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Waste management health risk assessment: A case study of a solid waste landfill in South Italy

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ABSTRACT

An integrated risk assessment study has been performed in an area within 5 km from a landfill that accepts non hazardous waste. The risk assessment was based on measured emissions and maximum chronic population exposure, for both children and adults, to contaminated air, some foods and soil. The toxic effects assessed were limited to the main known carcinogenic compounds emitted from landfills coming both from landfill gas torch combustion (e.g., dioxins, furans and polycyclic aromatic hydrocarbons, PAHs) and from diffusive emissions (vinyl chloride monomer, VCM). Risk assessment has been performed both for carcinogenic and non-carcinogenic effects. Results indicate that cancer and non-cancer effects risk (hazard index, HI) are largely below the values accepted from the main international agencies (e.g., WHO, US EPA) and national legislation (D.Lgs. 152/2006 and D.Lgs. 4/2008).

1. Introduction

Waste treatment plants are now large, complex facilities where different biological processes take place under controlled conditions. Because of the large amounts of waste material treated, landfills can face several environmental problems. Most of the problems come from the landfill gas (LFG), and from its non-methanic organic compounds (NMOCs). NMOCs comprise about 39% of the total in a MSW landfill gas (USEPA, 1997). LFG composition is influenced by several factors including composition of solid waste in the landfill, stage of decomposition, oxygen availability, moisture and rain infiltration, pH, organic amount and microorganism population. It is clear that LFG composition is not fixed but changes in time. The total amount of VOCs released from landfilling has a significative impact for the environment. In the United States, solid waste management contributes about 10% of the total VOCs release in the atmosphere (USEPA, 1997). The most practiced control technologies for LFG are flares, which meet the technical requirements of the EU IPPC Directive (96/61/EC, paragraph 4 of schedule 2), but several LFG energy recovery programs are also available to landfill owners/operators that will satisfactorily control LFG emissions.

Landfills are identified as a hazardous air pollutant (HAP) source under the Urban Air Toxic Strategy (USEPA, 1999) and there is an increased attention from the population for toxicological aspects due to municipal solid waste (MSW) landfilling. Problems for nearby residents come mainly from the fact that they are exposed to LFG emissions. Several HAPs are present in LFG, and some of these are carcinogenic. There is also concern for emissions of dioxins/furans associated with landfill flares for their carcinogenic effects. All combustion systems, through pyrolysis or thermal decomposition, can initiate reactions that lead to the formation of polycyclic aromatic hydrocarbons (PAHs) and other trace species. Polychlorinated dibenzo-p-dioxins (PCDDs) and furans (PCDFs) can also be formed by this mechanism, and are known to be present in LFG flaring emissions.

Potential routes of exposure are different for nearby residents, being not only inhalation of gas or particles, but also ingestion of contaminated home-grown food, drinking water from wells contaminated with leachates, skin contact via contaminated soil, etc. Studies have assessed the health risk near landfills (ATSDR, 1992; Comba et al., 2006; Elliott et al., 2006; Jarup et al., 2002; Redfearn and Roberts, 2002) but findings seem not to demonstrate an increased risk for the exposed population, possibly because evidence is not sufficient to establish the causality of the association (WHO, 2007).

The landfill under investigation is located close to Agrigento, it has an actual extension of about 50,000 m^2 and a volume of 1.114.000 m^3. It has been in use since 1995 with a final potentiality of 1.874.000 m^3. Municipal solid wastes are spread and compacted in shallow layers and covered daily with a 15–25 cm layer of soil. It is a hilly isolated area and the closest village (Siculiana) is located at about 5 km. Recently there has been public concern about

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toxicity induced by the landfill. For this reason this study has been commisionated in order to better understand the local environment. The landfill accepts non hazardous waste, mainly municipal solid waste.

2. Experimental

2.1. Analytical approach

Emission data were based on results obtained from campaigns performed in summer 2007 and 2008. Samples were collected from the torch, where the LFG is burned, and from ambient air, both inside and outside the plant, and analysed with standard official methods (UNI-EN 1948-2:2006 for PCDDs, PCDFs in torch emissions, and EPA 1613 for soil samples, M.U. 825:89 Man. 122 Part. III for PAHs, UNI-EN 13649-2002 for volatile organic compounds) and with internal methods for VCM. Briefly, this analyte has been collected with active sampling over ORBO 91 sampling tubes, after internal standard addition (deuterated VCM), and analysed by gas-chromatography/mass spectrometry after desorption with methanol.

This risk assessment was based on data obtained from normal, current management, of the facility.

2.2. Emissions

Gas surface emissions of VCM have been calculated by reverse dispersion modelling (prEN 15445, 2007) starting from ambient air concentrations in different points (Fig. 1) in the landfill area. Briefly combining ambient air concentrations measured at different detectors and metereological data, the emission flux and location has been estimated as described in prEN 15445, 2007 normative. Backward modelling methods, like the one used, runs atmospheric transport and dispersion models in the reverse direction from the receptors (detectors in this case), in order to define the unknown diffusive upwind source(s). The concept that air sampled for concentration measurement at a detector has originated from somewhere leads to an inverse transport equation with the same diffusion, deposition and decay terms as the forward transport equation. These methods are preferred when more sources are present, or when are unknown in term of strength or location, like in this case. The inverse transport plume (puff) has been estimated in the same framework used for the standard forward modelling resulting in an emission source strength and location estimation. Analytical results, as well as results from the reverse dispersion modelling, have been therefore used as inputs for the classical "forward" modelling to describe air diffusion and deposition. The model used was designed on a 10 × 10 km area with a 200 m grid. Source emission for PCDDs and PAHs (flare) were considered as a point source (heigth = 10 m, diameter = 0.68 m, capacity 26.8 m^3/s, temperature = 899 °C). Pollutants concentrations of the selected pollutants have been computed with a simulation model (CALPUFF 6.112) to estimate the highest 1 h value for the year considered, yearly annual average concentrations and depositions in correspondence to six sensitive receptors: Siculiana, Torre Salsa, Montallegro, Mortilla, Contrada Pileri e Contrada Milione (small villages and resorts). Metereological data has been acquired from a local meteo station installed in the landfil and from SIAS (Servizio Informativo Agrometereologico Siciliano). Output from the diffusion model for average and maximum annual PAHs, VCM and dioxin-like compounds ambient concentrations are reported in Figs. 2–4.

The impact of leachate contamination has not been evaluated as the landfill is situated in a non aquifer with no drinking water supplies or surface water receptors (UK EA, 2004).

2.3. Risk assessment

The risk assessment was based on maximum daily intake (MDI) for non-carcinogenic and chronic daily intake (CDI), for the carcinogenic assessment. Agents that cause cancer in humans or in animals have been considered to have no-threshold (i.e., there is no "safe" exposure level unless there are data to the contrary). With these chemicals, any exposure has some risk and, as exposure increases, the probability of a carcinogenic response increases (USEPA, 1986).

This approach was used for the main known carcinogenic compounds emitted from landfills coming both from landfill gas torch combustion (dioxin-like compounds and PAHs) and from diffusive emissions (VCM). The scenarios, over a lifespan period, included different exposure pathways (inhalation, soil ingestion, soil dermal

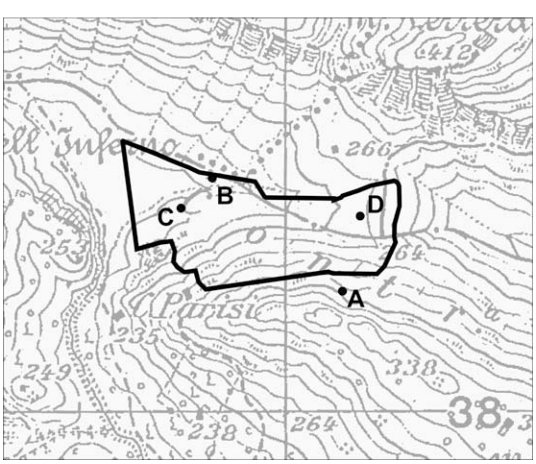

Fig. 1. Map showing the localization of the sampling points inside (B, C and D) and outside (A) the landfill.

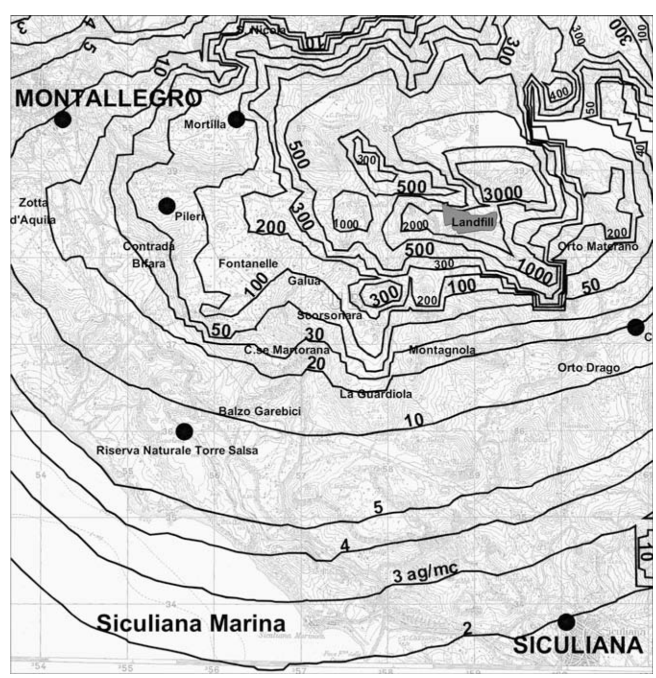

Fig. 2. PAHs average annual concentration in air expressed as ag/m^3 of B[a]P eq.

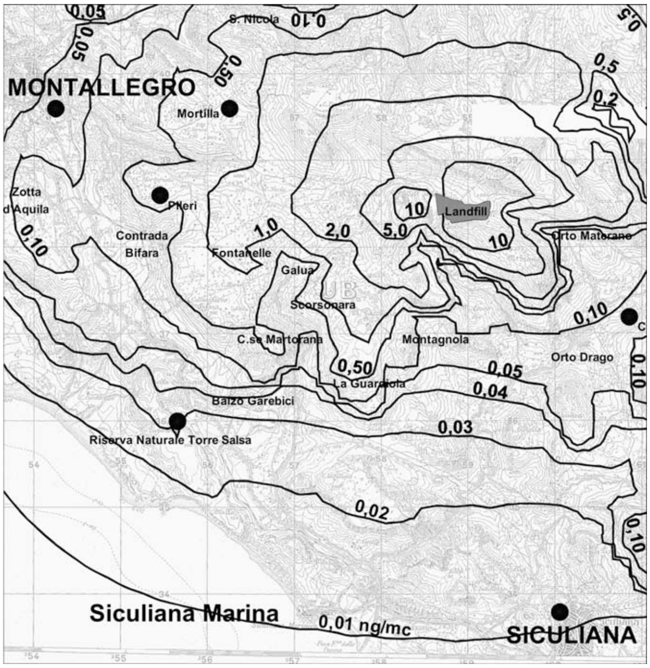

Fig. 3. VCM average annual concentration in air expressed as ng/m^3.

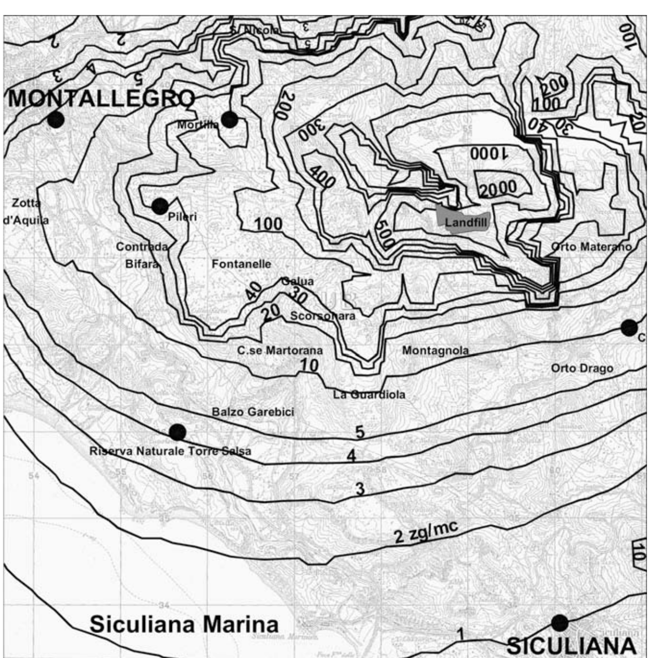

Fig. 4. Dioxin-like compounds average annual concentration in air expressed as zg/m^3.

contact, ingestion of home-grown products) for children and adults. Inhalation has been considered for VCM, dioxin-like compounds and PAHs. Soil ingestion and soil dermal contact has been considered for dioxin-like compounds and PAHs since the particles adsorbed fraction they deposits on soil. Ingestion of home-grown products has been considered considered only for dioxin-like compounds as they bioaccumulate and biomagnificate along the food chain. Risk for cancer and non-cancer effects have been estimated combining the exposure results with toxicological parameters and cancer potency values derived from the main international agencies for human health and environment protection.

In the risk assessment, the receptors of interest were located within 5 km from the landfill where people permanently or provisionally live.

Risk assessments for non-cancer effects the maximum daily intake (MDI) was calculated from:

$$\mathrm{MDI} = C \times \mathrm{CR} \times \mathrm{EF} \times \mathrm{ED}/(\mathrm{BW} \times \mathrm{AT}), \tag{1}$$

C is the concentration of chemical in air (mg/m^3), CR is the inhalation rate (m^3/day) or ingestion (mg/day), EF represents the exposure frequency (days or years), ED is exposure duration (years), AT is averaging time (years) and BW represents the body weight (kg).

For non-carcinogenic compounds, health effects were estimated using the equation:

$$\mathrm{HI} = (\mathrm{MDI}/\mathrm{RfD}), \tag{2}$$

where HI was the hazard index and RfD the Reference Dose (mg/kgBw day). RfD is defined as an estimate of daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime (USEPA, 1988).

For VCM, HI has been calculated using the inhalation reference concentration (RfC) and VCM concentration at receptors:

$$\mathrm{HI} = C \times \mathrm{RfC}. \tag{3}$$

Toxicological values for PCDDs, VCM and PAHs are reported in Table 1.

For carcinogenic effects, the chronic daily intake (CDI) was calculated considering the exposure for an average lifetime of 70 years:

Table 1
Toxicological values in milligrams of the chemical per kilogram of body weight (Bw) per day for PCDDs, PAHs and VCM. TWI is the tolerable weekly intake.

Pollutants	(mg/KgBw day)
PCDDs – 2,3,7,8-TCDD (EC SCF, 2001)	14.0×10^{-9} (TWI-ing)
PAHs – benzo(a)pyrene (ISS-ISPESL, 2009)	3.14 (RfD-inhalation)
VCM*	–

* VCM hazard index was calculated using the inhalation reference concentration 0.1 mg/m^3 (US EPA, IRIS, 2000).

Table 2
Slope factor values in milligrams of the chemical per kilogram per day used to value the cancer risk.

Pollutants	Slope factor	
	Sf ingestion (mg/kgday)$^{-1}$	Sf inhalation (mg/kgday)$^{-1}$
PCDDs – 2,3,7,8-TCDD (ISS-ISPESL, 2009)	1.5×10^5	1.16×10^5
PAHs - benzo(a)pyrene (ISS-ISPESL, 2009)	–	7.32
VCM*	–	–

* VCM cancer risk was calculated using the inhalation unit risk value 8.8×10^{-6} μg/m^3 (US EPA, IRIS, 2000).

Table 3
Point and non-point emission data used for the dispersion model calculations.

Pollutants	Concentration (ng/Nm^3)	Flux (ng/s)
PCDDs, PCDFs, PCBs (WHO–TEQ)	1.17×10^{-3}	7.60×10^{-4}
PAHs – (benzo(a)pyrene Eq.)	3.28	1.50
VCM	50	5.07×10^5

Table 4
Annual average of dioxins (as WHO–TEQ), PAHs (as B[a]P eq./m^3) and VCM, calculated by CALPUFF, for air samples, at receptors locations.

Receptor	PCDD–PCDF fg (WHO–TEQ)/m^3	PAHs pg B[a]P eq./m^3	VCM ng/m^3
Montallegro	5.5×10^{-6}	1.1×10^{-5}	6.0×10^{-2}
Mortilla	3.0×10^{-5}	5.9×10^{-5}	7.5×10^{-1}
Contrada Pileri	4.3×10^{-5}	8.4×10^{-5}	7.6×10^{-1}
Contrada Milione	9.8×10^{-6}	1.9×10^{-5}	8.4×10^{-2}
Riserva Naturale Torre Salsa	4.1×10^{-6}	8.1×10^{-6}	3.1×10^{-2}
Siculiana	1.0×10^{-6}	1.9×10^{-6}	1.1×10^{-2}

Table 5
Annual average deposition of PCDDs, PCDFs and PAHs, calculated by CALPUFF, in soil samples at receptors locations.

Receptor	PCDD–PCDF mg(WHO–TEQ)/kg DM	PAHs mg B[a]P eq./kg DM
Montallegro	1.3×10^{-13}	2.5×10^{-10}
Mortilla	6.1×10^{-13}	1.2×10^{-9}
Contrada Pileri	9.1×10^{-13}	1.8×10^{-9}
Contrada Milione	1.9×10^{-13}	3.8×10^{-10}
Riserva Naturale Torre Salsa	1.2×10^{-13}	2.4×10^{-10}
Siculiana	2.1×10^{-14}	4.2×10^{-11}

Table 6
Maximum concentrations (hour with highest value during the year) of dioxins (as WHO–TEQ), PAHs (as B[a]P eq./m^3) and VCM, calculated by CALPUFF, for air samples, at receptors locations.

Receptor	PCDD-PCDF fg (WHO–TEQ)/m^3	PAHs pg B[a]P eq./m^3	VCM ng/m^3
Montallegro	2.0×10^{-4}	3.9×10^{-4}	4.1
Mortilla	2.5×10^{-3}	4.9×10^{-3}	130
Contrada Pileri	1.4×10^{-3}	2.8×10^{-3}	39
Contrada Milione	3.5×10^{-3}	7.0×10^{-3}	9.4
Riserva Naturale Torre Salsa	5.4×10^{-4}	1.1×10^{-3}	3.8
Siculiana	1.9×10^{-4}	3.8×10^{-4}	2.0

$$\mathrm{CDI} = \mathrm{MDI}(70\ \text{years}). \quad (4)$$

Those agents that cause cancer in human or in animals have been considered to have no-threshold (i.e., there is no "safe" exposure level unless there are data to the contrary). With these chemicals, any exposure has some risk and, as exposure increases, the probability of a carcinogenic response increases (USEPA, 1986). Risk assessment for carcinogenic effect was calculated using this equation:

$$R = \mathrm{CDI} \times \mathrm{Sf}, \quad (5)$$

R is the cancer hazard risk and Sf (kg day/mg) represents the chemical's carcinogenic potency after administration. In Table 2, Sf values for inhalation and ingestion that are used to calculate cancer risk are reported. For VCM carcinogenic risk from inhalation exposure was calculated using the equation:

$$R = C \times \mathrm{UR}, \quad (6)$$

where C is the VCM concentration at receptors and unit risk (UR) is a continuous lifetime exposure from birth.

3. Results

Ambient air and soil depositions have been calculated for every receptors with the diffusion model, starting from data obtained by the analysis of the samples collected for both point and non-point emissions (Table 3). For PCDDs, PCDFs and PAHs, both deposition and air concentration have been calculated while for the volatile VCM only air concentration has been used.

In Tables 4 and 5, the annual average concentrations calculated for all measured pollutants, both in ambient air (Table 4) and in soil (Table 5), at different receptors' locations are reported. In Table 6 are reported the annual maximum concentrations in ambient air for all measured pollutants.

The highest values have been always detected at the Contrada Pileri receptor which is a location where people do not live permanently, whereas the lowest concentration were measured at Siculiana, a small town located few kilometers from the landfill. Tables 7–11 show the results of the exposure doses and health risks. The MDI is always slightly higher for children in comparison with adults and the reason is that the CR (Eq. (1)) is higher for children in relationship with their body weight.

3.1. PCDDs and PCDFs

The hazard index (HI) results for PCDDs and PCDFs obtained with total daily exposure for children and adults are reported in Table 7. For a non-carcinogenic compound the maximum acceptable level for HI is 1. As these HI values are 7–9 orders of magnitude below 1, the *potential* for adverse health effects are limited.

Results in Table 8 summarize cancer risk following a lifetime exposure, obtained for these pollutants. It appears that cancer risk due to measured emissions and soil depositions varies between 5.5×10^{-12} (5.5 expected cases out of 1000 billion) in Siculiana to 2.4×10^{-10} (2.4 expected cases out of 10 billion) in Pileri. These risks are 10^6 and 10^4 times lower than established acceptable values in Italy (D.Lgs. 152/2006 and D.Lgs. 4/2008).

3.2. PAHs

Table 9 reports non-cancer risk for PAHs exposure while their inhalation, ingestion and dermal contact of soil particles carcinogenic risk results are summarized in Table 10.

Also for PAHs, HI values appear to be much lower than 1 indicating limited risk due to these compounds, as well a low cancer incidence, with a calculated total risk R being in the 10^{-13} to 10^{-14} range.

Table 7
Exposure values as maximum daily exposure (MDI) data and hazard index (HI) for PCDDs and PCDFs in children and adults at the receptors. Routes of exposure considered were inhalation, soil ingestion, soil dermal contact and home-grown vegetables.

Receptor	MDI (pg/kgBW-day)		Hazard index (HI)	
	Children	Adults	Children	Adults
Montallegro	6.8×10^{-8}	1.6×10^{-8}	3.4×10^{-8}	8.1×10^{-9}
Contrada Mortilla	3.3×10^{-7}	8.3×10^{-8}	1.7×10^{-7}	4.2×10^{-8}
Contrada Pileri	4.9×10^{-7}	1.2×10^{-7}	2.5×10^{-7}	6.1×10^{-8}
Contrada Milione	1.1×10^{-7}	2.7×10^{-8}	5.3×10^{-8}	1.3×10^{-8}
Riserva Naturale Torre Salsa	6.1×10^{-8}	1.4×10^{-8}	3.1×10^{-8}	6.9×10^{-9}
Siculiana	1.1×10^{-8}	2.8×10^{-9}	5.7×10^{-9}	1.4×10^{-9}

Table 8
Exposure values as chronic daily exposure (CDI) and carcinogenc risk (R) for inhalation and ingestion (soil ingestion, soil dermal contact and home-grown vegetables consumption) for PCDDs and PCDFs.

Receptor	CDI (pg /kgBW-day)		Risk (R)		
	Inhalation	Ingestion	Inhalation	Ingestion	Total
Montallegro	8.2×10^{-10}	2.2×10^{-7}	9.6×10^{-14}	3.3×10^{-11}	3.3×10^{-11}
Contrada Mortilla	4.4×10^{-9}	1.0×10^{-6}	5.2×10^{-13}	1.6×10^{-10}	1.6×10^{-10}
Contrada Pileri	6.4×10^{-9}	1.6×10^{-6}	7.4×10^{-13}	2.3×10^{-10}	2.4×10^{-10}
Contrada Milione	1.5×10^{-9}	3.3×10^{-7}	1.7×10^{-13}	5.0×10^{-11}	5.0×10^{-11}
Riserva Naturale Torre Salsa	6.1×10^{-10}	2.1×10^{-7}	7.1×10^{-14}	3.2×10^{-11}	3.2×10^{-11}
Siculiana	1.5×10^{-10}	3.6×10^{-8}	1.7×10^{-14}	5.5×10^{-12}	5.5×10^{-12}

Table 9
Children and adults exposure values (MDI) and hazard index (HI) for PAHs inhalation.

Receptor	MDI (pg/kgBW-day)		Hazard Index (HI)	
	Children	Adults	Children	Adults
Montallegro	6.5×10^{-6}	3.1×10^{-6}	2.1×10^{-15}	9.9×10^{-16}
Contrada Mortilla	3.5×10^{-5}	1.7×10^{-5}	1.1×10^{-14}	5.3×10^{-15}
Contrada Pileri	5.1×10^{-5}	2.4×10^{-5}	1.6×10^{-14}	7.7×10^{-15}
Contrada Milione	1.2×10^{-5}	5.5×10^{-6}	3.7×10^{-15}	1.8×10^{-15}
Riserva Naturale Torre Salsa	4.9×10^{-6}	2.3×10^{-6}	1.6×10^{-15}	7.4×10^{-16}
Siculiana	1.2×10^{-6}	5.6×10^{-7}	3.7×10^{-16}	1.8×10^{-16}

Table 10
Chronic daily intake and carcinogenic risk for inhalation, ingestion and dermal contact of soil for PAHs.

Receptor	CDI (pg/kgBW-day)		Risk (R)		
	Inhalation	Soil ingestion and dermal contact	Inhalation	Soil ingestion and dermal contact	Total
Montallegro	1.6×10^{-6}	9.0×10^{-6}	1.2×10^{-14}	6.6×10^{-14}	7.8×10^{-14}
Contrada Mortilla	8.8×10^{-6}	4.3×10^{-5}	6.4×10^{-14}	3.2×10^{-13}	3.8×10^{-13}
Contrada Pileri	1.3×10^{-5}	6.5×10^{-5}	9.2×10^{-14}	4.7×10^{-13}	5.7×10^{-13}
Contrada Milione	9.8×10^{-6}	1.4×10^{-5}	7.2×10^{-14}	1.0×10^{-13}	1.7×10^{-13}
Riserva Naturale Torre Salsa	1.2×10^{-6}	8.6×10^{-6}	8.9×10^{-15}	6.3×10^{-14}	7.2×10^{-14}
Siculiana	2.9×10^{-7}	1.5×10^{-6}	2.1×10^{-15}	1.1×10^{-14}	1.3×10^{-14}

Table 11
Results for hazard index (HI) and cancer risk (R) due to VCM inhalation at the receptors' sites.

Receptor	mg VCM/m3	Hazard index (HI)	Risk (R)
Montallegro	6.0×10^{-8}	6.0×10^{-7}	5.3×10^{-16}
Contrada Mortilla	7.5×10^{-7}	7.5×10^{-6}	6.6×10^{-15}
Contrada Pileri	7.6×10^{-7}	7.6×10^{-6}	6.7×10^{-15}
Contrada Milione	8.4×10^{-8}	8.4×10^{-7}	7.4×10^{-16}
Riserva Naturale Torre Salsa	3.1×10^{-8}	3.1×10^{-7}	2.7×10^{-16}
Siculiana	1.1×10^{-8}	1.1×10^{-7}	9.7×10^{-17}

3.3. VCM

For VCM, as the landfill is situated in a non aquifer with no drinking water supplies or surface water receptors, only inhalation intake has been used for risk assessment. Results for non-carcinogenic risk (HI) and cancer risk (R) are reported in Table 11.

Results for the different scenarios for cancer and non-cancer effects always showed risk estimates which were orders of magnitude below those accepted from the main international agencies (WHO, US EPA) and by local legislation (D.Lgs. 152/2006 and D.Lgs. 4/2008).

4. Conclusion

The study has been undertaken mainly to assess potential incremental human cancer and non-cancer health risk due to specific carcinogenic compounds emissions from this landfill. Samples have been collected to characterize combustion products at the torch, for dioxins, dioxin-like compounds and PAHs, while ambient air samples were used to assess gas surface VCM emissions. Emission data has been used to estimate the dose of carcinogens for nearby residents and cancer and non-cancer potential health risk has been evaluated for resident receptors.

Results of our analysis indicate that potential incremental cancer risks for residents in the vicinity of the proposed facility and health effects are negligible compared with the Italian legislation thresholds (i.e., less than one in one million for cancer risk). Thus, based on the results of this air quality health risk assessment, the landfill should not have a significant adverse impact on human health. Cancer risk results are slightly lower, about an order of magnitude, but still comparable, with recently published data (Morra et al., 2009) in a similar area, in Sicily with a dismissed landfill and a MSW incinerator.

The risk assessment study is still on-going and the approach used is under review in order to be fully optimized for the site. Ongoing studies are considering other pollutants, like benzene and particulate but, mainly, will look for diffusion model validation and receptor's air quality determination.

In order to provide perspective for the results of this risk assessment, it will be important to compare our results with the overall risk of cancer in the local population due to the exposure to the same carcinogens and to integrate our results with information from the local health authorities in order to provide a more comprehensive picture.

Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.wasman.2009.10.013.

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