LUNG INFLAMMATION AMONG CHILDREN WITH ASTHMA EXPOSED TO INDUSTRIAL AND TRAFFIC POLLUTION

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Background and Aims: Children with asthma are susceptible to the effects of ambient air pollution, including increased symptoms and lung inflammation. Industrial emissions are an important source of air pollution in many areas. Oil refinery emissions are of particular concern as they comprise a complex mixture of organic and inorganic pollutants. We examined associations between fractional exhaled nitric oxide (FeNO), an indicator of airway inflammation, and exposure to oil refinery and traffic emissions on the health of children with asthma in Montreal, Canada.
Methods: We recruited 69 children (age 8-13 years) with a physician diagnosis of asthma from schools and an asthma clinic. Subjects participated in the panel study for 10 consecutive days between October 2009 and April 2010. We measured personal exposures to sulphur dioxide (SO_2) using Ogawa passive samplers (Ogawa & Company, Pompano Beach, FL, USA) and fine particulate matter ($PM_{2.5}$) using Harvard Personal Environmental Monitors (HPEM, BGI, MA, USA) and continuously using the personal DataRAM (pDR-1200, MIE Inc, Bedford, MA). Filters were also analyzed for metals associated with oil refinery emissions. We recorded online FeNO daily using the NIOX MINO monitor (Aerocrine, Solna, Sweden) and collected participants' reports on health, medication use, and activities. Linear mixed-effects regression models with autoregressive correlation structure were used to estimate the association between FeNO and pollutant exposure.
Results: The geometric mean of FeNO was 20.9 ppb (geometric standard deviation: 2.2). Mean (SD) personal exposure to $PM_{2.5}$ was 9.5 (13.4) $\mu g/m^3$, while for SO_2 it was 0.81 (3.21) ppb. Preliminary models indicate that an increase of 10 $\mu g/m^3$ in previous 8-hour personal exposure to $PM_{2.5}$ was associated with a 1% (95% CI: 0.1-2.0%) increase in FeNO, adjusted for corticosteroid use, age and sex.
Conclusions: Preliminary results indicate an association between personal exposures to $PM_{2.5}$ and increased airway inflammation in children with asthma.