

## Genotoxic effects in workers of the plastic recycling industry exposed to micro- and nanoplastics

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Micro- and nanoplastics (MNPLs) pollution constitutes one of the greatest current threats for the environment and human health. In the present study, we investigated the internal exposure and early health effects induced by MNPLs exposure. Blood and buccal samples were taken from 17 male employees of a plastic recycling company (mean age, 44.3±3.0 y) and 21 male volunteers (45.6±2.7 y) non-occupationally exposed to MNPLs. Workers also provided an exhaled breath condensate (EBC) sample. The study was approved by the local ethics committees and all participants provided written informed consent. MNPLs were characterised by Confocal Raman Microscopy (CRM). Local chromosome damage was assessed by the micronucleus assay in buccal cells, whereas systemic DNA and chromosome damage was assessed from blood cells by means of the comet and micronucleus assays. Furthermore, the levels in plasma of ten pro-inflammatory cytokines and chemokines were also investigated. The number of particles in blood increased by 114% in recycling workers compared to the control group. Workers also showed a higher percentage of particles below 1 µm (40% vs 38%) and 1-10 µm (58% vs 54%) diameter, but a lower percentage of particles above 10 µm diameter (2% vs 6%). Particle composition was similar in both groups, except for polydimethylsiloxane, an additive used in the manufacture of thermoplastics, which was only detected in the group of workers. Furthermore, workers showed a statistically significantly higher rate of DNA damage than the corresponding control group, although the damage was not connected to oxidative stress, as well as a significantly higher level of interleukin (IL)-1 $\alpha$ , IL-1 receptor antagonist (IL-1Ra) and C-X-C motif chemokine ligand 5 (CXCL5). No significant differences were detected for the other genotoxic or inflammatory biomarkers. In conclusion, exposure to MNPLs during recycling-related processes seems to induce alterations of some pro-inflammatory cytokines and DNA damage.

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