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REVIEW ARTICLE

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The respiratory health hazards of volcanic ash: a review for volcanic risk mitigation

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Abstract Studies of the respiratory health effects of different types of volcanic ash have been undertaken only in the last 40 years, and mostly since the eruption of Mt. St. Helens in 1980. This review of all published clinical, epidemiological and toxicological studies, and other work known to the authors up to and including 2005, highlights the sparseness of studies on acute health effects after eruptions and the complexity of evaluating the long-term health risk (silicosis, non-specific pneumoconiosis and chronic obstructive pulmonary disease) in populations from prolonged exposure to ash due to persistent eruptive activity. The acute and chronic health effects of volcanic ash depend upon particle size (particularly the proportion of respirable-sized material), mineralogical composition (including the crystalline silica content) and the physico-chemical properties of the surfaces of the ash particles, all of which vary between volcanoes and even eruptions of the same volcano, but adequate information on these key characteristics is not reported for most eruptions. The incidence of acute respiratory symptoms (e.g. asthma, bronchitis) varies greatly after ashfalls, from very few, if any, reported cases to population outbreaks of asthma. The studies are inadequate for excluding increases in acute respiratory mortality after eruptions. Individuals with pre-existing lung disease, including asthma, can be at increased risk of their symptoms being exacerbated after falls of fine ash. A comprehensive risk assessment, including toxicological studies, to determine the long-term risk of silicosis from chronic exposure to volcanic ash, has been undertaken only in the eruptions of Mt. St. Helens (1980), USA, and Soufrière Hills, Montserrat (1995 onwards). In the Soufrière Hills eruption, a long-term silicosis hazard has been identified and sufficient exposure and toxicological information obtained to make a probabilistic risk assessment for the development of silicosis in outdoor workers and the general population. A more systematic approach to multi-disciplinary studies in future eruptions is recommended, including establishing an archive of ash samples and a website containing health advice for the public, together with scientific and medical study guidelines for volcanologists and health-care workers.

Keywords Volcanic ash · Respiratory · Health · Hazard · Risk mitigation · Review

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Introduction

Interest in the health consequences of the inhalation of volcanic ash has grown ever since Mt. St. Helens emitted, in a cataclysmic eruption, an extensive ashfall across populated areas of the north-western USA on May 18, 1980. Since then, a limited number of studies of variable quality have been performed on the health of populations affected by eruptions and on ash in laboratory settings. In this review, we have identified the most informative published papers and synthesised their conclusions to summarise the current state of knowledge and the limitations of research so far. This work is intended to inform volcanologists, health workers and others engaged in risk mitigation in areas of active volcanism worldwide.

Nine percent of the world's population (455 million people) live within 100 km of an historically active volcano (Small and Naumann 2001). Of all eruptive hazards, ashfall can affect the most people because of the wide areas that can be covered by fallout (Blong 1996). Although eruptions are often short-lived, ashfall deposits can remain in the local environment for years to decades, being remobilised by human activity or simply re-suspended by wind. Thus, advice on ash hazards is urgently needed after

an eruption to make informed judgements on the health risk to the population, as well to undertake risk assessments on those occupationally exposed to the ash. It is also important to be aware that volcanic ash forms the soils of many parts of the world (Ping 1999), exposure to which may occur in dust storms (Hefflin et al. 1994; Schwartz et al. 1999) and in agriculture, construction work and quarrying.

Volcanic ash was once widely regarded as relatively inert and non-hazardous to health, but new attitudes need to reflect developments in occupational medicine and the role of particles in air pollution (WHO 2000). This review focuses on the respiratory effects of ash particles, so other health hazards such as the chemical impacts of ash on water quality (including the toxicity of ash for grazing animals (Cronin et al. 2000)), trauma to the eye from ash particles (Fraunfelder et al. 1983) and the mental health consequences of ashfall events (Shore et al. 1986), will not be considered here. Deaths and trauma from roof collapses due the weight of the ash (Spence et al. 1996) are also not included. The physico-chemical characteristics of ash particles are discussed as these are important factors in the toxicity of natural dusts, but the potential irritant effects of adsorbed gases and aerosols are also omitted as few published studies have adequately addressed these.

Methods

We searched the volcanological, medical and toxicological literature for all references to volcanic ash and the health effects of eruptions (e.g. biomedical journal databases Medline (Ovid 1966 to 2005) and Embase (1988 to 2005), the ISI Web of Science (1981 to 2005) and the geological database GeoRef (1785 to 2005)). All potentially relevant studies with at least an abstract in English were obtained. It has been possible, for completeness, to include all the references found, though many papers are of limited quality. We have also included published work of our own and some additional material where it may help the reader's understanding. The main types of published studies are as follows.

1. *Clinical studies*: Clinical studies of individual patients involve diagnosis by chest X-ray, and lung function and capacity tests (spirometry). The most common spirometry tests are (1) forced vital capacity (FVC), defined as the maximum volume of air that can be exhaled as rapidly as possible following maximal inspiration. This is a measure of restriction of lung expansion such as can be caused by silicosis and (2) forced expiratory volume in one second (FEV_1), which is the maximum volume of air that can be breathed out in one second and provides a measure of airflow obstruction, as occurs in asthma and chronic obstructive pulmonary disease (COPD).
2. *Epidemiological studies*: Epidemiological methods are used to quantify the amount of disease in the population and to investigate associations between the exposure and the disease in order to elucidate causal factors. The main epidemiological study designs used for the assessment of the health effects of dusts are as follows: (1) 'Case-control' studies, which involve the enrolment of subjects with the disease of interest (cases), who are compared with subjects without the disease, or controls (characteristics of the individuals, and details of previous exposure to dust, together with other risk and confounding factors, such as smoking, are obtained by questionnaire); (2) 'prospective' or 'cohort' studies, which involve a well-defined group of people who have, or have had, a common exposure to dust, and are followed for the incidence of new diseases over time and (3) 'cross-sectional' or 'prevalence' studies which examine the health and exposure status of a population at a specific time such as at a defined period following an eruption.

Fig. 1 Ash exposure for a gardener on Montserrat, being measured using a DustTrak instrument which provides a direct PM_{10} reading. Note the clouds of fine ash being re-suspended in the breathing zone of the worker. The face masks are those recommended for protection against inhaling respirable ash particles (see IVHHN recommended dust masks: www.ivhhn.org)

In any risk assessment of the health effects of volcanic ash, the population's exposure must be incorporated and linked to the findings of health studies (see Searl et al. 2002). An inadequate surrogate measure is the depth of ash on the ground, which is best derived from isopach maps prepared by volcanologists. Measurements of airborne ash particle concentrations (Fig. 1) are made with techniques used in occupational hygiene surveys in dusty industries or in urban air pollution studies of particulate matter.

3. *Toxicological studies*: Toxicological studies in laboratory animals (in vivo) or with cell systems (in vitro) are used to study disease mechanisms, as well as to attempt to predict the consequences of exposure to a substance in humans. In vivo experiments on ash are carried out either by intratracheal instillation (injection), or by inhalation of particles (Martin et al. 1986).

   In vitro studies using tissue cultures provide rapid approximations of the effects of minerals on cells that are involved in the expression of toxicity in vivo (Martin et al. 1986). They are, however, far removed from the biological systems of humans and animals and cannot replicate other processes which may be affecting the reaction in vivo (Martin et al. 1986).

   The dose of dust administered and exposure time of the animals to the dust are key parameters in toxicological experiments. For the volcanological audience, we have not included details of dose, exposure time and some detailed aspects of cellular tests, except where necessary (e.g. Table 4), but this information is available in the original papers.

Characteristics of ash particles

Volcanic ash is defined as pyroclasts 2 mm or less in diameter (see Heiken and Wohletz 1985, where more details can be found on how ash is formed and on its identification). This review focuses on ash from volcanic plumes, unless otherwise stated. The different physicochemical properties of ash are critical to its biological reactivity and hence its respiratory health effects, but relatively little is understood about the disease mechanisms at the cellular level (see Mechanisms of particle toxicity section).

Freshly erupted ash differs from other natural dusts in several ways. The particle surfaces are un-weathered and are, therefore, not oxidised or leached and can carry condensed volatiles such as acids, polycyclic hydrocarbons and trace metals (Horwell et al. 2003a). Fine ash particles tend to fall in roughly spherical clusters (<100 μm), which readily break up on impact in dry conditions or when resuspended by vehicles and other human activity (Gilbert et al. 1991; Sorem 1982). Electrical charges are generated on ash particles, which may affect reactions in the lung (Williamson et al. 2001). Sulphuric and other acids adsorbed from the gases in the plume (Gilbert et al. 1991) may be present on the surface of ash particles, potentially adding to the irritancy of the ash on the airways. The strong acids may react with the glass and silicate components of the ash particles, altering their surface characteristics and forming calcium sulphate and sodium chloride as precipitated coatings on the ash. The acid salts can also combine with rain in forming crusts on top of deposits which makes the ash less easily re-suspended by wind. Sulphur dioxide (SO_2) or acid aerosols can trigger asthma attacks at very low concentrations in asthma patients so grounding of the gas plume as a cause of respiratory symptoms always needs to be excluded.

The grain size of ash particles is of critical importance and is conventionally defined in terms of the *aerodynamic diameter*. Particulate matter less than 10 μm diameter (PM_{10}) is classed as *thoracic*, and *respirable* if less than 4 μm (Quality of Urban Air Review Group 1996). The finer respirable particles can be breathed into the alveolar region of the lung and have the greatest toxic potential (Fig. 2). Recent research has shown that fine particles (<1 μm), and ultrafines, (<0.01 μm), are likely to be the most toxic (Expert Panel on Air Quality Standards 1995), but whether this applies to volcanic ash is not yet clear. Until recently, volcanologists did not routinely analyse PM_{10} or PM_4, thereby compounding the lack of information available for evaluating the health effects of eruptions. The reactivity of particles within the lung is related to the surface area and number of particles more than the mass of particles. It is, therefore, useful to quantify mineral assemblages in terms of number or surface area percent as well as weight percent (Horwell et al. 2003b). Figure 3 shows scanning electron microscopy images of ash from four volcanic eruptions. At the scale of the thoracic and respirable fractions, ash particles from eruptions of very different magma composition (e.g. basaltic and andesitic) are morphologically similar and it is not possible to

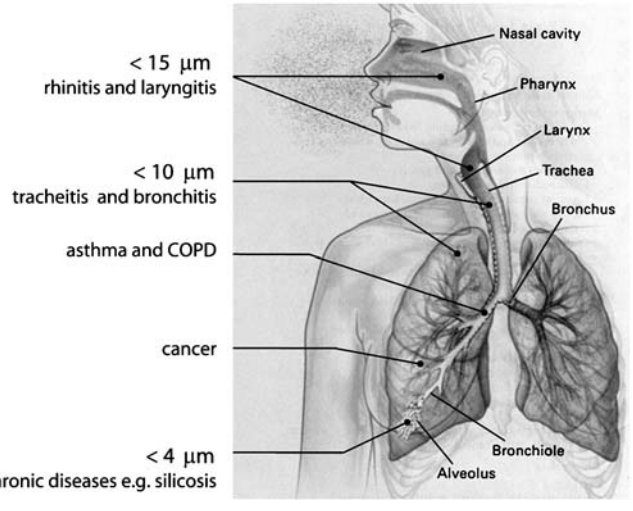

Fig. 2 Diagram of the lung showing the anatomy of the lung and airways and the penetration of ash particles of different sizes associated with their potential health effects. Modified from Beckett (2000)

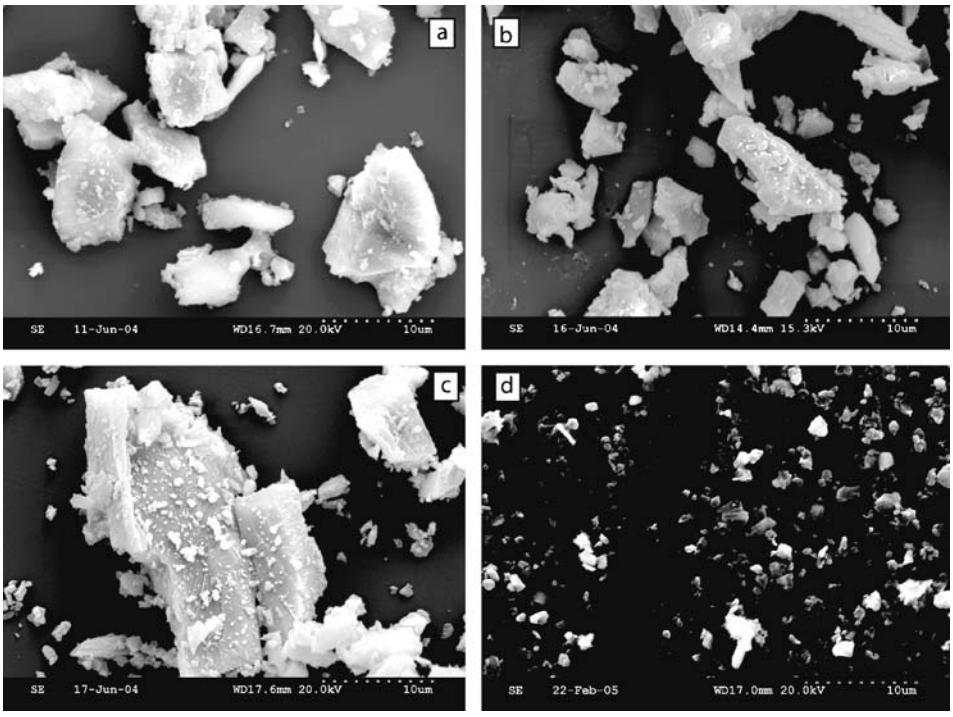

Fig. 3 SEM images of volcanic ash particles from four different eruptions. **a**) Cerro Negro, erupted 30 November 1995, basaltic; **b**) Mt. St. Helens, erupted 18 May 1980, dacitic; **c**) Sakurajima, erupted 1 January 1994, andesitic; **d**) Vesuvius, erupted 25 August AD79, tephritic/phonolitic. See Table 1 for further information about the samples. Note that the scale bar is 10 μm for all images. The Cerro Negro and Mt. St. Helens samples appear morphologically similar despite being compositionally distinct. The Sakurajima image displays a common observational feature: sub-micron particles adhering to coarser particles. This may affect the ability for ultrafine particles to reach the deep lung. The Vesuvius image illustrates the unusually fine grain size of this sample with all particles in the image being sub-4 μm diameter

determine composition of particles simply by observing the morphology.

Some experimental and observational work has been carried out on the variation of grain size with explosivity of eruption (Heiken and Wohletz 1985). Walker (1973) showed that phreatomagmatic pyroclasts have a much higher degree of fragmentation than do magmatic pyroclasts, producing more fine ash (<1 mm). Cronin et al. (2003) showed that phreatomagmatic eruptions from Ruapehu, New Zealand, produced finer ash than magmatic eruptions. However, neither of these studies quantified the respirable (<4 μm) material produced.

Numerous studies have shown the relationship between particle size of deposits and the mechanisms of transport and deposition of ash clouds (e.g. Heiken and Wohletz 1985). The importance of the location of ash sampling with respect to the volcano and grain-size distributions for the respirable fraction of selected volcanic ash samples are shown in Table 1 (Horwell, submitted). The percentage of respirable material varies greatly between samples. This variation is, in part, due to the distance from the vent at which the sample was collected and the magnitude and explosivity of the eruption. The Soufrière Hills sample and the Mt. Sakurajima sample were collected at similar distances from the vent; the difference in quantity of respirable material between the two samples is significant, given the variety of health studies which have been carried out on populations living close to these volcanoes. Unfortunately, most studies do not provide adequate information on the collection sites of their study samples.

The morphology of the ash particles may have health significance. Insoluble particles in the form of fibres may present a respiratory hazard similar to asbestos, depending upon the fibre length and width. Hazardous fibres are those whose length–diameter ratio is greater than 3, with a diameter less than 3 μm and a length greater than 5 μm. Fresh volcanic ash in samples from recent volcanic eruptions has not been found to be fibrous (e.g. Fig. 3), but weathering and hydration can cause fibrous alteration after thousands of years (e.g. to form zeolites). Concern is

Table 1 Variations in respirable material (<4 μm diameter) with volcano, distance from vent and explosivity

Volcano	Eruption date	Distance from vent	Respirable material (cumulative volume %)	Volcanic explosivity index
Merapi, Indonesia	11–19 Jul 1998	200 m	12.7	2
Soufrière Hills, Montserrat	5 June 1999	4 km	10.7	3
Sakurajima, Japan	1 Jan 1994	4 km	0.9	3
Vesuvius, Italy	24 Aug AD79	6 km	16.9	5
Etna, Italy	4 Nov 2002	~11 km	1.8	3
Pinatubo, Philippines	4 July 1991	20 km	9.8	6
Cerro Negro, Nicaragua	30 Nov 1995	20 km	0.6	2
El Reventador, Ecuador	3 Nov 2002	90 km	4.9	4
Mt. St. Helens, USA	18 May 1980	378 km	11.7	5

Grain size distributions measured by laser diffraction with either a Malvern Mastersizer 2000 or Coulter LS Analyzer (Adapted from Horwell, submitted)

sometimes raised about the presence of fine, acicular glass shards in volcanic ash, a feature of the ash from certain explosive eruptions (e.g. Mt. St. Helens, Baxter et al. 1983). Glass shards of a few microns in length are not known to have more health significance than other morphologies of volcanic ash. Newhall and Fruchter (1986) wrote a useful introduction for health professionals, which includes an example of a fibrous rhyolitic ash from a pre-historic eruption on Lipari, Italy.

Assessment of the mineralogical composition of volcanic ash is a crucial step in health hazard assessment. Silicic volcanic ash often contains crystalline silica, as quartz, cristobalite or tridymite polymorphs. Exposure to crystalline silica is well known in industry to cause silicosis, a fibrotic lung disease; it may also be a cause of lung cancer in some workers who have developed silicosis (International Agency for Research on Cancer 1997). If present, it is the most potentially toxic mineral in volcanic ash. Cristobalite is perhaps the most toxic silica polymorph and this is particularly pertinent in volcanic settings where it may be manufactured in a volcanic dome, by devitrification of volcanic glass, or in the volcanic edifice by vapour-phase crystallisation from a hydrothermal system.

Surface properties are understood to play a role in the health effects of particles. Several studies have shown that Fe^{2+} in volcanic ash could be the catalyst for a similar toxic reaction in the lung as caused by crystalline silica (Horwell et al. 2003a; Cullen and Searl 1998; Cullen et al. 2002). This has implications for the health hazard assessment of iron-rich basaltic ash, commonly thought to be of much lower toxicity than andesitic or dacitic ash due to its lack of crystalline silica. A recent study has shown that fine-grained basaltic ash can generate hydroxyl free radicals (see Mechanisms of particle toxicity section), through interaction with hydrogen peroxide present in the lung, in greater quantities than most andesitic/dacitic ash samples (Horwell et al., submitted).

Respiratory health hazards of volcanic ash

The following summary, which is appropriate for volcanic ash and fine dusts in occupational and community settings, describes the respiratory health effects of most concern:

1. *Acute (short term) respiratory effects*: The acute manifestations observed after heavy ashfalls include attacks of asthma and bronchitis, with an increased reporting of cough, breathlessness, chest tightness, and wheezing due to irritation of the lining of the airways by fine particles. Sometimes, especially in older people, asthma attacks can be fatal. Although less dangerous, asthma attacks in children can be very frightening and lead to rapid hospitalisation. Asthma attacks are not confined to known asthma patients, as many people will not have been previously diagnosed. Inhalation of fine ash can also exacerbate previously present disease, e.g. chronic bronchitis (Baxter et al. 1983) or advanced heart problems. The acute respiratory effects are variable after ashfalls, while mortality, as an end point, is rarely examined in any of the studies.
2. *Chronic (long term) respiratory effects*: The chronic health condition of most concern is silicosis, a diffuse nodular fibrosis (scarring) of the lungs (Hendrick et al. 2002). For silicosis to occur, three main conditions have to be fulfilled: (1) a high proportion of fine particles in the ash; (2) a high concentration of crystalline silica (quartz, cristobalite or tridymite) and (3) exposure to the ash in significant amounts, typically over a period of years to decades. Early lung changes cause no symptoms and most sufferers remain in this mild category, but the condition can progress even after exposure has ceased and may lead to premature death. Most concern is for the risks to children in ashfall areas, as there is little information available on their susceptibility to silicosis. In addition, exposure to dust containing crystalline silica can cause a recurrence of previously silent tuberculosis of the lungs. Individuals who have developed silicosis may be at a higher-than-average risk of developing lung cancer, as discussed below.

   Chronic obstructive pulmonary disease (COPD) is a general reaction of the lung to dust particles following prolonged exposure, sometimes leading to the irreversible narrowing of the airways and chronic mucous hypersecretion (Calverley and Walker 2003). A form of non-specific pneumoconiosis can also develop from chronic, high exposure to silicate dusts, a condition not unlike silicosis, but less harmful.

   No human cases of silicosis or other chronic lung disorders due to volcanic ash have been reported in any of the reviewed papers, but very few of the studies included the long-term health consequences of exposure. Such studies are expensive and difficult to set up, especially in developing countries and remote communities.

Mechanisms of particle toxicity

Dust particles are always present in the air we breathe, and the body has defence mechanisms to enable particles to be removed or to attempt to counter their harmful effects. Despite extensive research, the mechanisms through which minerals, including crystalline silica, interact with the lung are not precisely known (Fubini et al. 2001). A brief summary of the current understanding is presented here to aid comprehension of the results of the toxicological experiments.

The lung comprises over 40 cell types, each with its own specialised function (Mossman 1993). Epithelial cells line the airways of the lung, with some acting to defend the lung. Fibroblasts are responsible for making collagen. Both epithelial cells and fibroblasts are target cells of mineral-induced disease. Cells of the immune system (e.g. macrophages) occur in both the airways and air spaces (Mossman 1993).

Particles are mostly trapped by mucus which covers the walls of the airways. Ciliated epithelial cells move the mucus, with trapped particles, up to the throat, where it is eliminated by swallowing or coughing (Holt 1987). Particles that penetrate the non-ciliated, alveolar region of the lung (particles in the respirable fraction, <4 μm) encounter macrophages which engulf the particles (phagocytosis) in an attempt to clear the lungs of the pathogen (Mossman 1993) and transport particles to the hilar lymph nodes, where they can be stored. However, macrophages can be detrimentally affected either by toxic particles such as crystalline silica or, alternatively, by overloading with a high burden of relatively inert particles such as titanium dioxide (Cullen et al. 2002), leading to a toxic response rather than removal of the particle.

After phagocytosis, enzymes strip the particle of adsorbed matter, leaving the surface of the particles free to react. Various surface sites on the particle, including free-radical-generating sites (see Horwell et al. 2003a), may then react with the cell contents. The cell may also react to the particle by producing an 'oxidative/respiratory burst' where it rapidly synthesises an array of toxic reactive oxygen species (ROS) in an attempt to break down the pathogen (Hansen and Mossman 1987). Crystalline silica stimulates the respiratory burst in alveolar macrophages (Vallyathan et al. 1992). The formation of so many toxic substances may lead to macrophage death.

During macrophage death, the particle and corrosive cell contents are released into the lung. The particle is then free to be engulfed again, starting a continuous cycle which may eventually stimulate persistent inflammation and abnormal production of collagen by fibroblasts, forming fibrotic nodules and eventually silicosis (Fubini and Wallace 1999). The cell contents, once in the lung, can react with cell lipids causing lipid peroxidation (oxidative deterioration of polyunsaturated fatty acids, (Halliwell 1984)) leading to further generation of radicals. Radicals may also react directly with DNA, causing strand breakage as the radicals extract an electron from the DNA (Lehnert 1993; Shi et al. 1995). Both processes may eventually yield cell mutation and cancer.

Early studies (prior to Mt. St. Helens)

Horton and McCaldin (1964) documented the impact of the 1963–1965 ashfalls from Irazu, Costa Rica, on the population of the capital, San Jose. The study was the first to be published which mentioned that volcanic ash can exacerbate pre-existing lung disease. Transient, acute irritant effects of volcanic ash and gas on the eyes and upper respiratory tract, as well as the exacerbation of chronic lung diseases such as chronic bronchitis, were reported during heavy ashfalls, and for some days after. Symptoms were especially severe when exposure was concurrent with a chest infection. Around 50% (by particle number) of the ash was <5 μm in diameter, but only a small proportion was fresh material. Horton and McCaldin found that the ash contained 1% crystalline silica and considered the possibility that freshly erupted volcanic ash could contain harmful amounts of respirable crystalline silica.

The onset of transient asthmatic bronchitis in a number of previously healthy infants was reported following the eruption of La Soufrière volcano, St. Vincent, West Indies in 1979 (Leus and West 1981). This was the first publication to refer to asthma following an ash eruption.

Several examples of tuffaceous pneumoconiosis have been documented from quarry/mine workers (Babolini et al. 1978; Dashtoyan et al. 1980; Faraone and Majori 1958). Pneumoconiosis was found in pumice stone workers in Lipari, Italy, within a few years to decades of initial exposure (Faraone and Majori 1958); the pumice dust is highly respirable in size (78–93% <5 μm) with 1–5% crystalline silica reported (Babolini et al. 1978). Several studies report health effects from breathing transformation products of weathered volcanic materials (e.g. fibrous, asbestiform minerals) such as fibrous zeolites including erionite. Malignant mesothelioma, a rapidly fatal cancer usually only associated with asbestos exposure, has been reported in the Cappodocia area of central Anatolia, Turkey (e.g. Baris et al. 1979). More recently, a weathered, fibrous volcanic amphibole has been linked to cases of mesothelioma at Biancavilla, a village on Mt. Etna, Sicily (e.g. Paoletti et al. 2000).

Mt. St. Helens, USA

The eruption which first brought the health effects of volcanic ash to serious scientific attention was Mt. St. Helens on May 18 1980, when areas of central Washington State experienced around 10 cm of ashfall, with adjacent states such as Idaho, receiving sufficient ash to create acute anxiety. The eruption occurred before any contingency plans had been drawn up to deal with volcanic-ash-related hazards (Buist and Bernstein 1986), but health investigations were initiated within a few days of the eruption (Baxter et al. 1981). Asthma was of greatest concern, but health workers were also worried about the long-term risk of silicosis from the respirable ash which contained crystalline silica.

Characterisation of the ash

Initially, there were several conflicting opinions on the composition of the Mt. St. Helens ash. The first analyses found crystalline (or 'free') silica content in over 60% of the ash by weight (wt%). This erroneous result was, in fact, the SiO_2 oxide percentage (SiO_2 in bulk samples of ash is expected to be around 60–65% for a dacitic composition) rather than the concentration of the crystalline silica particles in the ash. This confusion led to widespread speculation that silicosis posed a serious risk to the population. Several other analyses proved unreliable, with reported crystalline silica levels ranging from not detectable, up to 20 wt% (Dollberg et al. 1986). The differing reports were due to disagreements in the

interpretation of XRD diffraction patterns since the major diffraction peak for cristobalite is masked by the adjacent plagioclase feldspar peak. Further analyses showed that quartz, cristobalite and tridymite comprised ~7 wt% of the <10 μm fraction, of which 4% was cristobalite (Dollberg et al. 1984; Fruchter et al. 1980; Gage and Farwell 1981; Green et al. 1982). These results were obtained by wet-sieving the samples to retrieve the <10 μm fraction and then dissolving all non-crystalline silica minerals with phosphoric acid (the Talvitie method (Talvitie 1951)) so that XRD patterns could be clearly interpreted.

The mineralogy of the Mt. St. Helens ash was determined using optical microscopy, scanning electron microscope with energy-dispersive X-ray (SEM-EDX) and X-ray diffraction (XRD) (Davis et al. 1981). Analyses of the ash composition were carried out by Sarna-Wojcicki et al. (1981a,b,c), and the morphology of the ash was determined using the methods reported by Heiken (1972). The Mt. St. Helens ash contained varying ratios of glass and crystalline material depending on the distance of deposition from the volcano. Of particular importance for the potential respiratory effects was the finding that over 90% of the particles, by count (equivalent to 10% by weight (Craighead et al. 1983)), were <10 μm diameter (Fruchter et al. 1980). This was determined by dry sieving in conjunction with aerodynamic particle-size separation and SEM.

Epidemiological and clinical studies

The epidemiological and clinical studies carried out following the Mt. St. Helens eruption are summarised in Tables 2 and 3. Baxter et al. (1981) assessed the acute impact of the ashfall. A hospital surveillance system showed a 3–5-fold increase in emergency room visits and 2–3-fold increase in hospital admissions for respiratory conditions in the week following the eruption in the two hospitals in Yakima, mainly in people with pre-existing asthma and respiratory diseases. One of us - PJB - and his colleagues reviewed the mortality statistics at the time and no obvious increases in daily deaths were observed. Total suspended particulates (TSP), a measure of inhalable material (equivalent to material <100 μm in diameter), averaged 3,000–33,000 μg m^{-3} daily, falling to 50–250 μg m^{-3} only after rain fell a week after the eruption. Figure 4 shows the increase in emergency room visits during May 1980 (Baxter et al. 1983), and the corresponding rise in TSP above the Environment Protection Agency Emergency Level during this time.

Two epidemiological studies examined the relationship between ash exposure and the exacerbation of pre-existing respiratory problems (Baxter et al. 1983). The first was a case-control study with patients who had presented with asthma and acute bronchitis in the most heavily impacted communities in the 4 weeks following the eruption. In the second study, patients with known chronic respiratory disease were contacted. Their experiences were compared with those of similar patients from an unaffected rural community. The studies concluded that pre-existing chronic respiratory diseases are important risk factors for adverse respiratory reactions in ashfalls, with one third of the patients experiencing worsened symptoms following ash exposure compared to a much lower proportion amongst the healthy controls. Half of those patients reported that they could not resume their usual activities for at least 3 months following the eruption (Baxter et al. 1983).

Children were also studied following the Mt. St. Helens ashfalls. Two studies investigated the acute effects of volcanic ash on lung function (Buist et al. 1983; Johnson et al. 1982) using standard spirometry tests (see Methods section). Buist et al. (1983) studied summer-camp children located in an area affected by 1.2 cm of ashfall from the June 12 eruption. Measured exposure levels were quite low and regular testing showed no overall effect on lung function. Buist et al. (1983) and Johnson et al. (1982) concluded that there was no effect on lung function in the studied children. In contrast, hospital admission rates for childhood asthma doubled in Spokane County, Washington, in the 2 months after the May 18 eruption compared with the same period in the following year (Kraemer and McCarthy 1985; Nania and Bruya 1982).

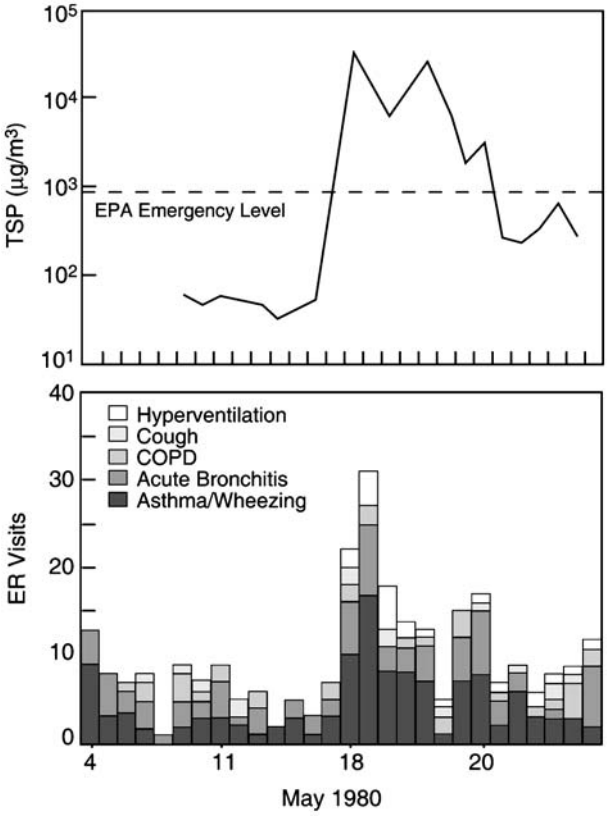

Fig. 4 24-h average concentrations of total suspended particulates and number of emergency room visits due to acute respiratory problems at the two major hospitals in Yakima, May 4 to June 2 1980. The Environmental Protection Agency's Emergency Action Level for potential health problems due to increased air pollution is a 24-h mean concentration of 875 μg m^{-3} of TSP. Adapted from Baxter et al. (1983) and Bernstein et al. (1986)

Table 2 Summary of epidemiological studies

Volcano	Author	Study type	Number of participants	Study information	Result
Mt. St. Helens	Baxter et al. (1981)	Emergency room surveillance	33 hospitals	Surveillance of hospital emergency room attendances for respiratory conditions	Increase in asthma attendances. Few hospital admissions, no deaths
Mt. St. Helens	Nania and Bruya (1982)	Emergency room surveillance	1 hospital and control hospitals	Surveillance of hospital emergency room attendances for respiratory conditions	10% increase in visits and increase in respiratory symptoms in first 3 weeks
Mt. St. Helens	Baxter et al. (1983)	Case-control (questionnaire)	39 asthma patients, 44 bronchitis patients	Hospital patients with lung problems answered questionnaires following eruption	Pre-existing problems were exacerbated following eruption
Mt. St. Helens	Baxter et al. (1983)	Case-control (questionnaire)	97 chronic lung-disease patients	Patients with prior respiratory problems answered questionnaires	1/3 of patients said respiratory symptoms worse following eruption
Mt. St. Helens	Kraemer and McCarthy (1985)	Hospital surveillance	7 hospitals	Paediatric asthma hospitalisations compared with expected data	Two-fold increase in admissions in 2 months following eruption
Mt. St. Helens	Norton and Gunter (1999)	Cross-sectional (database)	24,247 observations	Observations from Idaho Dept. of Vital Statistics Database	No correlation between PM_{10} and respiratory disease
Mt. Sakurajima	Wakisaka et al. (1978)	(Abstract only, in Japanese)	Unknown	Asthma in schoolchildren from areas of different ash exposure	Monthly prevalence higher in areas within 20 km of volcano
Mt. Sakurajima	Wakisaka et al. (1983b)	(Abstract only, in Japanese)	Unknown	Asthma in schoolchildren from areas of different ash exposure	Volcanic activity related to increased bronchial asthma
Mt. Sakurajima	Wakisaka et al. (1983a)	(Abstract only, in Japanese)	Community of Kagoshima Prefecture	Standard mortality ratios compared for areas of different exposure	Higher mortality for bronchitis and emphysema found in areas of high ashfall
Mt. Sakurajima	Wakisaka et al. (1984)	(Abstract only, in Japanese)	Municipalities of Kagoshima, Tarumizu and Sakurajima	Standard mortality ratios compared for cities of different exposure	Deaths exceeded expected rates during times of heavy ashfall
Mt. Sakurajima	Wakisaka et al. (1985)	(Abstract only, in Japanese)	Population within 50 km radius of Mt. Sakurajima	Mortality at increasing distance from volcano	<30 km from volcano, deaths exceeded expected. Declined with distance
Mt. Sakurajima	Wakisaka and Yanagihashi (1986)	(Abstract only, in Japanese)	39 communities around Mt. Sakurajima	Week-to-week variations in mortality compared with volcanic air pollution	Correlation of deaths with SO_2 and volcanic ash exposure
Mt. Sakurajima	Higashi et al. (1987)	Prospective	380 women	Seasonal variation assessed	Increased symptoms in winter. Not known if relates to ash exposure
Mt. Sakurajima	Yano et al. (1986)	Cross-sectional (questionnaire)	2,006 women	3 ash-exposed areas. 30–59 years old	Respiratory disease low in all areas, but higher where higher TSP
Mt. Sakurajima	Yano et al. (1990)	Case-control (questionnaire)	1,991 women	2 cities (one high-exposure, one control). 30–59 years old	Increase in symptoms with increased TSP
Mt. Sakurajima	Uda et al. (1999)	Case-control (questionnaire)	4,310 exposed and 1,656 control children	Comparison of asthma in schoolchildren	No difference between exposed and control groups
Soufrière Hills	Cowie et al. (2001b)	Symptom prevalance study (questionnaire)	465 respondents	Montserratians relocated to UK	Respiratory symptoms higher than general UK population

Table 2 (continued)

Volcano	Author	Study type	Number of participants	Study information	Result
Mt. Spurr	Gordian et al. (1996)	Questionnaire	11,300 State of Alaska and municipal employees and dependents	Association between particulate matter, temperature and outpatient visits	Increased PM_{10} correlated with increased visits one year after eruption
Mt. Spurr	Choudhury et al. (1997)	Insurance claims surveillance	~10,000 state employees and dependents	Association between respiratory illness and PM_{10} pollution	Significant positive association
Cerro Negro	Malilay et al. (1996)	Medical records surveillance	~300,000 residents	Visits to health-care facilities	Rates of visits increased in affected area-mainly children
Ruapehu	Bradshaw et al. (1997)	Case-control (questionnaire)	1,392 asthmatics	Symptoms in known asthmatics following eruption	No association between asthmatics living in control area and ash fall
Ruapehu	Hickling et al. (1999)	Case-control	37 census areas in study group and 49 in control group	Discharge rates from hospitals in ash-affected areas	Borderline increase in bronchitis rates
Mt. Tungurahua	Tobin and Whiteford (2001)	Questionnaire	302 respondents	Recalling health of children for 2 weeks previous to surveys	Increased tuberculosis and respiratory infections, pronounced in children

Standard mortality ratio=(observed deaths in study population/expected deaths in study population)×100

A study on loggers was carried out by the National Institute of Occupational Safety and Health (NIOSH) (e.g. Bernstein et al. 1981, 1982; Merchant et al. 1982). Loggers were recruited randomly from four logging camps in Washington State affected by the ashfall on May 25 and from two unexposed camps in Oregon. FVC and FEV_1 (see Methods section) were measured in June 1980 and chest X-rays taken. Although exposure to crystalline silica was thought to exceed recommended limits, actual exposure was probably less, due to respiratory protection. Acute respiratory symptoms were significantly higher among exposed loggers than the non-exposed workers. The tests were repeated in September 1980 and no significant differences in symptoms or lung function were found.

The NIOSH study was continued as a 4-year prospective study (Buist et al. 1986) to determine the long-term effects of volcanic ash exposure. The loggers were tested annually from 1980 to 1984. Loggers working in ash-fall areas experienced a small, short-term, reversible decline in lung function which was greatest in magnitude and duration for those working in the immediate vicinity of the blast zone. The control group also displayed a similar but lower-magnitude trend, indicating that part of the pattern was due to non-volcanic-related factors. Chest X-ray changes were not observed, but this was expected as fibrotic changes usually take many more years to become manifest.

The epidemiological results are consistent with the hypothesis that the ash acted as an irritant on the airways, leading to an increase in mucus and inflammation. The duration of exposure was considered to have been too short to present a long-term risk from silicosis or COPD. Within 3 months, the ash was beginning to be incorporated into the soil, and had been mostly removed from residential areas.

More recently, Norton and Gunter (1999) sought to determine the relationship between respiratory disease in the farming population of Idaho and exposure to quartz-rich dust. The predominant wind direction in Idaho is from the west, from Washington state. PM_{10} in the study area comprised up to 60 wt% Mt. St. Helens volcanic ash. There was no association between PM_{10} levels and respiratory disease and no excess risk of lung cancer in their study, but the levels of COPD were found to be elevated in farmers and in the general Idaho population when compared to the US population, though the significance of this is not clear.

Toxicological studies

Experimental studies for the evaluation of the health effects of minerals were adapted for the study of Mt. St. Helens ash (Craighead et al. 1983; Martin et al. 1983b). Nineteen studies were carried out on the biological effects of Mt. St. Helens volcanic ash: nine in vitro studies, six intratracheal in vivo studies and four inhalation in vivo studies. A summary of the main results is given in Table 4; the reader is directed to Martin et al. (1986) for a detailed overview.

Of the nine in vitro studies, five found the ash to cause cell toxicity (Fruchter et al. 1980; Green et al. 1982; Robinson and Schneider 1982; Vallyathan et al. 1983a,

Table 3 Summary of clinical studies

Volcano	Author	Study type	Number of participants	Study design	Result
Mt. St. Helens	Bernstein et al. (1981, 1982); Merchant et al. (1982)	Case-control (questionnaire, spirometry and X-ray)	697 loggers	Affect of ash exposure on loggers	Acute symptoms significantly higher among exposed loggers immediately following exposure, with no difference 6 months later
Mt. St. Helens	Johnson et al. (1982)	Spirometry tests	120 children	Acute effects of volcanic ash on lung function in children	Short-term exposure induces less acute effects than air pollution
Mt. St. Helens	Buist et al. (1983)	Spirometry tests	101 children	Acute effects of volcanic ash on lung function in children	No strong evidence of change
Mt. St. Helens	Buist et al. (1986)	Prospective (spirometry)	712 loggers	Long-term effect of ash on loggers	Loggers experienced a short-term reversible decline in lung function
Mt. Sakurajima	Research Committee on Volcanic Emissions (1982)	Case-control (spirometry, X-ray, examination, case history)	2,136 adult residents (2,164 controls)	2,136 adult residents (2,164 controls)	No pneumoconiosis. Slight decrease in pulmonary function
Mt. Sakurajima	Yano et al. (1987) (in Japanese)	Cross-sectional (spirometry, X-ray, examination, questionnaire)	266 loggers	Long-term effect of ash on loggers.	No respiratory impairment observed
Soufrière Hills	Cowie et al. (2001a)	Spirometry, X-ray and symptoms questionnaire	421 subjects	Workers with high exposure	Prevalence of respiratory symptoms low, except for gardeners and road workers. No chest abnormalities

1984) but it was less toxic than quartz and similar to other low-toxicity minerals. Four studies found the ash to be non-toxic (Adler et al. 1984; Bonner et al. 1998; Castranova et al. 1982; Dodson et al. 1982; Martin et al. 1984a) in not stimulating macrophages or causing an inflammatory response.

Vallyathan et al. (1984) published the only comparative in vitro study of ash from three different volcanoes: Mt. St. Helens, Galunggung (Java, Indonesia) and El Chichón (Mexico). Haemolysis (red blood cell death) and alveolar macrophage cytotoxicity assays were used. All samples were toxic to macrophages, but ash samples from Galunggung and El Chichón were more potent than the Mt. St. Helens ash. Haemolysis activity was less for ash than quartz and the level of toxicity appeared to correlate with the surface area of the samples. The crystalline silica content of the samples (in the <10 μm fraction) ranged from 1.50–1.95%.

The in vivo studies also gave varied results. Of the six instillation studies, three found the ash to be mildly toxic (Akematsu et al. 1982; Beck et al. 1982; Kornbrust and Hatch 1984) while two showed fibrosis and granulomas in the lung (Sanders et al. 1982, 1983; Vallyathan et al. 1983b). One study did not find the ash to be toxic (Raub et al. 1985). Of the four inhalation studies, the outcome in two was the development of fibrosis (Martin et al. 1983a; Wehner et al. 1986), but there was no evidence of toxicity in two studies (Raub et al. 1985; Wiester et al. 1985). Sanders et al. (1982) and Vallyathan et al. (1983a) studied animals for 400 days and 6 months, respectively, thereby allowing the analysis of the long-term effect of volcanic ash in the lung. In both studies the ash was less toxic than quartz particles.

The inhalation studies which found fibrosis and lesions (Martin et al. 1983a; Wehner et al. 1986) did so after administering very high doses of ash (50–100 mg m^{-3}) over long time periods. The two studies which did not observe toxicity both used lower doses (≤10 mg m^{-3}) and had a shorter follow-up.

Five other toxicological studies (Fedan et al. 1981; Grose et al. 1985; Martin et al. 1984b; Schiff et al. 1981; Wehner et al. 1984) carried out on the Mt. St. Helens ash examined the effects of ash on antibacterial defences, muscle response, clearance rates and cilia viability.

Problems with the above studies include the lack of standardised samples and controls used for analyses and the rush to generate information leading to poorly planned studies. Despite the seemingly conflicting results, we can

Table 4 Summary of toxicological studies

Volcano	Author	Study type	Study design	Result	Comments
Mt. St. Helens	Fruchter et al. (1980)	In vitro	Rabbit alveolar macrophages. Ash compared with soil and quartz	Less toxic than quartz	
Mt. St. Helens	Castranova et al. (1982)	In vitro	Rat alveolar macrophages and cells	No change in cells	No controls
Mt. St. Helens	Dodson et al. (1982)	In vitro	Human alveolar macrophages	Ash not toxic to macrophage	No controls
Mt. St. Helens	Green et al. (1982)	In vitro	Rabbit alveolar macrophages and haemolysis	Little toxicity of macrophages. Moderate haemolysis	No controls
Mt. St. Helens	Robinson and Schneider (1982)	In vitro	Rabbit alveolar macrophages. Ash compared with quartz and TiO_2 standards	Less toxic than quartz. Similar to TiO_2	
Mt. St. Helens	Vallyathan et al. (1983a)	In vitro	Haemolysis of sheep red blood cells. Compared with quartz, asbestos and gypsum	Ash less toxic than quartz but same as asbestos and gypsum	Well controlled
Mt. St. Helens	Adler et al. (1984)	In vitro	Rat epithelial cells. Compared with feldspar, cristobalite and quartz	No acute or long-term effects of ash	Well controlled
Mt. St. Helens	Martin et al. (1984a)	In vitro	Human alveolar macrophages. Ash compared with quartz	Ash not a stimulus of inflammation in lung	No inert control
Mt. St. Helens, Galunggung, El Chichon	Vallyathan et al. (1984)	In vitro	Haemolysis and alveolar macrophages. Ash compared with quartz	Ash toxic to macrophages. Less haemolysis than quartz	
Mt. St. Helens	Bonner et al. (1998)	In vitro	Comparison of Mexico City PM10 with MSH ash on rat alveolar macrophages	Ash did not stimulate macrophages	Ash used as inert control
Mt. St. Helens	Akematsu et al. (1982)	In vivo	Rat instillation for 7 days	Ash caused cell injury	No controls
Mt. St. Helens	Beck et al. (1982)	In vivo	<5 μm ash instilled in hamsters. Ash compared with Al_2O_3	Ash similar toxicity to Al_2O_3 and less than quartz	Well controlled
Mt. St. Helens	Sanders et al. (1982, 1983)	In vivo	Rats studied for 400 days following instillation. Compared with quartz, soil and saline	Ash produced pulmonary fibrosis and lymph node hyperplasia, but less than quartz	Well controlled
Mt. St. Helens	Vallyathan et al. (1983b)	In vivo	Rat instillation for 6 months	Ash caused granulomas	No controls
Mt. St. Helens	Kornbrust and Hatch (1984)	In vivo	Instillation of ash for 6 months to examine lung lipids. Compared with quartz	2× increase in phospholipids (compared with 11× for quartz)	No inert control
Mt. St. Helens	Raub et al. (1985)	In vivo	Coarse (12μm) ash and quartz instilled in rats	Ash showed no change in pulmonary function	
Mt. St. Helens	Martin et al. (1983a)	In vivo	Inhalation of rats (100 mg m^{-3}, <6.7 μm) for 2 weeks. Observed for 9 months. Compared with quartz	Mild acute lung injury (quartz: immediate silicoproteinosis). After 6 months-fibrosis by ash and quartz	No inert control
Mt. St. Helens	Raub et al. (1985)	In vivo	Inhalation of rats (9.4 mg m^{-3}) for 2 h daily, 5 days	No immediate or long-term effects	
Mt. St. Helens	Wiester et al. (1985)	In vivo	Inhalation of guinea pigs (10 mg m^{-3}) for 2 h	Ash exposure reduces tracheal reactivity	No controls

Table 4 (continued)

Volcano	Author	Study type	Study design	Result	Comments
Mt. St. Helens	Wehner et al. (1986)	In vivo	Inhalation of rats (5 or 50 mg m^{-3}), 6 h/day, 5 day/week, up to 24 months. Compared with quartz.	High dose showed lesions: less than quartz. Low dose: minimal effects.	No inert control
Mt. Sakurajima	Yano et al. (1985)	In vitro	Ash tested for inflammation response of cells. Compared with TiO_2 and asbestos	Little toxicity and less toxic than inert dust	Well controlled
Mt. Sakurajima	Shirakawa et al. (1984)	In vivo	Injection and inhalation of rats (29 mg m^{-3})	Ash induced bronchitis, emphysema, degeneration of blood vessels, fibrosis	No controls
Mt. Sakurajima	Kariya et al. (1992)	In vivo	Histopathological changes in 25 dogs	No obvious effects in canine lung	
Soufrière Hills	Cullen and Searl (1998)	In vitro	Cytotoxicity on human epithelial cells and rat alveolar macrophages. Compared with toxic and inert dusts.	Less toxic than quartz, but greater toxicity than inert control	Well controlled
Soufrière Hills	Wilson et al. (2000)	In vitro	Haemolysis and human epithelial cell toxicity. Compared with toxic and inert dusts	Less toxic than quartz, but greater toxicity than inert control. Haemolysis dose-dependent	Well controlled
Soufrière Hills	Cullen et al. (2002)	In vivo	Inhalation of rats for 5 days/week for 8 weeks. Compared with inert dust	Greater inflammation in ash-exposed rats than inert control	No fibrogenic control
Soufrière Hills	Cullen et al. (2002)	In vivo	Instillation of ash into rat lung for 24 h. Compared with quartz and inert controls	Inflammation similar to inert controls	Well controlled
Soufrière Hills	Cullen et al. (2002)	In vitro	Haemolysis and human epithelial cell toxicity. Compared with toxic and inert dusts	No significant difference between ash and inert control	Well controlled
Soufrière Hills	Housley et al. (2002)	In vivo	Instillation of rats for up to 9 weeks. Compared with cristobalite, quartz and inert controls	Little inflammation or enlargement of lymph nodes compared with toxic dusts	Well controlled
Soufrière Hills	Lee and Richards (2004)	In vivo	Instillation of rats for up to 49 weeks. Compared with anorthite and cristobalite	Enlargement of lymph nodes and increased cell numbers observed at highest dose towards end of experiment	Well controlled

For information on dose and exposure time of Mt. St. Helens studies, see Martin et al. (1986), Table 1

tentatively conclude that the Mt. St. Helens ash was much less toxic than pure crystalline silica in normal lungs, but could form pulmonary fibrosis in large doses over long durations of exposure (Martin et al. 1983b). Therefore, people (including outdoor occupational groups) who were exposed to this ash were probably at low risk of fibrotic disease as exposures had been short and generally not very high.

Bonner et al. (1998) compared the respiratory toxicity of Mt. St. Helens dust with urban ambient PM_{10} from Mexico City in in vitro experiments with rat alveolar macrophages. They found that all Mexico City samples provoked the macrophages to produce a response while volcanic ash produced no response.

Mt. Sakurajima, Japan

Mt. Sakurajima, in the south of Kyushu Island, Japan, has been in eruption since 1955. It has frequent, often daily, summit explosions, exposing the population of Kagoshima Island and the nearby city of Kagoshima (10 km west) to volcanic ash. The volcano erupts andesitic/dacitic dome-forming lava but dome-growth phases are short lived because of the frequent explosions. There is, therefore, limited time for cristobalite production through vapour-phase crystallisation and devitrification, as was found, for example, at the Soufrière Hills volcano, Montserrat.

It has been estimated that ~1,100 tons km^{-2} of ash are deposited every month around Mt. Sakurajima (Yano et al. 1985). The frequency of eruptions and quantity of particulate matter in the atmosphere have prompted concern about the potential health hazard of the ash. Several epidemiological and toxicological studies have been carried out and are summarised below.

Characterisation of the ash

Despite many epidemiology and toxicology studies having been published on the Mt. Sakurajima ash, none of them contain sufficient data on the characterisation of the ash itself. Many of them (e.g. Yano et al. 1987) state that the ash particles were too large to be respirable and there was negligible crystalline silica present. Yano et al. (1985) studied the physico-chemical characteristics of Mt. Sakurajima ash collected 8 km from the crater using a high-volume air sampler with cyclone over 24 h. The elemental composition of the sample was measured with SEM-EDX and transmission electron microscopy (TEM) indicating that around 30 wt% of the ash was silica, although it is not clear if this refers to crystalline silica. Table 1 shows that the Mt. Sakurajima sample analysed by Horwell (submitted) contains <1 wt% respirable material, indicating that the ash is comparatively coarse and, therefore, is unlikely to be a major chronic respiratory health hazard even over long exposure durations.

Epidemiological and clinical studies

At least ten epidemiological studies and two clinical studies have been carried out on the population surrounding Mt. Sakurajima. These are summarised in Tables 2 and 3. Wakisaka et al. (Wakisaka et al. 1978, 1983a,b, 1984, 1985; Wakisaka and Yanagihashi 1986) carried out at least six epidemiological studies (published in Japanese) which address asthma in schoolchildren and variations in mortality and disease at different distances from the volcano. Their studies found that there was higher asthma prevalence in areas receiving higher ashfall. However, Uda et al. (1999) also examined asthma in schoolchildren, but did not find any difference between the exposed and control groups.

In 1982, the Research Committee on Volcanic Emissions (1982) reported on a medical study involving residents in the town of Sakurajima, situated 5 km from the crater (2,136 adult residents studied, 50% of total population) and a control area (Oura, 50 km southwest of the crater, 2,164 adult residents studied, 75% of total population). No cases of pneumoconiosis were observed despite some participants having been exposed to the ash for decades. A slight decrease in lung function was detected in women in the Sakurajima area compared to the control area. It was concluded that the TSP concentrations were not high enough to cause major changes in lung function.

Yano et al. (1986) carried out a cross-sectional epidemiological study on 2006 women, aged 30–59, from three areas subjected to different concentrations of airborne ash. The results showed that non-specific respiratory diseases and related symptoms were very low in all areas, but when the three areas were compared, there was an increase in symptoms with increasing TSP levels. This study was repeated by Yano et al. (1990) in another high-exposure area and a corresponding control area, with a similar number of participants (1991 women aged 30–59). The prevalence of non-specific respiratory disease was slightly higher in the high-exposure area, consistent with the results of the previous study.

Higashi et al. (1987) concentrated on the seasonal variation in TSP levels. Results showed that, for most of the questions relating to respiratory symptoms, positive responses were higher in winter compared to summer. It is not clear if these results were related to exposure to volcanic ash.

Yano et al. (1987) studied the lung function of loggers working in three forested areas exposed to Mt. Sakurajima ash. No cases of respiratory impairment were observed and there were no differences among loggers in the different forest areas, despite the TSP varying with distance from the volcano. Yano et al. (1990) believe that their negative findings were because the ash was of non-respirable size and was relatively inert.

Toxicological studies

Yano et al. (1985) examined the in vitro cytotoxicity of the ash and its ability to elicit biological responses related to inflammation. The ash produced little cytotoxicity in cultures and was considerably less toxic than TiO_2, the inert control dust.

Shirakawa et al. (1984) observed that the inhalation of ash led to histopathological changes in the respiratory organs of study animals. The ash induced bronchitis, pulmonary emphysema, degeneration of blood vessels, dust nodes and fibrosis. They used high exposure levels (29 mg m^{-3}), but no positive or negative control dusts.

Kariya et al. (1992) examined the amount of intrapulmonary particulate deposits (IPD) and histopathological changes in 25 abandoned or stray dogs in areas affected by volcanic ash in comparison with 13 abandoned or stray dogs in a control area. Age-associated increase of IPD was noted, but mean IPD values were not different between the two groups.

Soufrière Hills, Montserrat, West Indies

The Soufrière Hills volcano began erupting on 18 July 1995 after a repose of about 400 years. Elevated levels of cristobalite in the ash were believed to have been produced by devitrification and vapour-phase crystallisation processes within the andesite dome (Baxter et al. 1998). The dome-forming eruption caused intermittent ashfalls in populated areas of the island during the cycles of dome building and collapse. This has presented a more significant exposure problem than Mt. St. Helens because of the frequent ashfalls until 2003, during which the ash would be present and remobilised in the environment for days or weeks, until heavy rain fell (Fig. 5). In 2004, a probabilistic health risk assessment for the development of silicosis was completed which estimated the individual risk for people living in different parts of the island should the eruption continue overall for up to 20 years, as volcanologists had forecasted that the eruption had at least a 50% probability of continuing for more than 10 years (Cowie et al. 2003).

Characterisation of the ash

The Soufrière Hills ash is formed in several ways. Firstly, pyroclastic flows form as parts of the unstable dome collapse, generating lofting, co-ignimbrite ash plumes from the fragmented rock. The resulting fallout is known as a 'dome-collapse ash-fall' deposit. Secondly, during explosion episodes, ash is formed from the explosion itself and is also generated in pyroclastic flows which result from column collapse. The plumes which loft from the pyroclastic flows are entrained in the main column plume. Therefore the two explosion ash sources are treated as one here. Thirdly, ash-sized particles are also found in the pyroclastic flow deposit matrices from both explosive and dome-collapse eruptions.

Baxter et al. (1999) characterised the sub-10-μm fraction of the Soufrière Hills ash. They found that a dome-collapse ash-fall deposit typically contained 13–20% <10 μm particles. Explosive ash-fall deposits had 13–14% <10 μm material. As with the Mt. St. Helens studies, Baxter et al. (1999) quantified crystalline silica using several methods, including the removal of plagioclase and other minerals with the Talvitie method. They found that the sub-10 μm dome-collapse ash-fall deposit contained 10–27 wt% crystalline silica, significantly higher than for explosive ash-fall samples (4–6 wt%). The crystalline silica was composed of cristobalite, tridymite and minor quartz.

Horwell et al. (2003b) studied the respirable (<4 μm) fraction of the Soufrière Hills ash. Dome-collapse ash-fall deposits are significantly richer in respirable particles (12 wt%) than other tephra types, in particular the dome-collapse pyroclastic-flow matrices (3 wt%). Within the

Fig. 5 Driving a jeep in the Montserrat Exclusion Zone after the ashfall on 29 July 2001. The dry, fine ash is readily re-suspended by traffic. In populated areas, where the ashfalls were less frequent, conditions were much better due to regular removal of ash by road crews

respirable fraction, dome-collapse ash-fall contains the highest proportion of crystalline silica particles (20–27 number %, of which 97% is cristobalite), compared with other tephra types (0.4–5.6 number %). This enrichment of crystalline silica in the dome-collapse ash-fall is most pronounced in the very-fine particle fraction (sub-2 μm). This is due to significant size fractionation during fragmentation of pyroclastic flows, resulting in a fines-depleted pyroclastic flow matrix and a fines-rich dome-collapse ash-fall deposit (Horwell et al. 2001). For all samples (ash-fall deposits and pyroclastic flow matrices), the sub-4 μm fraction comprised 45–55 wt% of the sub-10 μm fraction. Horwell et al. (2003b) also studied the concentration of ash particles re-suspended by road vehicles on Montserrat (Fig. 5). The concentration decreased exponentially with height above the ground, indicating higher exposure for children compared with adults. For example, PM_4 concentrations at 0.9 m (height of a 2-year-old child) were three times that at 1.8 m (adult height). Baxter et al. (1999) and Horwell et al. (2003b) also quantified the other minerals in the ash, showing that plagioclase and glass were dominant, with minor amphibole, pyroxene and iron oxides.

Within the lung, it is the surface of the particle which will react with endogenous molecules leading to a toxic effect (see Mechanisms of particle toxicity section). Horwell et al. (2003a) have shown that the Soufrière Hills volcanic ash is capable of generating free radicals which may react with DNA and other cellular components, initiating a toxic reaction. Both dome-collapse and explosive ash generated three times as many radicals as a quartz control sample (Min-u-Sil standard) of known toxicity. The cause of the radical formation was not crystalline silica but, instead, Fe^{2+} from the surface of iron-containing minerals such as amphibole. Newly erupted samples were capable of generating more radicals than weathered samples where the Fe^{2+} is either leached or oxidised.

Epidemiological and clinical studies

Several epidemiological and clinical studies have been carried out on the Montserrat population. The studies have focused on (1) the children of the island, (2) the population who stayed on Montserrat and (3) the population who moved to the UK during the eruption, and are summarised in Tables 2 and 3.

Forbes et al. (2003) evaluated the effects of ashfalls on the respiratory health of children in Montserrat using a questionnaire and a lung function survey of 443 children on the school rolls in February 1998. The study took into account the different places of residence of children whose families had moved around the island in response to the gradual enlargement of the ‘exclusion zone’. In children aged 12 years and under, the prevalence of wheezing (an asthma symptom) was 18% overall and was greater in those who had been heavily or moderately exposed compared to the group who had been exposed to low levels. The prevalence of exercise-induced bronchospasm in 8–12-year olds was about four times higher in those who were currently heavily exposed to volcanic ash compared to those exposed to low levels. The results indicated a need to limit the exposure of children to volcanic ash and to ensure that they receive optimum treatment for their asthma.

Cowie et al. (2001a) targeted workers from all occupational groups with potentially high volcanic ash exposures. A survey was conducted in October 2000 on 421 subjects. The prevalence of respiratory symptoms was relatively low with no evidence of chest abnormalities on X-rays, despite many workers having been exposed to ash on a regular basis. Respiratory symptoms and decreased lung function were observed for gardeners and road-workers (two of the dustiest occupations, see Fig. 1) but there was no evidence that other occupations such as domestic cleaning, affected the health of the subjects.

Cowie et al. (2001b) carried out a survey on Montserrat residents who had relocated to the UK. There were 465 respondents to a postal questionnaire, a response rate of only 25%. The prevalence of respiratory symptoms was higher than in the general UK population. This may, in part, be due to the effects of moving to a new country with a change in climate and different disease immunities. A relationship between exposure to ash from heavy ash-clearing activities and the occurrence of respiratory symptoms was noted but there was no relationship with domestic occupations. The results of these two studies were reassuring, but the low response rate in the latter reduced the weight that could be placed on its findings.

Toxicological studies

Toxicological studies carried out on the Soufrière Hills ash are summarised in Table 4. All of the studies found the ash to have a similar or greater toxicity than inert controls but was significantly less toxic than quartz.

Cullen and Searl (1998) carried out in vitro tests on human epithelial cells and rat alveolar macrophages. The ash was compared to both toxic and inert dusts, and the effects of pre-washing the ash were investigated. The ash caused a level of epithelial cell injury similar to that of quartz (DQ12 standard) and clear dose-related effects. The ash was more reactive than the inert control (TiO_2) and less reactive than quartz in the macrophage. There was no significant change in the cytotoxicity following washing with water and no significant differences in toxicity between the three samples analysed, all of which had varying quantities of cristobalite, indicating that crystalline silica was not the only cause of reactivity. Cullen and Searl (1998) suggest that the toxicity observed could be due to Fe^{2+} in the ash.

Wilson et al. (2000) used the same samples of volcanic ash as Cullen and Searl (1998) and also examined the competence of human epithelial cells and the ability of volcanic ash to induce sheep blood haemolysis (destruction of red blood cells). Results showed a similar decrease in metabolic competence of epithelial cells to Cullen and Searl’s (1998) experiments. The ash samples induced a

dose-dependent increase in haemolysis, which was less than that of quartz but more than TiO_2. Neither the cristobalite content nor the surface area of the ash samples correlated with the observed reactivity of the samples.

Cullen et al. (2002) carried out an inhalation study, an instillation study and further in vitro studies. The inhalation experiment was designed to test the null hypothesis that, dose for dose, inhaled volcanic ash causes no greater lung inflammation than a low-toxicity control dust (see Table 4 for details). Animals were sacrificed at 14, 28, 42 and 56 days from the start of exposure. Lung fluid was examined for the presence of inflammation markers (e.g. increased cell numbers). Lung inflammation and cell damage were greater for ash-exposed rats than the control rats. It was concluded that the volcanic ash was more toxic than nuisance dust particles of similar size and that prolonged exposure could lead to fibrosis.

The instillation experiment was conducted to test the inflammatory response of volcanic ash with those of toxic and low-toxicity control dusts. After 24 h, the ash samples induced similar levels of inflammation to the TiO_2 control and a saline control. Quartz produced a significantly greater inflammation than the saline control.

The in vitro study documented in Cullen et al. (2002) is similar to that reported by Cullen and Searl (1998) and Wilson et al. (2000). Two tests were used: (1) the haemolysis of sheep red blood cells as an indicator of surface reactivity of the ash and its potential cytotoxicity; (2) use of epithelial cells as indicators of the ability of dusts to damage cell membranes. The ash was compared with TiO_2 inert control and DQ12 quartz, as with previous experiments. Neither the haemolysis assays nor epithelial cell tests showed a significant difference between TiO_2 and volcanic ash. From the results of the in vitro and in vivo studies and comparison with other inhalation studies, Cullen et al. (2002) concluded that the Montserrat ash has a toxicity similar to that seen with mixed coal-mine dusts and that the toxicity observed could be related to Fe^{2+}.

Housley et al. (2002) conducted an in vivo instillation study where two samples of volcanic ashfall (one explosive, one dome-collapse) were instilled into rats, which were sacrificed at 1, 3 and 9 weeks. Bio-reactivity was assessed by increases in lung permeability and inflammation, changes in epithelial cells and increases in lymph-node size. The ash samples had little effect compared to cristobalite or DQ12 quartz, which induced inflammation and enlargement of lymph nodes.

A more detailed instillation study was carried out by Lee and Richards (2004). Rats were instilled with different doses of respirable dome-collapse ash-fall with control groups being exposed to anorthite and cristobalite (inert and toxic controls, respectively). The rats were sacrificed at different time intervals up to 49 weeks. The anorthite and low-dose ash treatments showed no toxic effects. Medium and high doses of ash caused enlargement of lymph nodes and increased cell numbers, most noticeably at 49 weeks, but no evidence of fibrosis. In contrast, the cristobalite treatment caused progressive inflammation and cell proliferation, and by 49 weeks, fibrosis of the lung and lymph nodes. The late occurrence of inflammation in rats exposed to volcanic ash suggests a delayed toxic effect which may be due to leaching of a protective aluminium or glass coating from the surface of cristobalite particles in the lung. A coating or intergrowth of glass or, theoretically, aluminium could be generated as cristobalite forms through devitrification in the dome, but it is not yet known if this is the case. These results indicate that short-term assays may not be a good measure of longer-term pathological effects.

A synthesis of the scientific evidence and a probabilistic approach to the silicosis risk

Following the release of the findings of the above toxicological studies, UK experts synthesised the results, concluding that the ash was moderately toxic to the lung, but less than was predicted from its cristobalite content (Cowie et al. 2003). Recent work on silicosis has shown that the health risk of siliceous dusts does not correlate as closely as is often supposed with their crystalline silica content (Health and Safety Executive 2002, 2003). Several factors can modulate the silicosis hazard of dusts containing crystalline silica. The UK Health and Safety Executive reviews (Health and Safety Executive 2002, 2003) highlight: (1) the polymorphic type (i.e. quartz or cristobalite); (2) the presence of other minerals, especially aluminium-containing clay, which is protective; (3) particle size and 4) variability between freshly fractured and 'aged' surfaces. All of these are likely to be relevant for volcanic ash. For its toxicity ranking, the expert group considered that the Montserrat ash should be regarded as a mixed-dust comparable to coalmine dust in which quartz particles are mixed with other minerals (kaolin, illite) and carbon particles. In such dusts, the reactivity of quartz and components such as iron can induce separate effects (Donaldson et al. 2001). The most relevant comparison for Montserrat was considered to be an epidemiological study of Californian diatomaceous earth workers (Hughes et al. 1998). The exposure-disease function from this study was applied to estimates of exposure on Montserrat to obtain the probability of developing silicosis from the volcanic ash (Cowie et al. 2003). The findings indicate that a significant risk (approximately >1%) of developing early radiological signs of silicosis exists for the population (especially children and outdoor workers such as gardeners) in the areas most frequently affected by ashfalls if the volcano continued its pattern of behaviour for more than 10 years. To protect the population and reduce this risk requires active measures to minimise ash exposure and regular health monitoring (Cowie et al. 2003).

Risk assessment exercises are required in future eruptions occurring in populated areas, as the health risks of ash from different volcanoes will be different. Indeed, it is only a working assumption (but a reasonable one) that the ash from the Soufrière Hills volcano will have the same toxicity ranking throughout its eruption.

Health studies at other volcanoes

Besides Soufrière Hills, Mt. St. Helens and Sakurajima, very few volcanoes have specifically been studied for the determination of the health hazard of their ash. The few studies performed make it difficult to draw firm conclusions about the toxic nature and characteristics of the ash studied.

Mt Pinatubo, Philippines

The eruption of Mt Pinatubo, on 15 June 1991, produced the greatest ashfall of the late twentieth century. Though most of the ash fell into the South China Sea, substantial quantities fell on communities in the vicinity of the volcano, affecting one million people. Major cities experienced <15 cm of ash deposit, but the weight of this deposit (combined with rainwater) led to roof collapse of many buildings with 320 deaths and numerous injuries. An epidemiological surveillance system was set up by the Department of Health, Manila, in clinics, hospitals and refugee centres (50,000 out of 60,000 people living within 30 km of the volcano had been evacuated to these centres just before the eruption), monitoring for respiratory and infectious diseases. No increases in acute respiratory diseases, including asthma, were found, despite a continuous ash plume which deposited fine ash for weeks. The ash, in composition and particle size, corresponds closely to the Mt. St. Helens ash. Moderately bad air pollution was caused as the ash dried and was resuspended by traffic. The absence of evidence for acute health effects was striking, but may have been related to the heavy rain which fell concurrently, compacting the ash deposits, aiding its removal from roads, and leading to less resuspension. Deposits have been substantially added to by lahar activity over the years, yet no studies on long-term health effects have been performed in the ash-affected communities.

Mt. Spurr, Alaska

Anchorage is a city of 240,000 people with few sources of industrial air pollution. The main sources of particulate pollution are vehicle related or from ashfall from volcanic eruptions. In August 1992, Mt. Spurr, 60 miles west of Anchorage, erupted, giving a 24-h average PM_{10} concentration of 565 μg m^{-3} in Anchorage on the day after the eruption, where the deposit was 3 mm in depth. Hourly PM_{10} levels reached a maximum of 3,000 μg m^{-3} in Anchorage on the day of the eruption. Gordian et al. (1996) were already carrying out an epidemiological study on the Anchorage population when the eruption occurred. The average PM_{10} concentration on the days following the eruption was 70 μg m^{-3}, compared with 40 μg m^{-3} during the unaffected period. Gordian et al. (1996) examined the association between average daily PM_{10} and temperature with daily outpatient visits for respiratory disease including asthma, bronchitis and upper respiratory tract illness. The study was conducted between May 1992 and March 1994. Characterisation of the ash showed that 20 wt% of the PM_{10} was <2.5 μm diameter. Concentrations of crystalline silica or other minerals in the ash were not estimated. Their results show that an increase of 10 μg m^{-3} of PM_{10} resulted in a 3–6% increase in visits for asthma and 1–3% increase for upper respiratory tract diseases. However, this trend was not observed in the period immediately after the eruption and only became apparent in 1993. This is probably because the Anchorage residents were advised to minimise exposures by staying indoors and wearing dust masks during and after the eruption. Gordian et al. (1996) also proposed that volcanic ash is not toxic until after mixing with combustion-related fine particles.

Choudhury et al. (1997) surveyed medical insurance claims of State and Municipal employees to investigate the association between upper respiratory tract infections and PM_{10} pollution (including volcanic ashfall), but none was found.

Popocatepetl, Mexico

Popocatepetl is situated 70 km southeast of downtown Mexico City, and 45 km southwest of the city of Puebla. After five decades of dormancy, Popocatepetl began a new phase of eruption on 21 December 1994, prompting the temporary evacuation of 76,000 people. This eruptive episode lasted over 4 months and deposited ash over an area of 4,000 km^2. Peak TSP levels occurred in the first 20 days (maximum 1,440 μg m^{-3}) and then decreased irregularly over the following 4 months. Rojas-Ramos et al. (2001) studied 35 non-smoker farmers (male and female) who live in small towns, 25 km to the east of the volcano. They collected data on lung function and symptoms immediately after the eruption and again after 7 months which showed increased incidence of respiratory symptoms and decrease in lung function of the farmers studied. The symptoms decreased to normal levels at the seventh month of testing, indicating a transient response. The ash reportedly contained <3.5% free silica as cristobalite. Some 32% of the ash sample was <38 μm (the smallest sieve size used). The volcano resumed its eruptive activity in 1996, with populations being intermittently exposed to low levels of ash.

Cerro Negro, Nicaragua

The eruption of Cerro Negro near the city of Leon, Nicaragua, on 9 April 1992 distributed an estimated 1.7 million tons of ash over a 200 km^2 area. Cerro Negro produces basaltic ash, with a high iron content and virtually no crystalline silica. Malilay et al. (1996) assessed the health effects of the ash on approximately 300,000 residents, using data obtained by the national epidemiologic surveillance system. Visits to health-care facilities for acute-respiratory illnesses increased in two study communities, one within (six times more frequent) and one near

(3.6 times more frequent) the disaster zone. Most of the visits were for infants and children less than 5 years old. In addition, many health clinics and the hospital in Leon, located in an area which had received 2–3 cm depth of ash, anecdotally reported marked increases in patients attending with asthma. Basaltic eruptions are associated with coarse ash formation, but the Cerro Negro ash contained a substantial proportion of PM_{10} (2 wt% of fall deposit in Leon) which was re-suspended by strong winds for weeks after the event. This eruption was important in being followed by an outbreak of asthma lasting weeks in the population following a single ashfall. Unfortunately, no follow up studies could be done and so the precise explanation for this dramatic impact is not known.

Mt. Ruapehu, New Zealand

Mt. Ruapehu is the southern-most volcano in the Taupo Volcanic Zone, central North Island, New Zealand. In June 1996, Mt. Ruapehu erupted for 2 days and deposited over 7 million tons of ash over central North Island, but with light ashfalls on the cities of Taupo and Rotorua. The acute health effects of this eruption were studied by Hickling et al. (1999). The study population comprised people living in areas which received >0.25 mm ashfall. The control population was unexposed to ash. Discharge rates from public hospitals were measured for 3 months following the eruption and compared with the corresponding periods for the previous 7 years. The only detrimental respiratory effect detected was a borderline increase in bronchitis.

Bradshaw et al. (1997) investigated the prevalence of respiratory symptoms in known asthma patients, following exposure to volcanic ash caused by another short eruption of Mt. Ruapehu in September 1995. A postal questionnaire was answered by 723 previously identified asthma sufferers 2 months after the first major eruption. The prevalence of nocturnal shortness of breath in the last 2 months was 29.3% in the exposed group and 24.7% in the non-exposed group, and 30.9% of the exposed group had an asthma attack in the previous 2 months compared to 31.9% of the non-exposed group. 48.4% of the exposed group used asthma medication in the 2 months following the eruption in comparison to 53% of the non-exposed group. The study therefore showed no association between living in an area exposed to volcanic ash particles and either asthma symptoms or the use of asthma medication.

Vanuatu Archipelago

Vanuatu, an archipelago of more than 80 islands, forms a portion of an island-arc system extending from New Zealand to New Britain. Continuous low-level basaltic volcanism from Yasur volcano on Tanna Island and Marum and Benbow vents on Ambrym Island, has occurred for as long as records have been kept on Vanuatu. Cronin and Sharp (2002) determined the impacts of volcanic emissions on the environment, particularly focussing on fluoride which is easily leached from the surface of volcanic ash and aerosols. Health clinics reported no unusual increases in chronic or acute respiratory problems in the population exposed to volcanic ash compared with other populations on the islands who were not exposed. Reasons cited for the lack of excess respiratory illness are the coarse grain size and low silica content of the ash.

Mt. Tungurahua, Guagua Pichincha and El Reventador, Ecuador

Mt. Tungurahua started a new eruption in September 1999. A significant ashfall occurred in October 1999, prompting the evacuation of 26,000 people from the rural area downwind of the volcano. The Ecuador Ministry of Health compared respiratory-disease incidence following the eruption with data from previous years. The results indicated that the incidence of upper and lower respiratory infections more than doubled between the two periods of study. A Ministry study of evacuated children found similar, but more pronounced, trends which increased over time (OPS 2000; Tobin and Whiteford 2001). The ash closely resembled the Soufrière Hills ash in particle size and crystalline silica concentration (Baxter 2003).

The activity of Guagua Pichincha volcano began to escalate in 1998 and two moderate-sized ashfalls occurred in Quito on 5 October and 25 November 1999. An epidemiological study (OPS 2000) showed increases in acute health conditions in Quito during this period. The ash contained substantial respirable material and an elevated cristobalite content (Baxter 2003).

The eruption of El Reventador volcano on 3 November 2002 resulted in a moderate ashfall in Quito (depth 3–4 mm) and surrounding communities (depth 3–5 cm). The eruption lasted from 3–21 November and concerns arose that the ash and gas emissions from the volcano, located 80 km north-east of Quito, were adversely affecting air quality and the health of the population in Quito. However, hospitals reported that the health impact had been small, with only slight increases in the numbers of patients with upper and lower respiratory tract problems. Some deaths and injuries were reported to have occurred due to falls from roofs during the ash clean-up (Baxter 2003).

Discussion and conclusions

The small number of clinical, epidemiological and toxicological studies on the health hazard of volcanic ash reported here span less than half a century, and they present a limited view on the acute and chronic health consequences of short and long-term exposure. The reasons for this are related to the limited scope of many of the studies, the infrequency of volcanic eruptions near major population centres and their short duration, and the lack of preparedness for undertaking studies under disaster conditions. Toxicological studies can be criticised as being no substitute for long-term epidemiological follow-up of

populations exposed to volcanic ash, as their results may be of limited applicability to humans and real-life exposures. Despite this, it is possible to draw some general conclusions from the studies about the likely effects of inhaling volcanic ash following an eruption.

Acute health effects

The variable reporting of asthma-like symptoms after eruptions suggests that the main effects could be short-lived and depend upon the mode of generation of the ash and the fineness of the particles, as well as other factors. However, Burney (1999) has highlighted the difficulty of establishing consistent evidence for a link between air pollution and asthma in studies of urban air pollution, however self-evident such an association would seem. Anderson (1999), in a review of major air pollution episodes around the world, also noted the variability in reported effects on asthma, and could offer no explanation. Therefore, we should not be surprised at the wide differences in symptoms reported after volcanic eruptions. Traditional views on asthma are against the condition being caused for the first time by exposure to ash or coarse dust particles, but people with pre-existing asthma and other chronic lung conditions are at greater risk from very dusty conditions, especially when the ash particles are mainly respirable in size (e.g. Baxter et al. 1983, Forbes et al. 2003).

According to our own experience on Montserrat, the extent that exposure to ash led to worsening of symptoms in asthma sufferers was not adequately studied. A frequently given reason for leaving the island was that a family member, particularly a child, was suffering with asthma. The study of asthma in schoolchildren was undertaken after many families had already left the island (Forbes et al. 2003). Importantly, the study found that the disruption of life by the volcanic crisis had led to children receiving inadequate treatment, in that few were using inhalers. In future, prevalence studies assessing the effects of ash on asthma symptoms and the availability of medication should be undertaken as early as possible in an eruption crisis as part of the risk assessment.

Chronic health effects

Without information on the quantity and content of the respirable fraction, many of the studies which aim to observe the possible long-term effects of volcanic ash on respiratory health are of limited value. If a dust is already known to be too large to inhale, or does not contain potentially toxic minerals, it could be argued that the motivation for carrying out expensive toxicological or epidemiological studies would be removed. However, our knowledge of what factors constitute the respiratory toxicity of particles continues to rapidly expand at the time of writing. Only the eruptions of Mt. St. Helens and the Soufrière Hills volcano have had robust and full characterisation studies carried out on the ash. This is a basic requirement before evaluating the acute and chronic health hazard in the population.

Follow-up studies, possibly 20 years or more after the onset of exposure to volcanic ash, are required for human studies of chronic pulmonary effects. The 4-year follow-up study on loggers following the eruption of Mt. St. Helens indicated that there was no need for further studies, as lung function tests showed normal results and exposure to the Mt. St. Helens ash was not prolonged or high enough to have caused respiratory disease. It is possible, however, that a few highly exposed workers could have developed lung abnormalities since those initial studies were undertaken. On Montserrat, it is likely that a few islanders will have been exposed to sufficient ash to cause a risk of developing silicosis, so there is a need for follow-up studies to determine whether the volcanic ash is a long-term respiratory health hazard. This is especially pertinent because the Soufrière Hills volcano is in its eleventh year of eruption with ash emissions (at the time of writing), thereby presenting a long-term exposure risk for the population (Searl et al. 2002).

The probabilistic risk assessment on Montserrat was undertaken to predict the chances of developing silicosis if the eruption and ashfalls continued for up to 20 years. The potential for developing silicosis, particularly in children, has been an important consideration in policy making during the crisis, and it has been dependent on obtaining good exposure data in the population (Searl et al. 2002). Above all, the mainstay of risk assessment in occupational studies has been the measured exposure levels to crystalline silica (mainly as cristobalite) in the ash, which was used for making broad judgements on the health risk at Mt. St. Helens and Montserrat. This cannot be taken as the sole risk criterion for the development of silicosis, however, as the presence of other minerals in the ash can have a profound influence on the toxicity of crystalline silica in the lung (Health and Safety Executive 2002). This important point is rarely made in any of the studies. In addition, only at Mt. St. Helens (Dollberg et al. 1984, 1986) and Montserrat (Baxter et al. 1999; Horwell et al 2003b) have the limitations of conventional chemical analytical techniques for crystalline silica been explored and allowance made for analytical variation using different methods by undertaking inter-laboratory comparisons on duplicate ash samples.

Lengthy eruptions and chronic exposure to ash also leads to concerns over the risks of COPD and lung cancer, in relation to the presence of crystalline silica, but we found no applicable studies which investigated the incidence of these diseases. The classification of crystalline silica as a human carcinogen by the International Agency for Research on Cancer (IARC) has prompted regulatory agencies to reconsider setting lower occupational exposure limits for this substance (Health and Safety Executive 2003; NIOSH 2002). The risk of lung cancer in exposed people is probably very small in the absence of pre-existing silicosis, but COPD may arise in individuals heavily

exposed to siliceous dusts in the absence of radiological silicosis (Hnizdo and Vallyathan 2003).

Guideline exposure values

The studies are inadequate for developing air-quality guidelines and occupational exposure limits to ash in volcanic eruptions, but some guidance can be given in general terms. There is a large body of dose-response information for PM_{10} based on anthropogenic particulate pollution epidemiological studies (e.g. Daniels et al. 2000; Peters et al. 2001; Zanobetti et al. 2002) allowing predictions of the short-term health effects associated with particulate pollution. These include increased symptoms of airways disease such as asthma and bronchitis, and associations with increased daily mortality from respiratory and cardiovascular causes (Expert Panel on Air Quality Standards 2001; WHO 2000). However, urban air pollution is a mixture of coarse particles, including ordinary dusts, as well as the products of vehicle exhaust emissions which are believed to be the most toxic factors, and so their applicability to volcanic risk is limited.

Nevertheless, guidelines on airborne particulate safety levels were developed on Montserrat (Table 5) based on the UK standard for PM_{10} of 50 μg m^{-3} calculated as a 24-h rolling average (Expert Panel on Air Quality Standards 1995, 2001) and the US exposure limit of 50 μg m^{-3} for crystalline silica, a time-weighted average exposure applied to workers who work 40 h per week for 46 weeks per year for 40 years (NIOSH 2002). This level was also adjusted for 24-h exposure in the general population on the island using a factor of five, as a guide to limiting exposure (Searl et al. 2002), but there was no published precedent for setting such a guidance value.

Future work

Most explosive eruptions form fine, respirable ash which could be the cause of an acute or chronic health hazard for the exposed populations. Ash should be routinely studied for particle shape (to exclude the possible presence of asbestiform fibres) and should be assessed for the proportion of particles <10 μm and <4 μm. The coarser fraction may be important in provoking asthma or bronchitis symptoms, but the finer fraction may also have a role in provoking inflammatory responses in the airways and the alveoli. Ash from magma with a silicon dioxide composition of less than 58% is unlikely to contain crystalline silica, but this review shows the need to study the crystalline silica content of ash from the more siliceous magmas, and the potential for free radical generation in all magma types. In large or long-lasting eruptions, risk assessments should be undertaken to determine the likely health impact on local populations. These will require detailed estimation of cumulative exposure to erupted and re-suspended ash, as well as a minimum number of epidemiological and toxicological studies.

Investigations are needed immediately after eruptions where respirable ash is emitted to allow rapid assessment of the health hazard to affected communities. In the past, volcanologists have not routinely analysed ash for its health implications, so there are few relevant studies available on the proportion and characteristics of the respirable material in most eruptions. Future work needs to involve a dedicated database of well-characterised ash samples, providing information on quantities of respirable material, composition and physical and chemical surface properties. Guidelines for collection of ash and analysis of samples have been prepared under the International Volcanic Health Hazard Network (IVHHN), an IAVCEI commission, to standardise the systematic collection of samples and documentation of the characteristics of volcanic ash. Future collaboration between volcanologists and health professionals is essential so that human health impacts can be correlated with different eruptions and emitted ash types, and a sound base of evidence can be established on the health effects of volcanic ash.

Table 5 Air quality alert levels (1-h averages, μg m^{-3}) for PM_{10} on Montserrat

PM_{10} (μg m^{-3})	Ash alert level	Recommended action
<50	Low	None
51–100	Raised	Those who have experienced effects on health during past dusty episodes, especially asthma sufferers, should ensure they have masks available
101–300	Very high	Masks should be worn
>300	Alert	Masks should be worn and efforts made to reduce exposure

Adapted from Searl et al. (2002)

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