

The possible impact of “sequential co-exposure” on ozone associated adverse health effects. Preliminary data from cumulative cross sectional and prospective studies in Milan.

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Aim of the study: The aim of this study has been to report on preliminary data concerning the possible impact of ozone as a single pollutant or together with other pollutants in the occurring of clinically relevant diseases.

Methods: Two monitoring campaigns (July-September 2008) (December-January 2009) covering the entire City area were performed in Milan, Italy. In particular, gaseous pollutants, - O₃, NO₂, CH₆H₆ - were monitored using passive samples (PASSAM, CH) in 50 different urban sites. Recorded values were processed by a GIS based geostatistical software to give the concentration profiles for the 3 selected pollutants. Fine particulate matter (PM₁₀) concentrations were measured in 5 urban sites representative of different areas of the City by gravimetric samplers. The heavy metal composition was analyzed by XRF techniques, during a 30-day-period.

Results: Benzene and NO₂ were representative of traffic emission, in particular during the Summer period. Both NO₂ and benzene (representative of traffic emission) showed elevated values in sites with intense vehicular traffic, as in the highly congested city centre and along the heavy-traffic roads. Ozone showed an opposite behaviour, being higher where NO_x and benzene were lower.

In particular, Ozone values were higher inside the city gardens and parks. In fact, in vegetation-rich areas, the absence of traffic scavenging is summed up to the vegetation-released ozone precursors. Ozone average concentrations seldom reached values of more than 90 µg/m³ (Fig. 1).

The greatest exposure was found in the City park, where ozone precursors were abundant and traffic related scavengers were relatively scarce. Close to City edge, exposures were lower, due to the reduced quantity of precursors and the increased quantity of traffic related scavengers. City center had an intense vehicular traffic, the emission of which contributed to the local ozone consumption. Interestingly, there was a reduction in ozone concentration during the first 3 weeks of August, in concomitance with the population reduction, because of Summer holidays, and a subsequent increase in September (Fig.1).

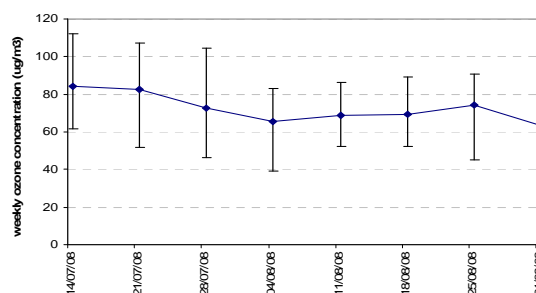


Figure 1. Weekly means of ozone concentration recorded by passive samplers at 50 different sites within the city of Milan in the summer 2008.

Conclusions: Clinical effects from environmental pollution can be considered as the end result of a complex mixture, including variable concentrations of various pollutants. In the occurrence of the health outcome not only PM_{2.5} and/or PM₁₀, but also gaseous pollutants (ozone, NO_x, SO₂) and biological components cooperate together through concomitant or sequential co-exposure. A “sound” possibility for ozone to be dangerous is to be responsible for “sequential coexposure”, i.e. to fill the gap, to make damage even during “the summer window”, which could otherwise permit logical and physiological repair of previous damages, the ones that have been accumulated in the various cells, epithelia, because of other pollutants, the effect of which is more prevalent in Winter, or during Spring or Autumn. In this way, Ozone provides a continuous damage to human tissues, that will have no time left for re-epithelization, cell or tissue repair, due to the uninterrupted activity of multiple pollutants, each one having a preferred season, but also all acting together and in ordered sequence to guarantee a continuous damage. In this respect, not only concomitant co-exposure with SO₂ and other pollutants from combustion sources, but also sequential exposure, after damage caused by PM₁₀, PM_{2.5}, pollen and bacteria, could offer a “sound” pathophysiological working hypothesis for a causative role of ozone in the occurrence of pollution related adverse effects.

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