**Introduction**

Recent epidemiological studies have shown that atmospheric pollution caused by airborne particulate matter (PM) has a negative impact on human health. Adverse outcomes include exacerbation of respiratory symptoms, reduced lung function, chronic bronchitis, cardiovascular diseases and mortality. Heterogeneous studies suggest that such health effects can depend on PM chemical composition, as well as site and (possibly) season. Chemical analysis shows PM to comprise of many inorganic (Trace metal/Inorganic ions), organic, and elemental materials, several of which are toxic. For example, sulphuric metal ions such as zinc have been found to be linked to lung injury. Metals on PM can be found within the matrix of an insoluble component, within a soluble salt, or complexed to a surface. Metals which can be available in more than one stable form can be evaluated on an electron transfer and therefore demonstrate some capacity to generate oxidants. Therefore it is important to determine the contribution of metals and the other chemical components, to the adverse health effects observed in PM epidemiology studies. Hence we have utilized various protocols for measuring cellular effects, which might be suitable for measuring the toxicity of PM (e.g. assays to determine cell damage, cell defence, membrane damage and reactive oxygen species (ROS) production). For example, the lactate dehydrogenase (LDH) assay has been used to evaluate cell damage by measuring the enzyme activity leakage in the medium. The reduced glutathione (GSH) assay has been employed to evaluate how GSH, one of the primary biochemicals for cell defence, can be influenced by PM toxicity. Cytokine induction by PM is determined by quantitatively measuring IL-6, (a major inducer of the acute phase reactions in response to inflammation or tissue injury). Reactive Oxygen Species have been monitored by fluorimetric assay (DCFH-DA) which evaluates the ROS produced in exposed cells compared to control cells. To investigate the biological effects of PM2.5 at a sub cellular level, human epithelial pulmonary A549 cell line was exposed for three days to different concentrations of PM2.5 (0, 5.5, 11.0, 22.0 µg/cm²) and the above mentioned assays were employed.

**Methodology**

One experimental purpose of the project was to evaluate the variability in toxicity of PM sampled during different seasons and in different geographical sites in Cork city.

To perform the toxicology experiments, the PM was extracted from polyurethane for cell cultures in a pozzolana extraction: a technique based on a modified method that by Baltsan et al (2008). The ASE4 instrument was chosen as a model for measuring cell toxicity exposed for 72 hours to different concentrations of PM2.5 (0, 5.5, 11.0, 22.0 µg/cm²).

Vitamin: 20 metals comprising As, Sb, Hg, Mo, Zn, Sn, Pb, Cu, Cr, Ni, Fe, Cd, Sr, Mn, Mg, Y, Co, Cr, Cu, Ti and Al were analyzed by ICP-AES after microwave assisted acid digestion and water based extraction.

**Results**

The ability of PM2.5 to produce Reactive Oxygen Species (ROS) was determined according to the method of Sony & Ash (1975) (modified as per Lippmann et al, 1997). The reduced form of Glutathione (GSH) which provides defence against ROS by scavenging free radicals and reducing H2O2 was determined according to the method of Thomas & Biesheuvel (1995). Activity of IL-6 was determined using the colorimetric detection kit from Bio-Rad (Assay kit units).

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- ERC Office, Department of Chemistry and Environmental Research Institute, University College Cork, Cork, Ireland

**References**


**Keywords:** PM2.5, transition metals, toxicity, ROS, GSH, IL-6, IL-8

**Project Overview**

- **ERITASK:** Project Overview
- **P1. Field Measurement of PM2.5 in Cork City Area**
- **P2. *Total Chemical Analysis Method Development***
- **Chemical Compositions of PM2.5**
- **P3. Toxicological Effects of PM2.5**
- **P4. Source Apportionment Modelling**

**Conclusion and the Way Forward**

The treatment of the A549 cell line with PM2.5 induced a non-concentration dependent effect on the cytotoxicity of cells as compared to controls. The A549 cell line was chosen as a model for measuring cell toxicity exposed for 72 hours to different concentrations of PM2.5 (0, 5.5, 11.0, 22.0 µg/cm²). PM2.5 exposure for 72 hours significantly reduced Glutathione levels compared to control cells. To investigate the biological effects of PM2.5 at a sub cellular level, human epithelial pulmonary A549 cell line was exposed for three days to different concentrations of PM2.5 (0, 5.5, 11.0, 22.0 µg/cm²) and the above mentioned assays were employed.

**Keywords:** PM2.5, transition metals, toxicity, GSH, LDH, IL-6, IL-8

**Project Outline**

- **ERITASK:** Project Overview
- **P1. Field Measurement of PM2.5 in Cork City Area**
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**Synthesis**

1. Urban/City site
2. Urban Background/Landfill site
3. Rural site

Sampling Sites

3 sampling sites – 4 seasons each

LDH 3 day exposure mean

**Preliminary Statistical Analysis**

Applying linear regressions in the metals and ROS results a positive correlation can be found showing a direct relationship between transition metals content in PM2.5 and ROS production (Table 1).

<table>
<thead>
<tr>
<th>Correlation</th>
<th>Cu</th>
<th>Zn</th>
<th>Co</th>
<th>Fe</th>
<th>Ni</th>
<th>Mn</th>
<th>Cr</th>
<th>NiO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cu</td>
<td>0.709</td>
<td>0.8388</td>
<td>0.8553</td>
<td>0.7602</td>
<td>0.5732</td>
<td>0.8349</td>
<td>0.5400</td>
<td>0.7984</td>
</tr>
</tbody>
</table>

The UBS site exhibited the highest total metal and transition metal concentrations shown in Fig. 7. This could be largely due to the influence of the UBS landfill site (UBS Spring) as compared to the City site. The order of Total and Transition metal concentrations, starting with the highest concentrations was found to be: UBS site > City site > Rural site (Fig. 7).

**Further Information**

- More information regarding this study can be found on our research directory website using the following link:
  - http://crac.ucc.ie/research_part.html