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Respiratory hazard identification of combined exposure to gasoline exhaust and respirable volcanic ash in a multicellular human lung model

Abstract *

Urban populations situated proximal to active volcanoes can experience sustained exposure to anthropogenic pollution (*e.g.* vehicle exhaust particles) as well as to volcanic ash (VA) during and following an eruption. Inhalation of anthropogenic pollution is known to cause, or exacerbate, respiratory and cardiovascular diseases. Furthermore, exposure to VA can exacerbate such disease states. Understanding the impact of a combined exposure of anthropogenic pollution and VA to human health, however, remains limited.

The aim of this study was to assess the biological impact of combined exposure to respirable VA and freshly-generated complete gasoline exhaust. A multicellular human lung model (an epithelial cell-layer with human blood monocyte-derived macrophages and dendritic cells) cultured at the air-liquid interface was exposed to diluted exhaust (1:10) continuously for 6hrs, followed by immediate exposure to respirable VA (0.54±0.11 and 0.39±0.05 $\mu g/cm^2$ from the Soufrière Hills volcano, Montserrat and Chaitén, Chile, respectively) as a dry powder for 18hrs. Subsequently, cells were exposed again for 6hrs to diluted exhaust, and a final 18hr incubation. Cultures were then assessed for cytotoxic, oxidative stress and (pro-)inflammatory responses.

Results indicate that, at sub-lethal concentrations, no significant changes in oxidative stress markers (HMOX1, NQO1) or (pro-)inflammatory mediators (IL-8, IL-1β) were evident at the gene-level for either ash type. The lack of any (pro-)inflammatory response is in contrast to that observed with diesel exhaust particles combined with Soufrière Hills VA, under similar experimental conditions. In summary, the combined vehicle exhaust and VA exposure has a limited biological impact to lung

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Affiliations and Authors *

Author Information
Ines Tomašek (Presenting)

Affiliations
Institute of Hazard, Risk and Resilience, Department of Earth Sciences, Durham University, Durham, United Kingdom
Bionanomaterials group, Adolphe Merkle Institute, University of Fribourg, Fribourg, Switzerland

Author Information
Claire J. Horwell

Affiliations
Institute of Hazard, Risk and Resilience, Department of Earth Sciences, Durham University, Durham, United Kingdom

Author Information
Christoph Bisig

Affiliations
Bionanomaterials group, Adolphe Merkle Institute, University of Fribourg, Fribourg, Switzerland

Author Information
David E. Damby

Affiliations
United States Geological Survey, Menlo Park, USA

Author Information
Pierre Comte

Affiliations
Bern University for Applied Sciences, Nidau, Switzerland

Author Information
Jan Czerwinski

Affiliations
Bern University for Applied Sciences, Nidau, Switzerland

Author Information
Alke Petri-Fink

Affiliations
Bionanomaterials group, Adolphe Merkle Institute, University of Fribourg, Fribourg, Switzerland
Chemistry Department, University of Fribourg, Fribourg, Switzerland

Author Information
Martin J.D. Clift

Affiliations
In Vitro Toxicology group, Swansea University Medical School, Swansea, United Kingdom

Author Information
Barbara Drasler

Affiliations
Bionanomaterials group, Adolphe Merkle Institute, University of Fribourg, Fribourg, Switzerland

Author Information
Barbara Rothen-Rutishauser

Affiliations
Bionanomaterials group, Adolphe Merkle Institute, University of Fribourg, Fribourg, Switzerland

Author Approval *