

REVIEW ARTICLE

Characterization and bioreactivity of respirable airborne particles from a municipal landfill

Lata Koshy¹, Timothy Jones², and Kelly Bérubé¹

¹School of Biosciences, Cardiff University, Museum Avenue, Cardiff, UK, and ²School of Earth and Ocean Sciences, Cardiff University, Park Place, Cardiff, UK

Abstract

With an increasing population and greater pressure on land-use, the possible problems of landfilling are of increasing concern. These concerns include the possible adverse health effects arising from living in the vicinity of municipal solid waste (MSW) landfills. Human exposure to potential landfill emissions by respiratory, gastrointestinal or dermal mechanisms warrants further investigation. PM₁₀ and PM_{2.5} from a UK landfill were physicochemically characterized and their bioreactivity screened by a plasmid scission assay in comparison with an urban PM collection. Preliminary data from human toxicology pathway-specific microarrays indicate landfill PM₁₀ presents a comparable geobiological insult to urban PM₁₀ in a human tracheobronchial tissue model.

Keywords: Landfill; PM₁₀; toxicity; bioreactivity; plasmid scission assay

Introduction

A recent study by Elliot et al. (2001) estimated that 80% of the UK population reside within 1.5 miles of a working or closed landfill site. The major municipal solid waste (MSW) landfill emissions from an operating site are particulate matter, leachate and landfill gas. Although there are many sources of literature available on landfill leachate and gas characterization, this mini-review will focus on the less reported field of ambient airborne particles related to landfills. Particulate matter (PM) with aerodynamic diameters of less than 10 or 2.5 µm (PM₁₀ and PM_{2.5}) is recognized as an important cause of adverse human health (Englert 2004, Kappos et al. 2004, Brunekreef & Forsberg 2005). There is a current paucity of information on landfill PM, meaning conclusions about landfill PM require further research (Defra 2004).

Landfill dust is generated by a variety of mechanical and chemical processes. These include: the movement of heavy dustcarts and site vehicles over dry unpaved access roads and previously deposited waste; diesel exhaust fumes and brake emissions from on-site vehicles; action of tipping waste at the working face raises plumes

of dust, notably on elevated ground, which are exposed to windy conditions; waste compaction by bulldozers and crushers; and stockpiles of bare earth required for daily waste coverage are susceptible to resuspension and dispersion by wind (Fitz & Bumiller 2000). Arid and windy conditions may lead to higher levels of entrained PM in the local atmosphere. Human exposure can occur via respiratory, dermal and ingestion pathways, and is sensitive to meteorological conditions. Vega et al. (2001) performed resuspension of surface dust from a Mexico City landfill, obtaining PM₁₀ and PM_{2.5} fractions for physicochemical analyses. Interestingly, their gravimetric analysis revealed a similar mass distribution to that of the UK landfill. However, their chemical analysis revealed a significantly different metal profile, with Al being the most significant metal component, whilst Fe was the most significant metal present in the UK landfill presented in this review.

In a literature-based modelling study, Macleod et al. (2006) estimated the amounts of airborne incinerator residual ash, which could be potentially released as particulates from landfills. Although these sites were not specific MSW sites in the UK, which no longer accept

such controlled wastes, their assessment positively identified the atmospheric dispersion of dusts as the main pathway for human exposure. The main route of exposure to landfill PM is by inhalation and possibly to a lesser extent, by ingestion. The re-dispersion of accumulated dust on clothing can increase personal exposure to PM; however this is more likely within the landfill workforce rather than public exposure (Poulsen et al. 1995).

It is important to find sensitive biological indicators of the potential harm that these emissions may cause following human exposure. Linking toxicological analysis to the geochemical data should help to determine accurately the degree of toxicity of environmental emissions produced by landfills. This study aims to determine the levels of respirable landfill particulates released from a UK MSW landfill, and to identify the acellular bioreactivity of these pollutants. We also present our preliminary data of transcriptional changes in a human epithelial tracheobronchial model upon exposure to landfill PM₁₀, and compare these observed effects to urban PM₁₀ exposure.

Physicochemistry

Sample collection

The study site is a municipal solid waste landfill, located within a major UK city. Sampling at an urban location approximately 2.5 miles from the landfill was also

undertaken. Two collecting systems were utilized in this study during October 2007. A Negretti selective-inlet system was used to collect PM₁₀ on polycarbonate filters for characterization. The system operates at a flow-rate of 30 l min⁻¹, drawing air along a set of defined horizontal plates, through to the polycarbonate filter. Pre-calibrated settings prevent PM with a mean aerodynamic diameter of greater than 10 µm, from progressing through the elutriator (Greenwell et al. 2002). A Harvard high-volume collector, configured to collect particulates of 10–2.5 µm and 2.5–0.1 µm, was used to accumulate PM₁₀ onto polyurethane foam substrates for bioreactivity assays (Jones et al. 2006).

PM characterization

Gravimetric and scanning electron microscopy (Moreno et al. 2004) analyses of landfill and urban PM revealed a similar size distribution of PM₁₀ at these two locations; however, it also revealed significantly increased PM₁₀ generation at the landfill compared with the urban city location, i.e. 42 and 13 µg m⁻³, respectively. Airborne dust emissions are predominantly episodic, and highly dependent upon site operations and ambient conditions. This also means that extrapolation of gravimetric measurements to the long-term air quality indicators may not be the most appropriate use of this data (Macleod et al. 2006). Both sites generated PM₁₀ with the vast majority of particles distributed within 2.5–0.1 µm aerodynamic

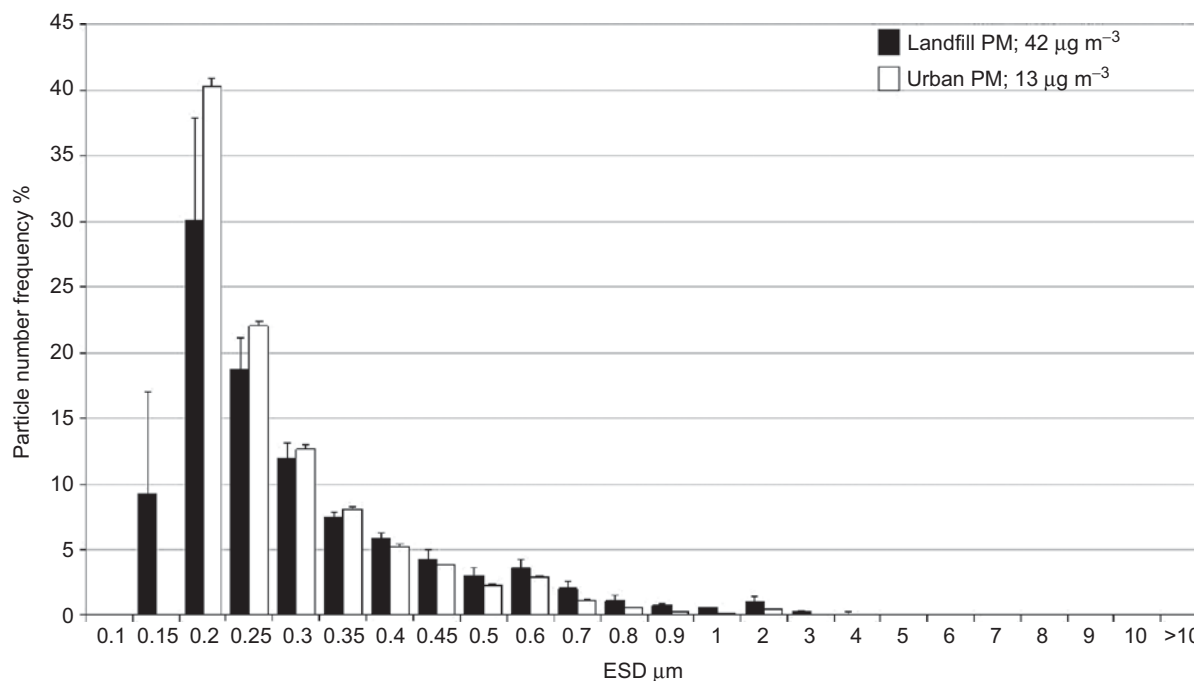


Figure 1. Skewed size distribution of PM₁₀ collected from a municipal solid waste landfill and a nearby urban location, both located in the UK. The results indicate the vast majority of PM from both locations are respirable. Note the small fraction of PM 0.1–0.15 µm in the landfill collection. The landfill PM mass concentration was much higher than the urban location.

diameter (Figure 1). The dominant PM types were identified as either mineral or soot, with the dominant PM at the landfill site being mineral (98% particle number), whilst the urban PM₁₀ sample comprised mostly, of anthropogenic soot PM (95% particle number).

Metal analysis

The PM_{10-2.5} and PM_{2.5-0.1} from both study sites were microwave acid-digested and analysed for total trace metals by ICP-MS (Moreno et al. 2004). The elements of toxicological concern, Fe, Zn, Ni and Pb, were prevalent in all samples. Both PM fractions, PM_{10-2.5} and PM_{2.5-0.1}, collected at the landfill contained higher overall levels of metals than the corresponding urban samples (Figure 2A, B). Landfill PM_{10-2.5} was found to be significantly Fe-rich compared with the accompanying landfill PM_{2.5-0.1} fraction, or the urban PM₁₀ fractions (SPSS 15.0 statistics package; one-way ANOVA, $p < 0.01$). The landfill PM_{2.5-0.1} fraction

contained significantly higher levels of Zn and Pb than the other samples ($p < 0.01$).

Bioreactivity

Plasmid scission assay

Particulates were screened by the plasmid scission assay (PSA), as an *in vitro* measurement of oxidant PM-bioreactivity upon DNA (Donaldson et al. 1997). Aliquots (200 ng) of Φ X174 RF plasmid DNA were exposed to PM₁₀, PM_{10-2.5} and PM_{2.5-0.1}. The PM₁₀ sample was derived from proportional masses of PM_{10-2.5} and PM_{2.5-0.1}. Exposure of the supercoiled DNA to damaging, oxidative PM caused a conformational change in the tertiary structure of the plasmid, to the relaxed and/or linear forms. This resultant damage was detected by gel electrophoresis and semiquantified as a proportion of the total DNA present (Koshy et al. 2007).

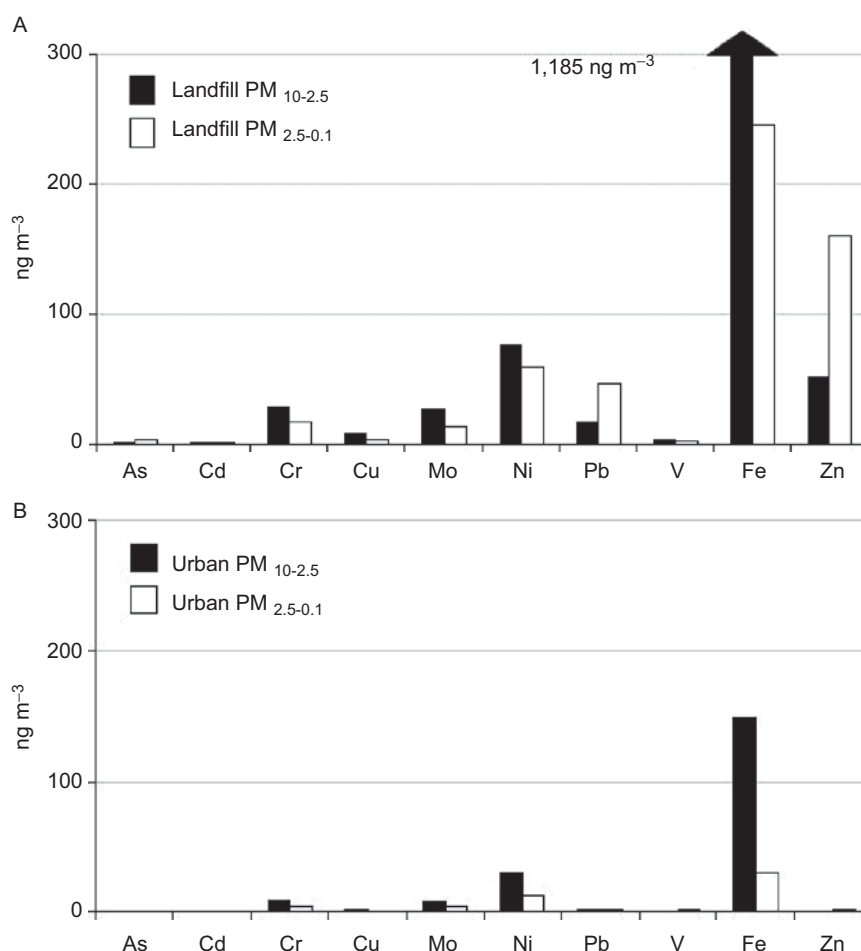


Figure 2. (A) Landfill PM; (B) urban PM. The results show that Fe is the most abundant metal in landfill and urban PM, and in the case of the landfill PM_{10-2.5} fraction, this was highly significant. The other dominant metals in both size fractions of landfill PM were Zn and Pb. The urban PM contained lower metal concentrations in both fractions compared with their respective landfill samples. However, as in the landfill collection, Fe was also the most abundant metal in the urban collection.

Data were compared for statistical significance between PM sizes intrasite, and between corresponding PM sizes of the two different sites (SPSS 15.0 statistics package; two-way ANOVA and least significant difference *post-hoc* testing). The PM_{2.5-0.1} fractions at both the landfill (Figure 3A) and urban sites (Figure 3B) were found to be significantly more reactive than their corresponding PM_{10-2.5} or PM₁₀ fractions ($p < 0.01$). This finding is in agreement with other studies which report smaller sizes of PM as being more reactive (Risom et al. 2005, Diociaiuti et al. 2001). Landfill PM_{2.5-0.1} exhibited a much higher oxidative capacity than the urban PM_{2.5-0.1} ($p < 0.05$), with the former causing 50% damage (TD₅₀) at 28 µg ml⁻¹, whilst urban PM_{2.5-0.1} elicited the same level of damage at 185 µg ml⁻¹. The landfill PM_{2.5-0.1} fraction was the dominant contributor to bioreactivity in the landfill PM collection. It was noted that there was no significant difference between the landfill PM₁₀ and PM_{10-2.5} fractions. The trend of bioreactivity of the landfill samples was in the order of PM_{2.5-0.1} > PM₁₀ = PM_{10-2.5}.

In contrast, a positive dose-response was observed with all fractions from the urban site, in which the extent of bioreactivity was significantly dependent upon the size fraction. The trend of bioreactivity of the urban site was in the order of PM_{2.5-0.1} > PM₁₀ > PM_{10-2.5}. All the urban size fractions, excluding PM_{10-2.5}, exhibited significantly higher

oxidative capacity when compared with their counterparts in the landfill collection ($p < 0.05$). Overall, the landfill PM₁₀ bioreactivity was dominated by the less reactive PM_{10-2.5} fraction, whilst the urban PM₁₀ bioreactivity was dominated by the more reactive PM_{2.5-0.1} fraction.

Exposure of landfill PM to human tracheobronchial epithelial cells in vitro

The commercially available EpiAirway-100 3-D model (MatTek Corp., Ashland, MA, USA) was exposed to PM_{10-2.5}, PM_{2.5-0.1} and PM₁₀ from the landfill, and the PM₁₀ fraction from the urban collection (500 µg ml⁻¹; 24h). Conventional toxicology and genomics were performed. The MTT assay was used to determine cell viability (mitochondrial activity), whilst the transepithelial electrical resistance (TEER) measurements for structural integrity were taken pre- and post-exposure. Genomic analysis of the exposed tissue was performed according to the GEArray Human Toxicology and Drug Resistance microarray protocol (SABiosciences, Frederick, MD, USA). Gene expression was determined by Significance Analysis

Table 1. Distribution of upregulated genes, grouped into gene families by percentage (landfill PM₁₀ = 66 genes; urban PM₁₀ = 57 genes).

Gene family	Examples of genes expressed in EpiAirway-100 (≥ 1.5 fold, $p < 0.05$)	Landfill PM ₁₀	Urban PM ₁₀
Drug metabolism	N-Acetyl transferase 2, glutathione peroxidase 1, peroxiredoxins 1 and 2, glutathione S-transferase, cytochrome P450 11B2	29	28
Transcription factors and regulators	Retinoic acid receptor α, MYST histone acetyltransferase, NFKB1, v-rel reticuloendotheliosis viral oncogene homolog B	15	14
Stress response	Superoxide dismutases 1 and 2, NADPH dehydrogenase 1, paraxonase 3, nudix type 1, DNA damage-inducible transcript 3	11	9
Cell growth, proliferation and differentiation	Cyclin-dependent kinases, V-erb-b2 erythroblastic leukaemia viral homolog, macrophage migration inhibitory factor	12	7
Chaperones and heat shock proteins	Chaperonins, heat shock proteins and transcription factors, suppression of tumorigenicity 13, crystallin αA	27	28
Apoptosis	BCL-like 2, tumour necrosis factor receptor superfamily 1A, E2F transcription factor, NF-κB inhibitor α	5	5
Cell cycle	Breast cancer 2, cyclin-dependent kinases 2 and 4, DNA-damage-inducible transcript 3	2	9

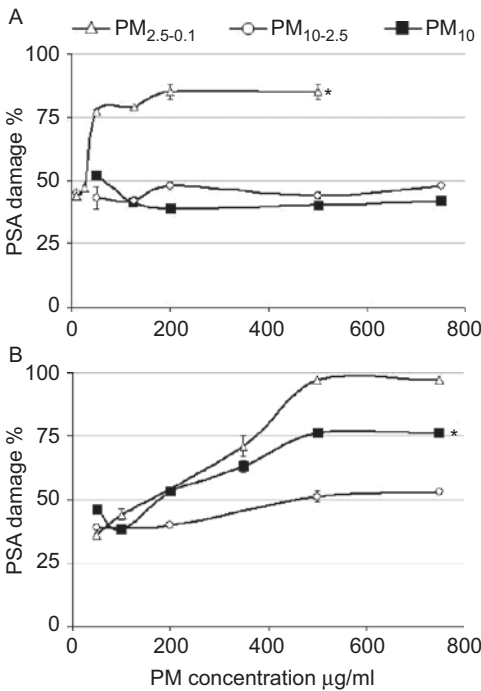


Figure 3. (A) Landfill PM; (B) urban PM. Plasmid scission assay of the different PM fractions collected at the landfill and urban locations. Data are expressed as mean ± SD ($n = 3$). *Statistical significance between corresponding size fractions from the two study sites ($p < 0.05$). The PM_{2.5-0.1} samples from both locations were the most bioreactive, with the landfill collection exhibiting the greatest oxidative capacity.

of Microarrays software (Stanford University), in which changes of greater than 1.5-fold ($p < 0.05$) were considered to be relevant. There were no significant adverse effects detected by TEER measurements, or by the MTT viability assay upon exposure to any of the three landfill PM fractions, or from the urban PM₁₀. However, preliminary microarray data of the landfill and urban PM₁₀ indicated a significant trend of upregulation in several transcription factors and drug metabolism enzymes (Table 1).

Conclusions

PM₁₀ collected at a municipal UK landfill displayed a highly skewed size distribution, with the majority of particles residing in the PM_{2.5-0.1} fraction, as amorphous particulates. The study here also demonstrated that acellular ROS-induced bioreactivity of the landfill PM_{2.5-0.1} fraction was shown to be significantly higher than a corresponding urban PM_{2.5-0.1} sample. To our knowledge, no other publications target MSW landfill PM₁₀ toxicity, and this preliminary data should be reported.

Acknowledgments

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

- Brunekreef B, Forsberg B. (2005). Epidemiological evidence of effects of coarse airborne particles on health. *Eur Respir J* 26:309–18.
- Defra. (2004). Review of Environmental and Health Effects of Waste Management: municipal solid waste and similar wastes. Department for Environment. London: The Stationary Office HMSO.
- Diociaiuti M, Balduzzi M, De Berardis B, Cattani G, Stacchini G, Ziemacki G, Marconi A, Paoletti L. (2001). The two PM_{2.5} (fine) and PM_{2.5-10} (coarse) fractions: evidence of different biological activity. *Environ Res* 86:254–62.
- Donaldson K, Brown DM, Mitchell C, Dineva M, Beswick PH, Gilmour P, MacNee W. (1997). Free radical activity of PM10: iron-mediated generation of hydroxyl radicals. *Environ Health Perspect* 105 (Suppl. 5):1285–9.
- Elliott P, Briggs D, Morris S, De Hoogh C, Hurt C, Jensen TK, Maitland I, Richardson S, Wakefield J, Jarup L. (2001). Risk of adverse birth outcomes in populations living near landfill sites. *BMJ* 323:363–8.
- Englert N. (2004). Fine particles and human health – a review of epidemiological studies. *Toxicol Lett* 149:235–42.
- Fitz DR, Bumiller K. (2000). Evaluation of watering to control dust in high winds. *J Air Waste Manage Assoc* 50:570–7.
- Greenwell LL, Moreno T, Jones TP, Richards RJ. (2002). Particle-induced oxidative damage is ameliorated by pulmonary antioxidants. *Free Rad Biol Med* 32:898–905.
- Jones T, Moreno T, Bérubé K, Richards RJ. (2006). The physicochemical characterisation of microscopic airborne particles in south Wales: a review of the locations and methodologies. *Sci Total Environ* 360:43–59.
- Kappos D, Bruckmann P, Eikmann T, Englert N, Heinrich U, Höppe P, Koch E, Krause GHM, Kreyling WG, Rauchfuss K, Rombout P, Schulz-Klemp V, Thiel WR, Wichmann HE. (2004). Health effects of particles in ambient air. *Int J Hyg Environ Health* 207:399–407.
- Koshy L, Paris E, Ling S, Jones T, Bérubé K. (2007). Bioreactivity of leachate from municipal solid waste landfills – assessment of toxicity. *Sci Total Environ* 384:171–81.
- Macleod C, Duarte-Davidson R, Fisher B, Ng B, Willey D, Shi JP, Martin I, Drew G, Pollard S. (2006). Modeling human exposures to air pollution control (APC) residues released from landfills in England and Wales. *Environ Int* 32:500–9.
- Moreno T, Merolla L, Gibbons W, Greenwell L, Jones T, Richards R. (2004). Variations in the source, metal content and bioreactivity of technogenic aerosols: a case study from Port Talbot, Wales, UK. *Sci Total Environ* 333:59–73.
- Poulsen OM, Breum NO, Ebbeløj N, Hansen M, Ivens UI, Van Lelieveld D, Malmros P, Matthiassen L, Nielsen BH, Nielsen EM. (1995). Collection of domestic waste. Review of occupational health problems and their possible causes. *Sci Total Environ* 170:1–19.
- Risom L, Mäler P, Loft S. (2005). Oxidative stress-induced DNA damage by particulate air pollution. *Mutat Res Fund Mol Mech Mutagen* 592:119–37.
- Vega E, Mugica V, Reyes E, Sánchez G, Chow JC, Watson JG. (2001). Chemical composition of fugitive dust emitters in Mexico City. *Atmos Environ* 35:4033–9.