-grown foods, nor did we assume exposure scenarios through contaminated fish as this is not relevant in this region.

The total population in the study area was 191,330 people. Information on the geographical distribution and the general age structure of the population was retrieved from the local registry office. Baseline incidences (I_0) for all causes of mortality, lung cancer, heart attack and stroke were respectively 1102.48, 64.79, 90.12 and 108.99 cases year^{-1} per every 100,000 dwellers (ASR-ER, 2007).

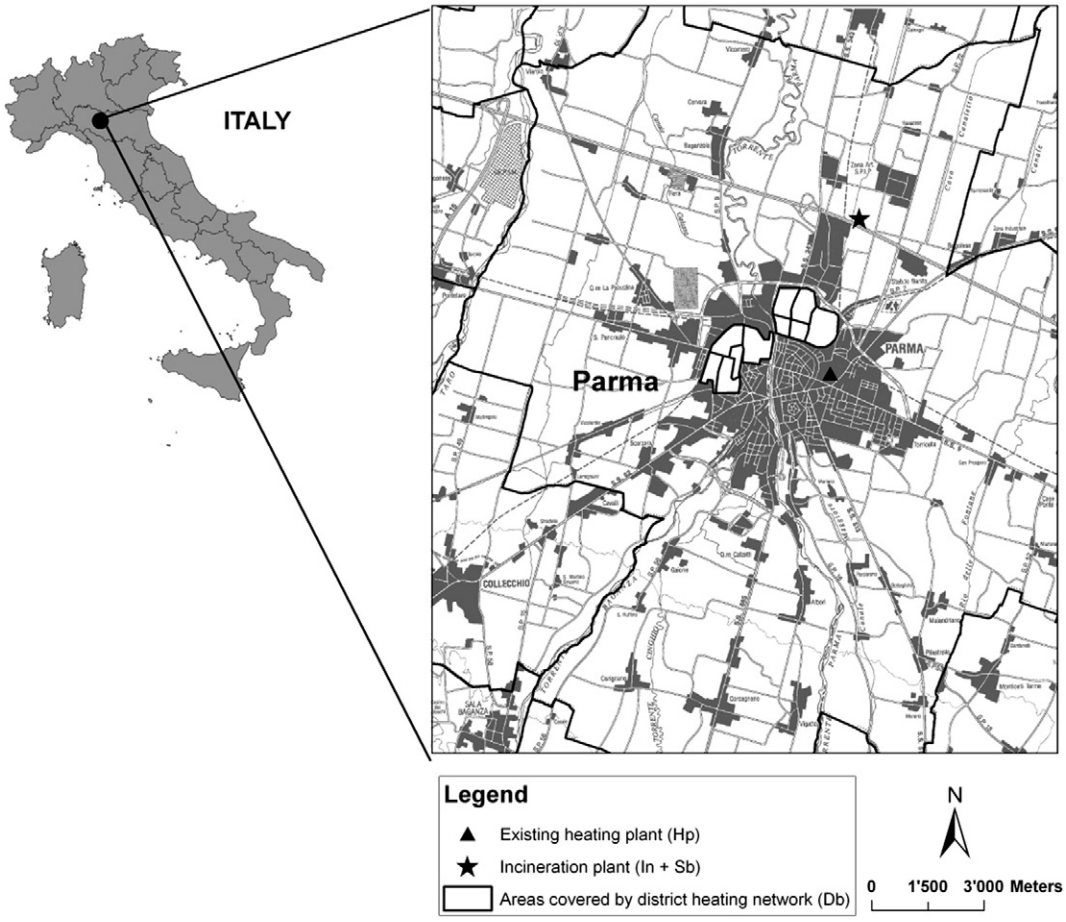

Fig. 2. Study area and location of the principal emission sources.

3.2. The waste incineration plant and the district heating network

The MSW incinerator is currently under construction in a northern area of the city of Parma, about 4 km away from the city center. When completed in 2013, it will have two grid furnaces, each one with a treating capacity of about 190 t day^{-1} of MSW and a maximum of 130,000 t $year^{-1}$. The plant is authorized to burn municipal solid wastes, sewage sludge, sanitary wastes and non-hazardous special wastes. A description of stack emissions is reported in ESM Table A.3 along with pollutant emission rates derived from expected pollutant concentrations and gas flow rate reported in the Environmental Impact Statement.

Heat recovered by waste incineration will supply a district heating network of about 20,000 equivalent inhabitants, distributed in ten residential districts of the city (Fig. 2). The estimated total amount of energy that will be provided in these districts by the heating network is 6.22×10^7 kWh $year^{-1}$.

3.3. Air quality simulations

We used the software WINDIMULA3® (Cirillo and Manzi, 1991; MAIND S.r.l, 2006) to model the atmospheric dispersion of pollutants. WINDIMULA3® is a multi-source Gaussian model that calculates deposition fluxes and allows the simulation of calm winds (i.e., wind speed <1 m s^{-1}), a condition frequently encountered in the study area, namely ca. 20% of annual data on hourly basis at the stack height. We used meteorological data relative to the annual period 15/10/2005–14/10/2006 provided by the Regional Environmental Protection Agency.

We analyzed two emission scenarios for criteria pollutants. In the first scenario (*ES1*), we considered only the new MSW incinerator (*In*) modeled as a point source. In the second scenario (*ES2*), we also considered the activation of the district heating network, the concurrent switch-off of domestic boilers (*Db*) as well as the reduction of the activity of a pre-existing district heating plant (*Hp*). In addition, we also considered the emissions of a supplementary gas boiler (*Sb*) to be constructed within the incineration plant.

Emissions under *ES2* scenario were analyzed on a seasonable basis, i.e. during the cold season (15 October–15 April) and during the warm season (16 April–14 October).

The emission balances (*EB*, mg $year^{-1}$) for the cold and warm seasons were:

$$EB_{cold} = +In + Sb - Db_{cold} - Hp \quad (1)$$

$$EB_{warm} = +In - Db_{warm}. \quad (2)$$

We quantified emissions from domestic boilers on the basis of the estimated energy consumption in the residential areas that will be supplied by district heating and assumed that this energy is produced through methane combustion (Energy Agency of Parma, *personal communication*). We used an emission factor of 24.1 mg kWh^{-1} for PM_{10} as reported for methane combustion in residential boilers (SNAP code 020202) in the national guidelines for emission inventories (ISPRA, 2012).

To account for seasonal changes in heat and water uses, we assumed that 20% of the total energy consumption is ascribed to hot water

production throughout the entire year and 80% imputable to house heating during the cold season only (ENEA, 2005).

The warm season emission rate (EF_{warm}, mg s^{-1}) for hot water production was assumed to be constant over the year and computed as follows:

$$EF_{warm} = Ehw_{tot}/(8760 \cdot 3600) \quad (3)$$

where Ehw_{tot} is the total emission due to hot water production (mg $year^{-1}$) and $8760 \cdot 3600$ is the number of seconds in a year.

The cold season emission rate (EF_{cold}, mg s^{-1}) was computed as:

$$EF_{cold} = Erh_{tot}/(4392 \cdot 3600) + Ehw_{tot}/(8760 \cdot 3600) \quad (4)$$

where Erh_{tot} is the total emission as a result of house heating (mg $year^{-1}$), and $4392 \cdot 3600$ is the number of seconds in the cold season.

The ten residential areas that will be connected to the new heating network (Fig. 2) were aggregated for modeling purposes into two circular areas of equivalent surface and treated in the second emission scenario (*ES2*) as distributed pollution sources: the pollutant concentration from these two sources was subtracted from that derived from the incinerator. Their emission height was assumed to be 15 m (ESM Table A.4).

3.4. Case study COPC

There is no general consensus on ERFs for chronic health effect of pollutants other than PM_{10}, e.g. NO_x and SO_x. In fact, PM_{10} concentrations generally exhibit a very high correlation with these criteria pollutants. Therefore, in epidemiological studies it may be particularly problematic to disentangle the health effect of SO_x and NO_x from that of PM_{10} (Torfs et al., 2007; WHO, 2003). As a consequence, HRA for criteria pollutants was performed only with reference to primary PM_{10} emissions so as to avoid double counting.

The evidence for an independent effect of tropospheric ozone (O_3) is stronger. However, O_3 modeling requires the use of more complex photochemical models, and this was beyond the scope of the present work.

Table A.1 in the ESM reports the ERFs for PM_{10}. We noted that the relative risks for some health outcomes (i.e. stroke, acute bronchitis and asthma) are not statistically significant. However, since the central estimate suggests that an effect of PM_{10} on those outcomes is indeed possible, we decided to use these ERFs in our RHA consistently with a precautionary approach. For ERFs relative to $PM_{2.5}$, a ratio $PM_{2.5}/PM_{10}$ of 0.7 was assumed according to Medina et al. (2005).

As for micropollutants, we modeled PCDD/F (as equivalent 2,3,7,8-tetrachloro-dibenzo-p-dioxin), PAH (as benzo[a]pyrene), mercury (as Hg^0, Hg^{2+} and methyl-Hg) and cadmium (as the sum of Cd + Tl authorized emissions).

We followed EPA (2005a) guidelines for the partitioning of different pollutants between the gas and particle phase (ESM, Table A.5). Mercury was assumed to be released as Hg^0 and $HgCl_2$ and 2% of deposited mercury was assumed to speciate to Methyl-Hg in soil (EPA, 2005a).

The CSFs and RfDs values reported in ESM Table A.2 were derived from the Integrated Risk Information System (IRIS) (EPA, 2008) and the Risk Assessment Information System (RAIS) (Oak Ridge National Laboratory, 2008).

ESM Table A.6 lists the values chosen for the other parameters of the model.

For PM_{10} we modeled both scenarios *ES1* and *ES2*, while micropollutants were modeled only for scenario *ES1*, as domestic methane boilers do not represent a relevant source for these chemicals.

3.5. Dietary habits

It is well known that food consumption may change substantially depending upon regional habits and local culinary traditions, and this might significantly affect the potential intake of environmental pollutants from food (Undeman et al., 2010). The North American diet in particular is known to be quite different from the typical Italian diet (Da Silva et al., 2009). We thus investigated whether alternative assumptions on diet composition may significantly affect health risk assessment.

Since detailed dietary data were not available for the province of Parma, we derived the average Italian diet composition from Turrini et al. (1991) on the basis of nation-wide food consumption data. According to the inclusion criteria presented in EPA (1997), food items were grouped in the eight food categories used in the EPA model (EPA, 2005a). When needed, we derived dry weight consumption values by correcting Turrini et al.'s data (1991) for water content on the basis of food composition tables developed by the Italian Institute for Research on Foods and Nutrition (INRAN, 2012). Average body weight for adults was set to 70 kg as in Walpole et al. (2012). No preparing and cooking losses were considered.

Health risk was first estimated for the Italian diet under the following assumptions:

- all food consumed by resident people was produced in the study area and, thus, potentially contaminated (i.e., $Floc + Favg = 1$);
- in "rural" cells 100% of vegetables in the residents' diet were home-grown ($FlocVEG = 1$) while only 50% of animal products were home-grown ($FlocANI = 0.5$) and the other 50% were produced elsewhere within the study area ($FavgANI = 0.5$);
- in "urban" cells 50% of vegetables were home-grown ($FlocVEG = 0.5$, $FavgVEG = 0.5$) and 100% of animal products came from the study area ($FavgANI = 1$)
- 50% of livestock's diet came from the cell, the other 50% from the study area.

We named this scenario "Partial Home-grown Italian Diet" (PHItD) and used it as our reference exposure scenario.

In order to test the sensitivity of model results to alternative assumptions of food origin and diet type, we also computed health risk for the following exposure scenarios (ESM, Table C.2):

- "Full Homegrown North American Diet" (FHNAD): EPA (2005a) standard North American diet, with the dietary consumption for "rural" cells exclusively supported by food produced in the same cell of residence and no consumption of contaminated animal products in "urban" cells;
- "Full Homegrown Italian Diet" (FHItD): as above but with the Italian diet;
- "Full Mixed Italian Diet" (FMItD): Italian diet under the extreme assumption that the dietary consumption is entirely supported by a perfect mix of food produced within the overall study area. Under this assumption, the same food contamination level applies to all the cells of the study area

3.6. Sensitivity analysis through Monte Carlo simulations

The results of a health risk assessment depend upon a large number of assumptions on processes and model parameterization. Therefore, we carried out a Monte Carlo analysis to assess what model parameters health risk is most sensitive to (Hwong-Wen, 2002; Lonati and Zanoni, 2012; Schuhmacher et al., 2001).

We implemented the "Tier 2" methodology presented in EPA (2001) as follows: first, for each model parameter we defined an uninformative uniform probability distribution over a range of ±50% the mean value reported in ESM Table A.6. We then drew a parameter value from each respective probability distribution and estimated the corresponding health risk in each of the 67 × 80 cells in which the study area was

Table 1
Results of atmospheric dispersion modeling for PM_{10}. Minimum, median and maximum over the study area for mean atmospheric concentrations of each period.

Averaging period	Statistic over the area	Mean PM_{10} concentration at ground level ($\mu g\ m^{-3}$) for each source			
		In	Sb	Db	Hp
Cold period	Minimum	4.2×10^{-4}	4.3×10^{-5}	1.7×10^{-3}	3.4×10^{-5}
		2.1×10^{-3}	2.6×10^{-4}	1.1×10^{-2}	1.4×10^{-4}
	Median				
	Maximum	2.3×10^{-2}	3.9×10^{-3}	7.6×10^{-1}	8.9×10^{-3}
Warm period	Minimum	3.8×10^{-4}	n.a.	6.4×10^{-5}	n.a.
	Median	1.9×10^{-3}	n.a.	6.0×10^{-4}	n.a.
	Maximum	1.8×10^{-2}	n.a.	4.4×10^{-2}	n.a.

Legend: In = incinerator, Sb = supplementary boilers, Db = domestic boilers, and Hp = existing heating plant. (n.a. = not active).

discretized. We replicated this process 10,000 times. For each cell we derived six percentiles of the distribution of risk (i.e., 2.5, 25, 50, 75, 97.5 and 99), and for each percentile we reported the average and maximum value over the entire area.

To determine which parameters mostly affected health risk, we computed for each model parameter j the Spearman rank correlation coefficients ρ_j between the 10,000 casually extracted values and the corresponding average health risk over the studied domain. ρ_j were then squared and normalized so as to sum to 1 and then ranked from the largest to the smallest. Thus, each coefficient represents the relative contribution of each input parameter to the total variance of the average risk of the area (EPA, 2001; Hwong-Wen, 2002).

To assess whether the resulting ranking was strongly affected by the shape of the probability distribution for model parameters, we re-ran the sensitivity analysis also by using beta distributions (shape parameters $a=4$, $b=5$) instead of uniform ones.

The Monte Carlo analysis was carried out only for the indirect risk of ingestion of micropollutants, as preliminary sensitivity analyses showed that inhalation caused only a very small incremental risk.

4. Results

4.1. Air quality simulation

Air quality simulations (Table 1, ESM Fig. B.1–B.3) show that the emission sources analyzed in the present study provide a very small contribution to the observed annual mean concentration of PM_{10} in the study area, i.e. about 40 $\mu g\ m^{-3}$ (APAT, 2008). The incinerator (*I*) and the domestic boilers (*Db*) are the most important emission sources, with maximum values of the annual mean of modeled 1-hour concentrations – equal to 0.02 and 0.40 $\mu g\ m^{-3}$, respectively — expected to occur close to the emission sources. In terms of mass balance (ESM Table A.7), the activation of the new incinerator increases PM_{10} input in the atmosphere (+0.71 and +1.74 tons $year^{-1}$ during the cold and warm seasons, respectively), even when considering the switching-off of the domestic gas boilers and the activation of the district heating network (Scenario *ES2*). Nevertheless, in the cold season (Table 1, ESM Fig. B.4) the contribution of domestic boilers to the mean annual atmospheric concentration at ground level is expected to be an order of magnitude higher than PM_{10} concentration due to the other emission sources, including stack emissions from incinerators. As a consequence, the full activation of district heating powered through heat recovery from the incinerator results in a general reduction in atmospheric concentrations at ground level (max reduction over the study area: $-0.76\ \mu g\ m^{-3}$) during the cold season. In the warm season, a small increase in concentrations is expected in a large part of the study area (max increase over the study area: $+0.01\ \mu g\ m^{-3}$) except for the city center of Parma, where a reduction of atmospheric concentrations is still expected (max reduction over the study area: $-0.04\ \mu g\ m^{-3}$) due to the switching-off of domestic boilers for the production of hot water (Table 1, ESM Fig. B.5).

Average annual concentration at ground level and cumulative annual deposition for micropollutants are reported in Table 2. Maximum fall-out concentrations are expected in less populated areas close to the waste incinerator plant, which is the only source of micropollutants analyzed in the present analysis. Maximum concentrations calculated by the model are one to four orders of magnitude below international guideline values for air quality (Table 2).

4.2. Health effect due to PM_{10}

The activation of the new incinerator alone (emission scenario *ES1*) is expected to cause a marginal increase in mortality and morbidity in the exposed population due to the increase in PM_{10} chronic exposure (Table 3). For general mortality, 1.6×10^{-2} additional cases $year^{-1}$ (95%CI: $5.3\times10^{-3} \div 2.9\times10^{-2}$) on a population of 191,330 exposed residents are expected, i.e. a +0.001% increase in the expected annual number of deaths.

The activation of the district heating network powered by the incinerator (emission scenario *ES2*) is expected to reduce atmospheric PM_{10} concentrations in some populated areas in the center of the city (ESM Fig. B.4–B.5), resulting in an overall reduction, albeit small, in mortality and morbidity (Table 3). For general mortality, the model showed a reduction of 5.1×10^{-1} cases $year^{-1}$ (CI 95%: $-1.7\times10^{-1} \div -9.3\times10^{-1}$), namely ca. 10 cases less over 20 years, on the entire population (−0.024% in annual number of deaths).

4.3. Health effect due to micropollutants

The analysis of the Italian reference diet shows great differences with respect to the standard EPA (2005a) farmer consumption profile. As shown in ESM Table C.1, vegetable consumption in the average

Table 2
Results of atmospheric dispersion modeling for micropollutants. Minimum, median and maximum on the study area for mean annual atmospheric concentrations and cumulative annual deposition fluxes. For concentrations, available reference values for health protection are shown.

Model output	Statistic over the area	*Chemical of potential concern* (*COPC*)				
		PCDD	Cd	PAH	Hg^{0}[a]	Hg^{2+}
Mean annual concentration at ground level ($\mu g\ m^{-3}$)	Minimum	6.5×10^{-12}	1.1×10^{-5}	6.4×10^{-7}	2.2×10^{-6}	8.6×10^{-6}
	Median	3.1×10^{-11}	5.1×10^{-5}	3.0×10^{-6}	1.0×10^{-5}	4.1×10^{-5}
	Maximum	2.8×10^{-10}	4.6×10^{-4}	2.7×10^{-5}	9.2×10^{-5}	3.7×10^{-4}
	Reference value	3.0×10^{-7}[b]	5.0×10^{-3}[c]	1.0×10^{-3}[c]	1.0[b]	
Cumulative annual deposition ($mg\ m^{-2}\ anno^{-1}$)	Minimum	1.1×10^{-9}	1.1×10^{-4}	1.3×10^{-4}	2.6×10^{-4}	1.4×10^{-3}
	Median	1.0×10^{-8}	1.5×10^{-3}	9.5×10^{-4}	1.6×10^{-3}	1.0×10^{-2}
	Maximum	1.4×10^{-7}	1.8×10^{-1}	1.4×10^{-2}	3.0×10^{-2}	3.8×10^{-1}

[a] For Hg^{0} only the gas phase is considered.
[b] WHO Europe (2000).
[c] EU Directive 2004/107/CE.

Table 3
Health effects from exposure to PM_{10} for emission scenarios ES1 (incinerator only) and ES2 (incinerator and district heating), computed as a sum over the entire area (95% CI in parenthesis). For ES2, the resulting number represents the net balance between the expected reduction of cases in the areas of reduced exposure and the expected increase in the areas of increased exposure.

Health outcome [units]	Scenario ES1	Scenario ES2
All causes mortality [case $year^{-1}$]	1.6×10^{-2} (5.3×10^{-3}; 2.9×10^{-2})	-5.1×10^{-1} (-1.7×10^{-1};-9.3×10^{-1})
Lung cancer [case $year^{-1}$]	1.2×10^{-3} (1.5×10^{-4}; 2.5×10^{-3})	-4.0×10^{-2} (-5.0×10^{-3}; -8.0×10^{-2})
Infraction [case $year^{-1}$]	3.9×10^{-3} ($3.0\times10-3$; 4.9×10^{-3})	-1.2×10^{-1} (-9.7×10^{-2}; -1.6×10^{-1})
Stroke [case $year^{-1}$]	5.2×10^{-4} ($-1.3\times10-3$; 2.6×10^{-3})	-1.7×10^{-2} (4.2×10^{-2}; -8.4×10^{-2})
Acute bronchitis [case $year^{-1}$]	9.8×10^{-3} (-7.0×10^{-4}; 2.0×10^{-2})	-3.2×10^{-1} (2.3×10^{-2}; -6.4×10^{-1})
Asthma in children, <15 years of age [extra days of bronchodilator usage $year^{-1}$]	6.4×10^{-1} (-2.5; 3.8)	-2.1×10^{1} (7.9×10^{1}; -1.2×10^{2})
Asthma in adults, ≥15 years of age [extra days of bronchodilator usage $year^{-1}$]	3.7×10^{1} (-3.7×10^{1}; 1.1×10^{2})	-1.2×10^{3} (1.2×10^{3}; -3.6×10^{3})
Restricted Activity Days, 15–64 years of age [num.$year^{-1}$]	1.9×10^{1} (1.7×10^{1}; 2.2×10^{1})	-6.2×10^{2} (-5.4×10^{2}; -6.9×10^{2})
Work Lost Days, 15–64 years of age [num.$year^{-1}$]	4.7 (4.0; 5.4)	-1.5×10^{2} (-1.3×10^{2}; -1.8×10^{2})
Minor Restricted Activity Days, 18–64 years of age [num.$year^{-1}$]	1.2×10^{1} (1.0×10^{1}; 1.5×10^{1})	-4.0×10^{2} (-3.2×10^{2}; -4.7×10^{2})
Lower Respiratory Symptoms, 5–14 years of age [extra days $year^{-1}$]	6.6 (3.3; 9.8)	-2.1×10^{2} (-1.1×10^{2}; -3.2×10^{2})
Lower Respiratory Symptoms, >15 years of age [extra days $year^{-1}$]	5.3×10^{1} (6.1; 9.9×10^{1})	-1.7×10^{3} (-2.0×10^{2}; -3.2×10^{3})

Italian diet is three times greater than what EPA's suggest for North American diet, while animal products consumption is three times lower.

Detailed results of health risk estimation for the reference *PHItD* exposure scenario are reported in Table 4 and Fig. 3, while risk assessment under alternative assumptions of diet composition and food origins are presented in ESM Fig. C.1.

The maximum value of lifetime average daily dose (LADD) for PCDD/F over the area is 2.5×10^{-3} pg-TEQ kg_{bw}^{-1} day^{-1}, i.e. three order of magnitude smaller than the Tolerable Daily Intake (TDI) for this contaminant, i.e. 1–4 pg-TEQ kg_{bw}^{-1} day^{-1} (Van Leeuwen et al., 2000).

Food of animal origin represents the principal exposure pathway to PAH, PCDD/F and Hg^{2+}, accounting respectively for 97%, 83% and 82% of the total dose, as a mean over the area (Fig. 4). Exposure to Cd and MeHg, is primarily through the consumption of vegetales (respectively 82% and 61% of the total dose). The importance of soil ingestion for health risk is negligible. Inhalation is the only exposure pathway for Hg^0, and also plays an important role in exposure to Cd (16% of the dose).

The maximum value of total risk for carcinogens (TCR) over the entire area is 4.1×10^{-6} (Table 4, Fig. 3). By combining the spatial distribution of resident population with that of health risk, a total of 0.29 new cases in 70 years attributable to incinerator emissions should be expected in the entire study area. Ingestion risk is strongly determined by the effect of PAH, while inhalation risk is mostly attributable to Cd.

The total Hazard Index (HI) for toxic pollutants reaches its maximum, i.e. 7.3% of the RfD, close to the incinerator, with a median over the entire study area of about 0.3% (Table 4, Fig. 3). For 94% of the population the HI value is below 1% of RfD. On average, over the entire study area, ingestion of MeHg and Hg^{2+} represents the most important contribution to total HI.

4.3.1. Health risk sensitivity to parameter value

The Monte Carlo analysis highlights a substantial variability in risk for micropollutants. The 2.5th and 99th percentiles of the maximum TCR over the study area span over almost two orders of magnitude between 3.3×10^{-7} and 1.1×10^{-5}, respectively (Table 5). Furthermore, by accounting for population distribution in the study area, the resulting cumulative number of cases expected in 70 years ranges between 0.03 (2.5th percentile) and 0.65 (99th percentile).

For toxic pollutants, the maximum HI over the study area never exceeds 1 and ranges between 0.5% (2.5th percentile) and 15.2% (99th percentile). The 99th percentile of the Monte Carlo simulations for maximum HQs over the area equals to 6% for Cd, 5% for Hg^{2+} and 4% for MeHg. Simulations performed using beta distributions of model parameters provided very similar results and were reported in ESM Table A.8.

The Monte Carlo analysis demonstrates that only a few parameters (between 7 and 12 over more than 90) explain the majority of the variability in health risk from micropollutant ingestion (Table 6). Health risk is mostly sensitive to the parameters defining the timing of exposure, such as the exposure frequency (*EF*, 16–43% of variance), the averaging time for carcinogenic effects (*AT*, 18–14%) and the emission duration (*tD*, 2–10% of variance). Other influential parameters are the toxicological reference values (*CSF*: 12–18%; *RfD*: 17–21%), biotransfer factors between different compartments (e.g., *BvFOR*: 6–11%; *BrAG*: 13%) and parameters related to the food consumption for both humans (e.g., *CrMILKagr*: 2–8%; *CrAGPurb*: 3%; *FlocVEGurb*: 9%) and animals (e.g., *QpSILmilk*: 7–12%). Finally, parameters that determine the initial conversion from atmospheric deposition values to soil concentrations, such as the soil bulk density (*BD*, 2–14%) and soil mixing depth (*ZsURB*, 6%), play an important role.

5. Discussion

5.1. Health effects of PM_{10}

The increase in mortality due to primary PM_{10} from the incinerator (scenario *ES1*) represents a very small fraction of expected number of deaths in the area (<0.01%). Similar results were reported in other simulation studies (Forastiere et al., 2011; Roberts and Chen, 2006; Schuhmacher et al., 2004). Nevertheless, as the annual average of daily concentration of PM_{10} recorded by monitoring stations in the city of Parma in the year 2006 was about 40 μg m^{-3} (APAT, 2008), i.e., the annual limit for the protection of human health as regulated by the Italian law, efforts should be made to reduce additional sources of exposure in the area. The *ES2* scenario shows that the negative effects caused by the small increase in PM_{10} concentration due to the new incinerator can be offset by the positive effects due to the switching-off of domestic boilers and the activation of a large district heating network powered by heat recovery from the incinerator.

To our knowledge, this is the first study that provides quantitative estimates of the potential benefits of thermal energy recovery from incinerators for district heating in terms of reduced human health risks at the local spatial scale. Previous Life Cycle Assessment (LCA) studies have assumed that energy recovery from waste combustion can compensate for the electricity produced by fossil-fuel power plants. For example, Morselli et al. (2008) estimated the health impact of the entire Regional incineration system through LCA, accounting for the health benefit deriving from energy recovery and avoided emissions. However, these LCA analyses are based purely on emission mass balance and, therefore, cannot explicitly account for the expected ground level concentration of pollutants in conjunction with the actual population distribution. Our study shows that in terms of mass balance, the increase in PM_{10} emissions due to the activation of the incinerator is not compensated for by the switching-off of the existing emission sources (ESM Table A.7). Yet, when accounting for both atmospheric dispersion

Table 4
Doses and health risks for micropollutants under the Partial Home-grown Italian Diet (PHItD) scenario. Median and (maximum) on the study area are shown. Number of cases is computed only for carcinogens, as a sum over the entire area.

COPC	Dose (mg kg^{-1} day^{-1})		Risk for carcinogens (adimensional)			Hazard Quotient for toxics (adimensional)	
	Ingestion	Inhalation	Ingestion	Inhalation	Number of cases	Ingestion	Inhalation
PCDD	9.4×10^{-13} (2.5×10^{-12})	4.8×10^{-15} (4.3×10^{-14})	7.7×10^{-8} (2.1×10^{-7})	5.5×10^{-10} (5.0×10^{-9})	1.6×10^{-2}		
PAH	3.5×10^{-7} (8.5×10^{-7})	4.7×10^{-10} (4.2×10^{-9})	1.4×10^{-6} (3.4×10^{-6})	1.8×10^{-9} (1.6×10^{-8})	2.7×10^{-1}		
Cd	7.7×10^{-8} (1.3×10^{-5})	8.0×10^{-9} (7.2×10^{-8})		5.0×10^{-8} (4.5×10^{-7})	1.2×10^{-2}	7.4×10^{-5} (1.2×10^{-2})	
Hg0		1.6×10^{-9} (1.4×10^{-8})					3.3×10^{-5} (2.9×10^{-4})
Hg^{2+}	4.9×10^{-7} (3.7×10^{-6})	6.3×10^{-9} (5.7×10^{-8})				1.6×10^{-3} (1.2×10^{-2})	3.5×10^{-5} (3.2×10^{-4})
MeHg	1.3×10^{-7} (5.4×10^{-6})					1.3×10^{-3} (5.2×10^{-2})	
Total			1.5×10^{-6} (3.6×10^{-6})	5.3×10^{-8} (4.7×10^{-7})	2.9×10^{-1}	3.0×10^{-3} (7.3×10^{-2})	6.8×10^{-5} (6.1×10^{-4})

and population distribution, the increase in ground level concentrations of PM_{10} due to waste incineration is more than compensated by the reduction in PM_{10} concentration as a result of switching-off of domestic boilers achievable through district heating. Therefore, heat recovery for district heating is a key factor to effectively curb the environmental burden of a new waste incineration facility. Accordingly, efforts should be made to recover as much energy as possible, extend the district heating network so as to switch-off the largest number of domestic and non-domestic boilers. Additional benefits in terms of electricity consumptions could be obtained by using the district heating network also for air conditioning during the hot season.

5.2. Health effects of micropollutants

According to air quality simulations, the expected contribution of the MSW plant to long-term atmospheric concentrations of micropollutants at ground level is almost negligible when compared to international limits or guidelines (Table 2). Health risks computed through the use of the HHRAP model (EPA, 2005a) are also moderate (Table 4). The maximum value for TCR estimated in the area (i.e., 4.1×10^{-6}) is within the acceptable risk range (i.e., 10^{-6} to 10^{-5}) recommended by EPA (1998) for exposure to emissions from a single facility. Furthermore, when accounting for population distribution, less than 1 new mortality case due to waste incineration is expected over a lifetime. The maximum value for the total HI for toxics (i.e., 7.3% of the RfD) is smaller than the reference value of 25% recommended by EPA (1998) to account also for potential background exposures. These results derive from a set of conservative (i.e. protective) assumptions. For example, we assumed that the food consumed by residents, either purchased in the markets or home-grown, was in some way all contaminated.

The 99-th percentiles derived from the Monte Carlo simulation (Table 5) for (i) maximum TCR (i.e., 1.1×10^{-5}), (ii) number of lifetime

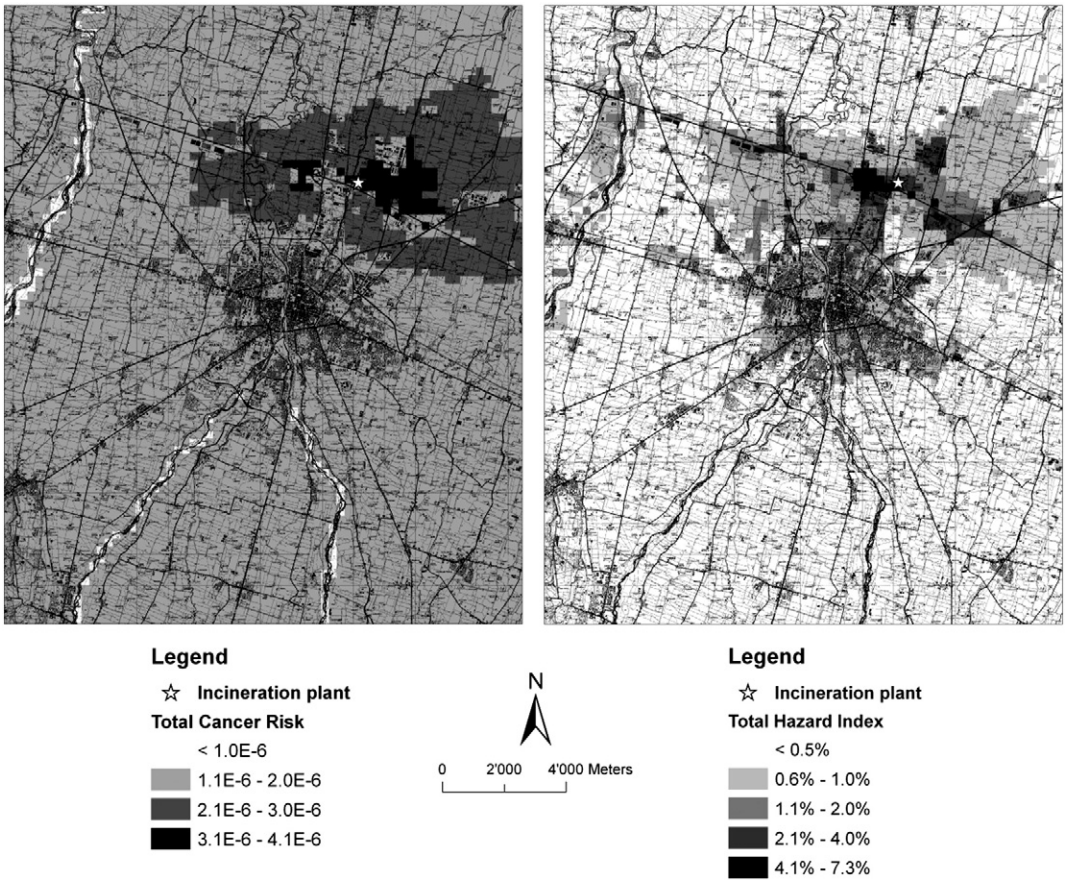

Fig. 3. Spatial distribution of Total Risk for Carcinogens (sx) and total Hazard Index (dx) over the study area.

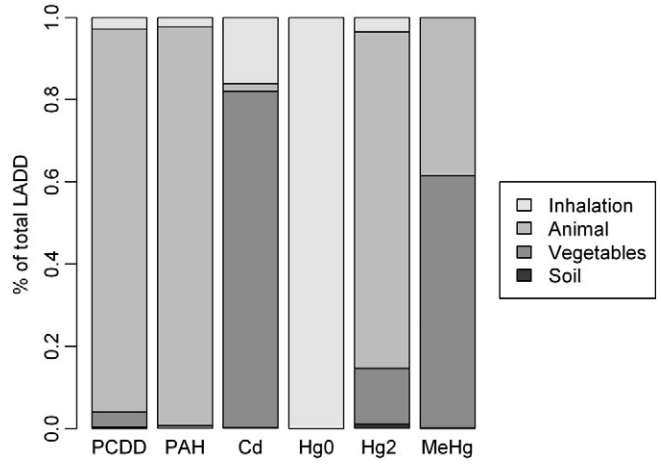

Fig. 4. Relative contribution (average over the study area) of different exposure pathways (inhalation and ingestion of food of animal origin, vegetables and resuspended soil) to the total dose.

cancer cases over 70 years (i.e. 0.65) and (iii) maximum HI (i.e. 15%) confirm that, even in the worst-case scenario, the health impact of the incinerator is expected to be moderate.

These results are in general agreement with those from other recent studies on health risk assessment for point emission sources that used a similar methodology (Cangialosi et al., 2008; Lonati and Zanoni, 2012; Roberts and Chen, 2006), although there are differences in the types of pollutants and exposure routes considered between our study and the ones cited above.

5.3. Dietary habits

Our analysis shows that the Italian reference diet derived from Turrini et al. (1991) data is quite different from the standard "reasonable maximum exposure" consumption profile suggested by EPA (2005a) (ESM Table C.1). The imbalance between consumptions of foods of vegetable and animal origin is in line with data presented in other available datasets on national dietary habits (WHO, 2006). Data from Turrini et al. (1991) used to derive the Italian diet profile were the only data sufficiently detailed to be grouped in the eight food categories used in the EPA model (EPA, 2005a). A comparison of aggregated consumption from Turrini et al. (1991) combined with the results of a more recent Italian survey (Leclercq et al.,

Table 5
Lifetime cancer risks and Hazard Indexes for micropollutants computed with Monte Carlo simulation. Median and (maximum) values on the study area are shown for six cut-off of the output risk distribution. Risk values are cumulated by type of effect and exposure pathway.

COPC category	Exposure pathway	Percentile on risk distribution					
		2.5	25	50	75	97.5	99
Carcinogens (risk)	Ingestion	1.3×10^{-7} (2.7×10^{-7})	3.4×10^{-7} (8.1×10^{-7})	5.7×10^{-7} (1.5×10^{-6})	9.6×10^{-7} (2.6×10^{-6})	2.4×10^{-6} (7.7×10^{-6})	3.1×10^{-6} (1.0×10^{-5})
	Inhalation	7.4×10^{-9} (6.6×10^{-8})	1.7×10^{-8} (1.5×10^{-7})	2.6×10^{-8} (2.3×10^{-7})	3.9×10^{-8} (3.5×10^{-7})	8.1×10^{-8} (7.3×10^{-7})	9.7×10^{-8} (8.8×10^{-7})
	Total	1.3×10^{-7} (3.3×10^{-7})	3.6×10^{-7} (9.5×10^{-7})	6.0×10^{-7} (1.7×10^{-6})	1.0×10^{-6} (3.0×10^{-6})	2.5×10^{-6} (8.4×10^{-6})	3.2×10^{-6} (1.1×10^{-5})
Carcinogens (no. of cases)	Total	3.0×10^{-2}	7.0×10^{-2}	1.2×10^{-1}	2.0×10^{-1}	5.1×10^{-1}	6.5×10^{-1}
Toxics (Hazard Quotients)	Ingestion	7.6×10^{-4} (5.1×10^{-3})	1.6×10^{-3} (1.5×10^{-2})	2.3×10^{-3} (2.6×10^{-2})	3.4×10^{-3} (4.4×10^{-2})	7.0×10^{-3} (1.2×10^{-1})	8.4×10^{-3} (1.5×10^{-1})
	Inhalation	2.0×10^{-5} (1.8×10^{-4})	3.5×10^{-5} (3.1×10^{-4})	4.7×10^{-5} (4.2×10^{-4})	6.3×10^{-5} (5.7×10^{-4})	1.1×10^{-4} (9.8×10^{-4})	1.2×10^{-4} (1.1×10^{-3})
	Total	7.9×10^{-4} (5.2×10^{-3})	1.6×10^{-3} (1.5×10^{-2})	2.4×10^{-3} (2.6×10^{-2})	3.5×10^{-3} (4.5×10^{-2})	7.1×10^{-3} (1.2×10^{-1})	8.5×10^{-3} (1.5×10^{-1})

Table 6
Results of probabilistic sensitivity analysis for ingestion risk. The contribution of each parameter to the total variance of the average risk over the area is shown in parenthesis. Only parameters with a contribution > 1% are shown, sorted by their relevance.

Chemical of potential concern (COPC)				
PCDD	PAH	Cd	Hg^{2+}	MeHg
AT (−18%)	EF (19%)	EF (19%)	EF (26%)	EF (43%)
CSF (18%)	AT (−14%)	RfD (−17%)	RfD (−21%)	VgSIL (17%)
EF (16%)	CSF (12%)	BD (−14%)	VgSIL (11%)	QpSILmilk (12%)
tD (10%)	BvFOR (11%)	BrAG (13%)	BvSIL (10%)	CrMILKagr (8%)
BvFOR (6%)	tD (9%)	FlocVEGurb (9%)	QpSILmilk (7%)	QpSILbeef (2%)
VgFOR (6%)	VgFOR (9%)	ZsURB (−6%)	BaMILK (5%)	BD (−2%)
BaBEEF (4%)	BaMILK (5%)	tD (4%)	CrMILKagr (2%)	tD (2%)
BaMILK (4%)	BaBEEF (4%)	CrAGPurb (3%)	tD (2%)	
QpFORmilk (3%)	CrMILKagr (4%)	ZsAGR (−3%)	ZsAGR (−2%)	
QpFORbeef (2%)	QpFORbeef (3%)	Ks (−2%)		
CrBEEFagr (2%)		CrAGPagr (2%)		
BD (−2%)				

Legend: AT — averaging time; BaBEEF — biotransfer factor, beef; BaMILK — biotrasfer factor, milk; BD — soil density; BrAG — soil–plant bioconcentration factor, aboveground vegetables; BvFOR — air–plant bioconcentration factor, forage; BvSIL — air–plant bioconcentration factor, silage; CrAGPagr — aboveground protected vegetable ingestion rate, farmer scenario; CrBEEFagr — beef ingestion rate, farmer scenario; CrMILKagr — milk ingestion rate, farmer scenario; EF — exposition frequency; FlocVEGurb = fraction of vegetables from the cell, urban scenario; Ks — soil loss constant; QpFORbeef — quantity of forage ingested, beef; QpFORmilk — quantity of forage ingested, milk; QpSILbeef — quantity of silage ingested, beef; QpSILmilk — quantity of silage ingested, milk; RfD — reference dose; CSF — Cancer Slope Factor; tD — time of combustion; VgFOR — empirical correction factor, forage; VgSIL — empirical correction factor, silage; ZsURB — Soil mixing depth, urban scenario.

2009) shows no appreciable differences. Another recent study confirmed that food consumptions did not vary significantly from 1991 to 2006 in Northern Italy (Pelucchi et al., 2010). While more detailed surveys of diet in the Parma province are encouraged, we are confident that the diet profile we derived by using Turrini et al. (1991) provides a fairer representation of the local diet than the one presented in EPA (2005a).

Our sensitivity analysis shows that dietary choices might have relevant implications in terms of exposure to environmental contaminants (ESM, Appendix C). The more vegetarian profile of the Italian diet with respect to the North American one (EPA, 2005a) notably reduces health risk caused by carcinogens, as the exposure to PCDD/F and PAH is primarily through ingestion of animal food. As for toxic pollutants, the FHItD and FHNAD exposure scenarios provide similar results, since there is a compensation between reduced exposure to Hg^{2+} (primarily via animal food) and increased Cd and MeHg exposure (primarily via vegetables).

The assumption in the PHItD reference scenario that the fraction of food purchased in local markets has a contamination given by a weighted average of contamination over the whole study area (ESM, Appendix D), leads to a homogenization of health risk with respect to the two full home-grown scenarios FHItD and FHNAD. Risk is reduced in areas of maximum fallout and increased in more populated areas of low fallout. Overall, the PHItD scenario results in a higher number of expected cancer cases (i.e., 0.28 for PHItD, 0.04 for FHItD and 0.09 for FHNAD).

It should be noted that in both PHItD and FMItD cases, COPC concentration in the food purchased in local markets depends upon the extension of the study area—the larger the area the lower the average level of contamination due to waste incinerator emissions. A careful definition of the extension of the study area is thus important to derive reliable estimates of food contamination and health risk.

5.4. Caveats and limitations

As in any modeling analysis, we provided here a simplified version of a complex system aimed at grasping the fundamental processes characterizing exposure to pollutants and their effects on human health. As such, our analysis is not exempted from limitations and additional research would be helpful to consolidate the methodology.

First, we analyzed only the long term health effect of primary PM_{10}. We did not account for other criteria pollutants – such as NO_x and SO_2, or for photochemical pollutants like tropospheric ozone and secondary particulate matter – nor for possible acute exposure to high concentrations in the case of malfunctioning of the incinerator's pollution abatement system. However, other studies have shown that actual emissions from these types of waste incinerator plants can be an order of magnitude smaller than the maximum legally authorized PM_{10} emission rate used in the present study (Biancolini et al., 2011; Buonanno et al., 2009; Buonanno et al., 2011; Forastiere et al., 2011). Therefore, our analysis should provide a conservative (i.e. worst-case) estimation of the associated health risk.

Additionally, a unique emission factor for domestic boilers was derived by ISPRA (2012) and used under the realistic hypothesis that all the domestic heat is produced in Parma through methane combustion. A more detailed analysis will be required in the future to provide a better characterization of the diffuse emission sources, since combustion for domestic heat generation is among the major sources of air pollution in urban settings along with traffic.

Moreover, several caveats characterize the assessment of health effects of micropollutants through the HHRAP model (EPA, 2005a), as thoroughly discussed in EPA (2005b); Hofelt et al. (2001) suggested that the HHRAP model largely over-predict bioaccumulation of PAH in animal foods. In our results, PAH was in fact the most important carcinogen, on average representing 89% of total carcinogenic risk. Other authors highlighted a substantial over-prediction of risks deriving from exposure to mercury (Palma-Oliveira et al., 2012). Overall, the HHRAP model could be considered quite conservative.

The number of micropollutants analyzed in the present study was limited by the lack of data in the Environmental Impact Statement of the proposed project on expected emissions of contaminants such as HMs and PCB. As overall health risk is assumed to be additive, simply accounting for further micropollutants would ultimately increase the TCR and HI. Further analysis will be required to account for the potential impacts of other pollutants not considered in the present study. To partially compensate for this limitation, we modeled PCDD/F and PAH in terms of their most toxic congeners (i.e., 2,3,7,8-TCDD and benzo[a]pyrene), since there is no information currently available about the emission profile of the stack.

Finally, the Monte Carlo simulation was carried out assuming simple non-informative uniform probability distributions for parameter sampling. The use of uniform distributions increased the uncertainty associated to health risk estimation and yielded values of the upper percentiles of health risk higher than in the case of beta distributions (ESM, Table A.8), thus providing health risk estimates consistent with a worse-case scenario. Moreover, as we wanted to focus our attention on the post-dispersion and -deposition processes, we did not carry out a sensitivity analysis for the parameters of the air quality model (e.g., particulate diameter, gas–particle partitioning, etc.). Here we refer to published studies explicitly addressing this issue (García-Díaz and Gozalvez-Zafrilla, 2012; Lonati and Zanoni, 2012; Yegnan et al., 2002).

6. Conclusions

The present study showed that, under the specific set of assumptions on exposure scenarios, transmission pathways and on the basis of the state of the art methodology for HRA, the proposed waste incinerator plant appears to cause a negligible increment to health risk, as long as it will be properly managed and emissions will never exceed the legal limits.

Our analysis also showed that the activation of district heating powered through heat recovery from waste incineration has the potential to compensate for stack emissions of particulate matter. Under these circumstances, it can be reasonable to locate a modern waste incineration plant in proximity of a densely populated urban area as long as it is possible to recover heat from waste combustion, convey it into an extensive district heating network and switch off as many domestic boilers as possible. Nevertheless, the reduction in exposure to macropollutants shall be carefully balanced against the increased exposure to micropollutants.

The caveats and limitations listed above suggest that our study should be considered a preliminary analysis and that the results should be taken cautiously. Moreover, even though our study showed a limited risk for the set of micropollutants analyzed in the present study, concern of resident population for plant mismanagement or for unexpected accidents or unreported emissions has to be considered legitimate and should not be undervalued. Adequate surveillance and monitoring systems should be also implemented to guarantee that emissions never exceed the authorized limits.

Whenever a choice was possible or practical given the available information, we made assumptions that generally tended to overestimate rather than underestimate the potential risk for human health. While several improvements and refinement could be certainly implemented in future studies of health risk assessment, we are confident that our modeling framework provided a robust preliminary estimate of health impacts of waste incineration under a variety of alternative exposure scenarios.

Appendix A. Supplementary data

Supplementary data associated with this article can be found in the online version, at http://dx.doi.org/10.1016/j.scitotenv.2012.11.079. These data include Google maps of the most important areas described in this article.

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