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The bioreactivity of the sub-10 μm component of volcanic ash: Soufrière Hills volcano, Montserrat

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ABSTRACT

With the recent eruption of the Icelandic volcano Eyafallajökull and resulting ash cloud over much of Europe there was considerable concern about possible respiratory hazards. Volcanic ash can contain minerals that are known human respiratory health hazards such as cristobalite. Short-term ash exposures can cause skin sores, respiratory and ocular irritations and exacerbation of pre-existing lung conditions such as asthma. Long-term occupational level exposures to crystalline silicon dioxide can cause lung inflammation, oedema, fibrosis and cancer. The potential health effects would be dependent on factors including mineralogy, surface chemistry, size, and levels and duration of exposure. Bulk ash from the Soufrière Hills volcano was sourced and inhalable (<2.5 μm) ash samples prepared and physicochemically characterised. The fine ash samples were tested for bioreactivity by SDS-PAGE which determined the strength of binding between mineral grains and lung proteins. Selected proteins bound tightly to cristobalite, and bound loosely to other ash components. A positive correlation was seen between the amount of SiO_2 in the sample and the strength of the binding. The strength of binding is a function of the mineral's bioreactivity, and therefore, a potential geo-biomarker of respiratory risk.

1. Introduction

When we are threatened with potential respiratory hazards, such as airborne fine volcanic ash, a health risk assessment needs to be undertaken immediately, so that action can be taken and the risk minimised. The fast options available are a geological (usually mineralogical) analysis of the dust and making comparisons with similar dusts, or chemo-biological screening assays (Fig. 1). These rapid assays are based on chemical, biological components or cell reactions. Each of these screening assays have their advantages and disadvantages, however, different assays can give quite different results for the same dusts; which is problematic for any meaningful risk assessment. Different assays target different potential causes of adverse health effects, such as generation of reactive oxygen species (ROS), bioreactive particle surfaces, and leachable (e.g. toxic metals) components. In addition to the leachable transition metals, concerns have also been raised about interactions between water-soluble components on particle surfaces and bronchoalveolar fluids [1], with elements such as yttrium and the lanthanides crystallising as phosphates in the interstitial lung spaces. In extreme occupational exposures this can result in dendriform pulmonary ossification forming in the lung [2]. Trace element values for the Montserrat volcanic ash is given in BéruBé et al. [3]. The main concerns with volcanic ash are minerals with bioreactive surfaces, although there are minor concerns about iron levels in some ashes.

The Soufrière Hills volcano is an active stratovolcano found on the Lesser Antilles island of Montserrat. The volcano has been erupting since July 1995 and continues to be active today [4–6]. Due to its continued eruptive state it is important to determine the long-term health risks of the volcanic ash [7]. The volcanic activity includes ash and steam venting, occasional pyroclastic flows and explosive phreatic eruptions [8–10]. The Soufrière Hills volcanic dome lava is predominantly a porphyritic andesite [4] and consists of approximately 45–55 wt.% phenocrysts (>300 μm), 15–20 wt.% microphenocrysts (300–100 μm) and 20–30 wt.% microlites (<100 μm) and a residual high silica rhyolite glass (76–79% SiO_2) [11]. The phenocrysts predominantly comprise of plagioclase, hornblende, orthopyroxene, titanomagnetite and minor quartz, whereas the microphenocrysts comprise clinopyroxenes, apatite and ilmenite [12]. Recent research suggests that gas-charged magma injection could act as a trigger for dome collapse [13]. The crystalline SiO_2 in the ash includes cristobalite, the high-temperature low-pressure polymorph, and is thought to be the most bioreactive SiO_2 strain as it is most likely to produce

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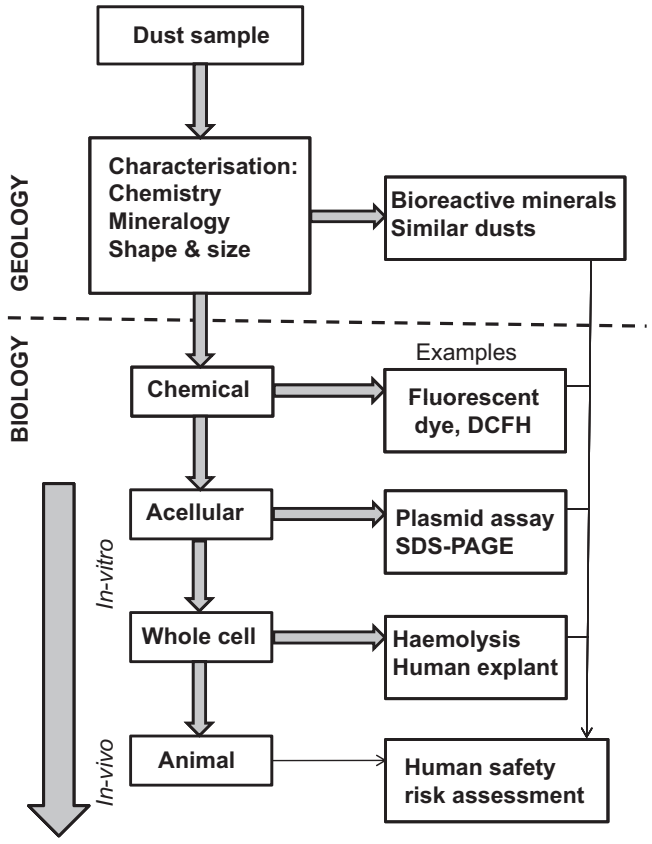

Fig. 1. A flow diagram of the pathways to assess mineral dust bioreactivity and human safety risk assessment.

surface radicals [14]. When particulate material (PM) 10 μm or less in diameter (i.e. PM10), is erupted or remobilised into the atmosphere by natural or anthropogenic disturbance (such as clean-up operations after eruptions), it can be inhaled and lodge into the upper (nose and mouth), lower (thoracic cavity) or distal (alveolar region) respiratory tracts (RT; Fig. 2). Coarse particles (2.5–10 μm) are only able to penetrate into the extra-thoracic region of the upper RT. Finer particles (2.5–1 μm), readily translocate into the lower RT, whereas ultrafine particles (<1 μm) can penetrate even deeper into the lung, reaching the alveoli, where oxygen and carbon dioxide exchange [15].

In the human lung, the first line of defence against inhaled PM such as volcanic ash is the specialised epithelial surface of the conducting airways [16,17]. This is coated in epithelial lining fluid (ELF) containing defence proteins (e.g. surfactants, mucous, antioxidants and anti-bacterial molecules). It is these molecules that either react against, or bind to, the surface of any foreign bodies [18]. The strength of the response of these chemicals can be seen as a measure of the bioreactivity of the respired particle [19].

In the lower RT, bronchial epithelial cells (e.g. ciliated cells) also have hair-like cilia, which beats a layer of mucus (adhesive glycoprotein secreted by goblet cells) that encapsulates the dust particles and carries them up the pharynx by the muco-ciliary escalator to be swallowed (Fig. 2). In the distal RT (i.e. alveolar region) mobile cells called alveolar macrophages are the main defence against airborne particles (Fig. 2) [17,20]. These cells are responsible for the phagocytosis (ingestion) of micro-organisms and PM that has been inhaled.

For PM that is highly respirable and able to deposit into the distal lung region, the first visible damage response takes place at the alveolar surface with abnormal leakage of fluid into the alveolar gas spaces. Swelling (oedema), the first stage of inflammation, is caused by secretions from damaged cells and release of inflammatory mediators (e.g. cytokines and chemokines) into the alveolar air spaces. Inflammation may become chronic and this can lead to increased proliferation of epithelial cells (i.e. hyperplasia) and in the longer-term, pulmonary fibrosis or emphysema (i.e. chronic obstructive pulmonary disease; COPD) through metaplasia (i.e. abnormal transformation of columnar cells into squamous cells; squamous metaplasia) of the airway epithelia [21–23].

It has been established that crystalline silica is more harmful in the lung when the particles have 'fresh' surfaces created by fracturing, resulting in a charged surface [24]. Thus, the highly attritional and corrosive environment of an erupting volcano should in theory generate particles with reactive surfaces that would interact with the lung cells and fluids. The alveolar macrophages, which are involved in cleaning-up of the lung, perform phagocytosis and

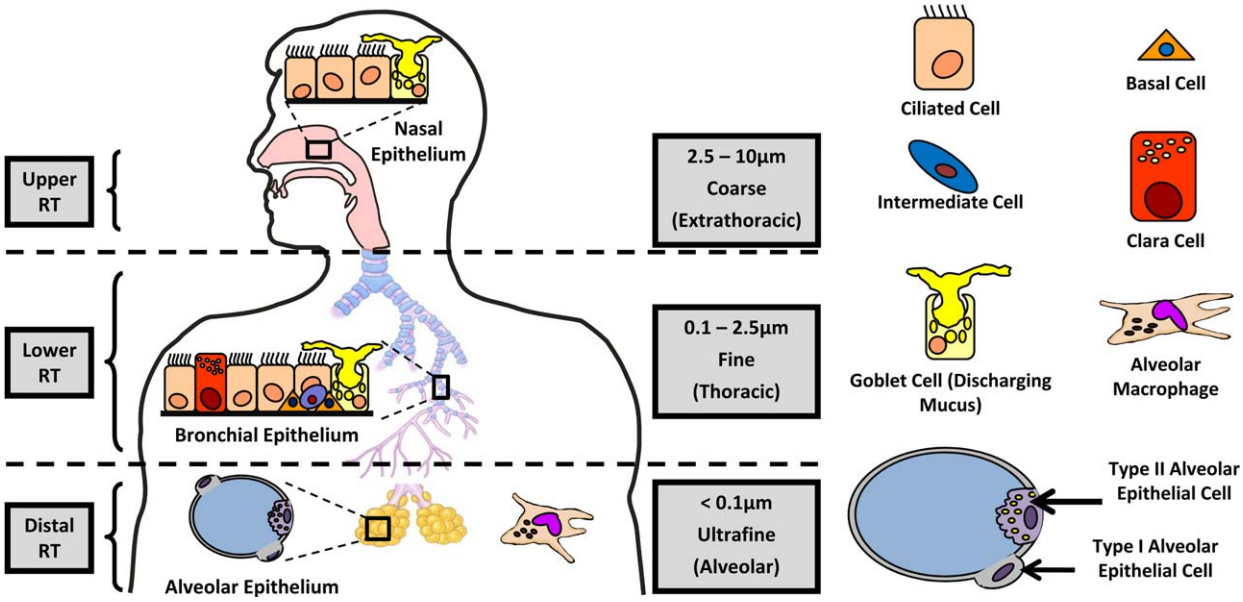

Fig. 2. Human respiratory tract.