Abstract

In everyday life, humans are exposed to toxic substances of anthropogenic origin. These substances can also be found in the ambient air and their impact poses a long-term risk for human health. Respirable particulate matter (PM) of aerodynamic diameter < 2.5 μm (PM2.5) is intensively studied, along with carcinogenic polycyclic aromatic hydrocarbons (PAHs), bound to it, such as benzo[a]pyrene (B[a]P), a reference carcinogenic PAH. Owing to small size, PM2.5 can penetrate the human body primarily via the airways and represent an increased health risk compared to larger particles. The negative health impacts of anthropogenic PM2.5, generated e.g. by fossil fuel combustion, are linked with its small size, relatively large surface, as well as with PAHs and other substances adsorbed on PM surface. PAHs, generated by an incomplete combustion of organic matter, can enter organism either via ingestion of contaminated food, water or via inhalation of polluted air. PAHs affect organisms via genotoxic, mutagenic, carcinogenic, embryotoxic and other adverse effects. One of the common denominators of these effects is oxidative stress, which is also considered to be the main mechanism of action caused by PM in the human organism. Oxidative damage induced by reactive oxygen species (ROS) may affect any cellular macromolecule. Since PAHs can cross the placenta, air pollution may impact a developing fetus via maternal exposure. Such exposure has been associated with preterm birth, low birth weight along with intrauterine growth restriction and potentially respiratory problems in childhood and adulthood. Due to the heterogeneous composition of polluted air, exposure evaluation and interpretation of the causal link between exposure and biological consequences is very complicated. Moreover, the resulting exposure of an individual is a multifactorial process that can be influenced by air pollution, genetic predisposition, lifestyle (i.e. smoking, diet), socio-economical and other environmental factors. The aim of this study was to analyze the impact of air pollution on oxidative DNA damage [8-oxo-7,8-dihydro-2'-deoxyguanosine (8-oxodG)] and lipid peroxidation [15-F_2t-isoprostane (15-F_2t-IsoP)] in urine and blood plasma from non-smoking mothers and their newborns from two localities differing in the level of air pollution: České Budějovice (ČB) and Karviná. The ČB group was selected as a control, since the levels of pollution in this locality are significantly lower than in Karviná. Sampling of biological material (urine, plasma) was performed in two periods with different levels of air pollution: in summer 2013 (low pollutant levels) and in winter 2014 (high pollutant levels).

In both sampling periods, the subjects in Karviná were exposed to significantly higher concentration of air pollutants than in ČB ($P < 0.001$). In both localities, the concentration of air pollutants was higher in winter season than in summer ($P < 0.001$). Biomarker levels (8-oxodG, 15-F_2t-IsoP) were expected to increase with increasing concentration of air pollutants. While in newborns from Karvina (winter 2014) the levels of 8-oxodG were significantly increased ($P < 0.001$) in comparison with ČB, in mothers from Karviná oxidative DNA damage levels were significantly decreased in comparison with the control locality ($P < 0.05$) in the same period. This discrepancy may be explained by adaptation of the adult organism to adverse environmental conditions and subsequent development of protective mechanisms. The levels of 15-F_2t-IsoP generally followed the same trend as 8-oxodG concentrations. The exception was observed for lipid peroxidation in samples from newborns collected in summer 2013, when 15-F_2t-IsoP levels were significantly higher in the control group ($P < 0.001$). This could be a result of the effect of other independent factors (e.g. type of delivery or anesthesia applied during delivery). Multivariate regression analysis of the effect of air pollution on oxidative stress in newborns from Karvina showed PM2.5 concentrations to be a significant predictor for 8-oxodG levels. Exposure to PM2.5 and B[a]P significantly affected lipid peroxidation.

This work demonstrates the application of 8-oxodG and 15-F_2t-IsoP as biomarkers of exposure to air pollution in newborns. In conclusion, the results suggest that air pollutants such as PM2.5 and B[a]P affect oxidative damage in newborns in a polluted regions. This effect was not seen in mothers.