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The chemical composition of ultrafine particles and associated biological effects at an alpine town impacted by wood burning

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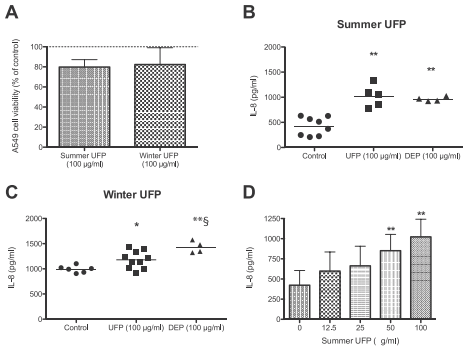
HIGHLIGHTS

- Physical-chemical properties and biological effects of UFPs emitted by wood burning were investigated at an alpine town.
- Cell cultures were used as sensors to test the toxicological properties of UFPs.
- UFPs collected in the summer were more active in inducing IL-8 release compared to winter UFPs.
- Genotoxic effects induced by UFPs were higher in winter than in summer samples.

GRAPHICAL ABSTRACT



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ABSTRACT

This work is part of the TOBICUP (TOxicity of BIomass Combustion generated Ultrafine Particles) project which aimed at providing the composition of ultrafine particles (UFPs, i.e. particles with aerodynamic diameter, d_{ae}, lower than 100 nm) emitted by wood combustion and elucidating the related toxicity. Results here reported are from two ambient monitoring campaigns carried out at an alpine town in Northern Italy, where wood burning is largely diffused for domestic heating in winter. Wintertime and summertime UFP samples were analyzed to assess their chemical composition (i.e. elements, ions, total carbon, anhydrosugars, and polycyclic aromatic hydrocarbons) and biological activity. The induction of the pro-inflammatory cytokine interleukin-8 (IL-8) by UFPs was investigated in two human cells lines (A549 and THP-1) and in human peripheral blood leukocytes. In addition, UFP-induced oxidative stress and genotoxicity were investigated in A549 cells. Ambient UFP-related effects were compared to those induced by traffic-emitted particles (DEP) taken from the NIES reference material "vehicle exhaust particulates". Ambient air UFPs induced a dose-related IL-8 release in both A549 and THP-1 cells; the effect was more relevant on summer samples and in general THP-1 cells were more sensitive than

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A549 cells. On a weight basis our data did not support a higher biological activity of ambient UFPs compared to DEP. The production of IL-8 in the whole blood assay indicated that UFPs reached systemic circulation and activated blood leukocytes. Comet assay and γ-H2AX evaluation showed a significant DNA damage especially in winter UFPs samples compared to control samples.
Our study showed that ambient UFPs can evoke a pulmonary inflammatory response by inducing a dose-related IL-8 production and DNA damage, with different responses to UFP samples collected in the summer and winter periods.

1. Introduction

Pollution due to particulate matter (PM) is currently of major concern, and it has been evaluated as the 9th highest risk factor in a study on the global burden of disease (Lim et al., 2012). Air pollution is considered as a major environmental risk to health affecting both developed and developing countries by the World Health Organization, which states that by decreasing air pollution levels, countries can reduce the burden of heart diseases, lung cancer, acute and chronic respiratory diseases (Dominici et al., 2006; Pope and Dockery, 2006; WHO, 2014). These effects are believed to be due to exposure to PM10 and PM2.5 (i.e. particles with aerodynamic diameter lower than 10 and 2.5 μm, respectively), which can penetrate deep into the lungs (Johnson and Vincent, 2003; Pope and Dockery, 2006).

Particulate matter is composed of a complex mixture of inorganic and organic, liquid and solid particles suspended in atmosphere; it is often referred to as atmospheric aerosols (Hinds, 1999). It is one of the most challenging pollutants due to its complex nature having e.g. primary and secondary origin, a variety of size-distributions and chemical compositions, as well as being emitted by both natural and anthropogenic sources. Moreover, it is a priority issue in air quality and climate studies (Colbeck and Lazaridis, 2014). In urban areas major sources are largely ascribed to combustion processes, and among them wood burning for domestic heating is growing more and more also in Europe. Indeed, as promoted by the Biomass Action Plan of the European Commission (EC, 2005), it represents a renewable energy source and contributes to lower green-house-gases emissions. Moreover, considering the increasing trend in the cost of other fuels (e.g. natural gas and oil) residential biomass combustion will increasingly become a cheaper alternative.

Wood combustion is a relevant source for organic particulate matter (Daellenbach et al., 2016). In several areas - including the Po Valley in Northern Italy, which is one of the continental hot-spots for air pollution - wood combustion gives a significant contribution to the high PM concentration observed (Bernardoni et al., 2011; Piazzalunga et al., 2011; Bernardoni et al., 2013; Amato et al., 2016). Wood burning impact on air quality is mainly due to the use of low efficiency combustion appliances, especially in small villages where open fireplaces are largely in use. A survey carried out by Pastorello et al. (2011) reported that in Lombardy region (which is located in the Po valley) about 16% of households have generally old and inefficient wood burning appliances, and 10% of the houses are equipped with new generation combustion systems (e.g. pellet or chips automatic stoves, log wood innovative stoves).

In PM10 and PM2.5 samples collected in the Po Valley atmospheric organic pollutants with a strong toxicological impact, such as Polycyclic Aromatic Hydrocarbons (PAHs) and dioxins were detected and related to wood combustion (Belis et al., 2011; Piazzalunga et al., 2012). PAHs are a class of substances of great interest because of their adverse effects on human health (IARC, 2010) due to their mutagenic and carcinogenic properties (Agudelo-Castañeda and Teixeira, 2014); indeed, PAHs are known to cause DNA adducts (Godschalk et al., 2000; Squadrito et al., 2001). Among PM components, it is also important to note that transition metals may induce DNA strand breakage by inducing ROS (Chapman et al., 1997).

Recent studies indicate that inhalation of wood smoke emissions can affect pulmonary immune defence mechanisms. Moreover, macrophages (i.e. the main defence of the lung providing the link between the non-specific and specific defence mechanisms of the respiratory tract) together with lung epithelial cells are the principal targets for wood smoke-induced immunotoxicity (Zelikoff et al., 2002). Several epidemiological studies (reviewed in Naeher et al., 2007) concerning exposure to biomass smoke both indoor and outdoor indicate an association with increased risk of respiratory illness and decreased lung functions. Susceptible subpopulations include asthmatics and children, this being consistent with results observed in studies on ambient air pollution impact on human health.

There is still a gap of knowledge on UFPs (i.e. airborne particles with aerodynamic diameter, d_{ae}, lower than 100 nm) health impact as they are usually not monitored on a routine basis and data on UFPs physical-chemical properties are also very scarce in the literature (Ntziachristos et al., 2007; Terzano et al., 2010). UFPs are typically emitted by combustion processes, so that in urban areas traffic and domestic heating are often the most relevant ones. As UFPs have a large specific surface area and longer residence times in lung compared to larger size particles, they are believed to induce strong and prolonged lung inflammation. There are few epidemiological studies on UFPs and (cause-specific) mortality with inconsistent results presented. Cardiovascular effects due to UFPs were reported in few epidemiological studies (Wichmann et al., 2000; Oberdörster et al., 2005). These can be explained by induction of pulmonary inflammatory response, translocation of UFPs from the lung into circulation with subsequent toxicity to vascular epithelium, alteration of blood coagulation, interference with autonomic nervous system activity, all of which constitute the causal link between particle inhalation and risk of cardiovascular diseases (Terzano et al., 2010; Saber et al., 2014; Chen et al., 2016). Despite the documented respiratory health effects of PM2.5, in contrast, the literature remains inconclusive and the respiratory health effects appear independent from particle mass exposures although evidence for a relationship between UFPs and children's respiratory is increasing (Heinzerling et al., 2016; Lanzinger et al., 2016).

There is also growing concern that air pollution exposure may increase the risk of lung cancer. Animal studies have shown that exposure to DEP (i.e. vehicle exhaust particles taken from NIES certified reference material n°8 (NIES, Ibaraki, Japan)) and other carbonaceous nanoparticles induces carcinogenic effects (Heinrich et al., 1995) but - due to the issue of rat lung overload - the relevance for humans is still a matter of debate (ILSI, 2000). Recently, the International Agency for Research on Cancer (IARC) Working Group has concluded that there is sufficient evidence in humans for the carcinogenicity of diesel exhaust (Group 1).

This work is part the TOBICUP (TOxicity of BIomass Combustion generated Ultrafine Particles) project which aimed at providing the composition of ultrafine particles emitted by wood combustion and elucidating the related toxicity. The first part of the project focused on laboratory tests in which room heaters were fed with wood and pellets of two different types and their emissions were investigated in terms of UFPs physical-chemical properties and biological effects (details in Corsini et al., 2017; Ozgen et al., 2017). The second part of the project investigated the composition and toxicity of UFPs emitted in outdoor air

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