In vitro toxicity of airborne emissions from combustion of graphene nanoplatelet-enabled epoxy nanocomposites

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Combustion is one of the processes occurring at the material's end of life. The combustion process can release the embedded nanoparticles from the nanocomposite's matrix and might transform the nanoparticle's properties[1]. An increasing use of graphene nanoplatelets (GNPs) as an additive in commercial products raises concerns about the potential risks of the released particles, especially human exposure to airborne fraction of the released GNPs since inhalation is one of the major exposure routes. Despite many studies about the release of nanoparticles induced by combustion [2], [3], the hazard assessment of the GNPs released from the combustion is still limited. Therefore, this study aims to characterize the particulate and gaseous emissions from the combustion of neat epoxy (EP) and GNP-enable epoxy composite (EP-GNP) and evaluate the biological effects of the emissions on human alveolar epithelial cells (A549) cultivated at air-liquid interface for up to 96 h after the combustion exposure. The particle modal sizes of the emissions from both EP and EP-GNP were in the respirable range of 4 µm. We found volatile organic compounds and polycyclic aromatic hydrocarbons (PAHs) in the soots and gases from the emissions. After the treatment with the emissions from both EP and EP-GNP, the quantification of lactate dehydrogenase suggested no adverse effects on cell membrane integrity. Cells treated with the emissions from EP-GNP, but not EP, showed a significant reduction in mitochondrial activity at 24 h time point. However, this effect was transient and values recovered to those of filtered air controls after 96 h. Release of the inflammatory chemokines/cytokines MCP-1 and GM-CSF was increased at 24 h time point after exposure to both EP and EP-GNP, while the values were dropped to control levels at 96 h. Expression of CYP1A1 gene, associated with metabolic activation of PAHs, strongly increased with exposure to both EP and EP-GNP at both time points. Exposure to EP-GNP emissions caused slightly higher CYP1A1 expression at 24 h than exposure to EP emissions. Our results reveal potential enhanced toxicity from GNP nanofillers, which should be considered in future risk assessment studies and the safe design and use of GNP-enabled nanocomposites.

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