


## AN ABSTRACT OF THE DISSERTATION OF

Bernadette Mae Longo for the degree of Doctor of Philosophy in Public Health  
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Title: The Kilauea Volcano Adult Health Study, Hawai'i, U.S.A

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After 22 years of eruption at Kilauea Volcano, Hawai'i, an environmental, epidemiological, and ethnographic study was conducted to assess for adverse health effects from chronic exposure to volcanogenic air pollution. Environmental data from 2003 indicated that Kau District, from 37 to 74 km downwind from the eruption, was being exposed to concentrations of volcanic sulfur dioxide gas (SO<sub>2</sub>) and fine aerosol particles ( $\leq 0.3\mu\text{m}$ ) that warranted public health concern. Ambient SO<sub>2</sub> average concentrations, measured with diffusion tubes, ranged from 6 to 34 ppbv over three-weeks. SO<sub>2</sub> penetrated indoors up to 71% of the ambient concentration. Fine aerosol concentrations, measured with a cascade impactor, ranged from 0.61 to 11.82  $\mu\text{g}/\text{m}^3$ . Volcanogenic air pollution is strongly correlated with altitude, displaying a pattern of increasing fine aerosol particles and decreasing SO<sub>2</sub> with rise in elevation, attributed to rapid oxidation as diurnal wind patterns blow the eruption plume from oceanic terrain landward to more abundant oxidation sources. In 2004, health effects were assessed by a cohort prevalence survey of 335 randomly sampled adult residents. Exposed cohorts of SO<sub>2</sub> and sulfate aerosol were from Kau District, and an unexposed control cohort from the extreme north end of the island. Prevalence ratios between the exposed cohorts and control revealed substantially increased prevalence of cough (310%), phlegm (270%), throat irritation (600%), rhinorrhea (740%), sinus congestion (160%), eye irritation (430%) and bronchitis (210%). Blood pressure and respirations were also significantly elevated. Elderly non-smokers had an 8 beat per minute faster average pulse rate than control counterparts. Qualitative descriptions of health responses to the eruption were obtained by open-ended questions and unstructured interviews in the natural setting.

Thirty-five percent of exposed participants perceived that their health had been affected by the eruption. Current and former smokers described being most affected. The human-health response to chronic exposure of volcanogenic air pollution may result in a sulfur illness syndrome with identified primary and secondary characteristics. Adult populations with long-term residency in active and degassing volcanic areas may experience an excess burden of cardio-pulmonary illness. Further work is warranted in all communities along Kilauea's plume path and at other degassing volcanoes worldwide.

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**The Kilauea Volcano Adult Health Study,  
Hawai'i, U.S.A.**

**by  
Bernadette Mae Longo**

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Presented on January 12, 2005.

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## CONTRIBUTION OF AUTHORS

All these data were collected solely by the author. Dr. Annette Rossignol assisted with the study design and writing of Chapters 2 - 4, and the data analysis of Chapter 3. Dr. Anita Grunder assisted with the study design, interpretation of the data, and writing of Chapter 2. Dr. Raymond Chuan provided technical assistance for Chapter 2. Dr. Joshua Green, MD, provided clinical supervision during the fieldwork for this study and assisted with the interpretation of the data for Chapters 3 and 4. This research was funded by the author.

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# **THE KILAUEA VOLCANO ADULT HEALTH STUDY, HAWAII, U.S.A.**

## **INTRODUCTION**

Throughout recorded history human populations have been challenged to interact with volcanoes. As population increased over the last millennium, migrations moved more people into Earth's volcanic areas. It is estimated that nearly 500 million people are at risk from eruptions of the world's 600 known active volcanoes (Baxter et al., 1999).

While vast amounts of human health research have been conducted on urban air pollution from anthropogenic sources, the natural environments of volcanoes have seen limited research into health effects on nearby populations. This study was the first environmental epidemiological investigation at Kilauea Volcano, on the Big Island of Hawai'i. Nearly continuous eruption with degassing activity has been occurring at Kilauea since January 1983. Fieldwork on the Big Island was conducted in August-September 2003 and April-July 2004. Results from this study were achieved as follows:

- (1) Volcanogenic sulfur dioxide (SO<sub>2</sub>) and fine particles ( $\leq 0.3\mu\text{m}$ ) were measured to assess the human habitant level of exposure,
- (2) Areas of risk to the human population were identified and classified geographically,
- (3) Prevalence of negative effects on human health were determined for population's exposed to SO<sub>2</sub> and fine particles,
- (4) Prevalence of selected diseases and symptoms in an unexposed control population were compared to the prevalence of these measures in the exposed population (prevalence ratios and prevalence differences), and
- (5) Descriptions of health effects and lived experiences in the exposed population were obtained by qualitative research methods.

This dissertation is composed of three manuscripts (Chapters 2, 3, and 4), which present the environmental, epidemiological and qualitative research of the study. In this Chapter, an extensive review of the literature describes the applicable principals of volcanology, Kilauea Volcano, the setting for the study, and the current body of

knowledge about human-health effects to air pollutants found in volcanic areas. The literature presented supports the significance and methodology of this study.

### **The Human-Environmental Interaction with Volcanoes**

Numerous hazards to human health are associated with volcanic eruptions. Since the 1600's, death by volcanic eruption numbers about 300,000 people. Causes of mortality include: pyroclastic flows of hot ash and gas, lava flows, ash fall, lahars and mud flows, gas-releases, earthquakes, landslides, tsunamis, septicemia or starvation (Blong, 1984; Baxter & Kapila, 1989; Fisher et al., 1997; Tanguy et al., 1998, Simkin et al., 2001). Cataclysmic volcanic events have occurred throughout historic time. Examples of volcanic natural disasters that have affected mankind historically include:

- (1) Vesuvius Volcano, Italy, 79 A.D., 16,000 deaths;
- (2) Krakatau Volcano, Indonesia, 1883, 36,000 deaths;
- (3) Mount Pelée Volcano, Martinique, 1902, 28,000 deaths;
- (4) Nevado del Ruiz Volcano, Columbia, 1985, 25,000 deaths; (Press & Siever, 2002; Tarbuck & Lutgens, 2002).

Human health is compromised by volcanic eruption. Morbidity from volcanic eruptions is mainly attributed to gas and particle emissions, ash fall, lava flows, and climactic changes. Volcanic events can lead to particle and gas contamination of the air resulting in respiratory diseases; contamination of water supplies resulting in acute and chronic diseases; fires ignited by lava and pyroclastic flows resulting in dermal burns; roof collapses from excessive weight of ash fall resulting in traumatic injuries; and loss of agricultural productivity resulting in starvation or malnutrition. Economic and transportation infrastructures can be disrupted, leading to a lack of access to needed emergency medical care, sustenance and food supplies.

In addition, displacement of individuals and populations can result in an array of mental and physical illnesses, some with long-term implications to health (Beck & Franke, 1996). Populations have been forced to migrate to safer lands or more productive agricultural ecosystems because of volcanic activity. Recently, the island of Monserrat has been devastated by the continual eruption of the Soufrière Hills volcano, displacing

most of the island's 12,000 residents, and leaving little viable land for agriculture or occupation (Tarbuck & Lutgens, 2002).

In any given year, about 50 of the Earth's active volcanoes erupt (Press & Siever, 2002). Highly populated urban areas currently under alert due to volcanic activity include Quito (Ecuador), Yogyakarta (Indonesia), and Mexico City. Following the 1980 eruption of Mount St. Helens, awareness about volcanic hazards surfaced in the people of the United States. Funds were provided to the United States Geological Survey (USGS) to monitor volcanoes. Presently, the USGS's Volcano Hazards Program researches, assesses, monitors, and assists with crisis response to volcanic activity within the United States and abroad (National Academy of Sciences, 2000). In 2003, the International Health Hazard Network (IHHN) was started to provide an organized forum for future research that encourages multidisciplinary and international collaboration. IHHN is a commission of the International Association of Volcanology and Chemistry of the Earth's Interior.

## **Study Setting**

This study was conducted on the Big Island of Hawai'i, U.S.A. (Figure 1.1). The island is composed of five shield volcanoes and their lava flows. The major population centers for the island are Hilo and Kona. The cohort areas for this study were the Kau District and the Hawi area as outlined in Figure 1.1.

The Big Island portrays a climate typical of the Hawaiian Islands. The windward, east side of the Island where Kau District, Hawi and Hilo are located, receives substantially more rainfall than the dry western, leeward side of the Island where Kona is located. There are seasonal climactic variations in rainfall with the heaviest amounts occurring in winter and spring.

Topography and meteorology play integral roles to the dispersion and human exposure of air pollution from Kilauea Volcano. Prevailing northeastern Pacific trade winds (8-16 km/hr) pass over Kilauea carrying volcanic emissions southwesterly over the Kau Desert, out over the ocean and terrestrially past the towns of Pahala and Na'alehu. The plume wraps around Mauna Loa Volcano (13,679 ft.), moving northwesterly along

the Kona coast, until finally being sent out to sea. The island has a trade wind inversion at 1,800 meters (6,000 ft) which prevents vertical mixing, thereby creating a marine air layer of abundant water vapor, anthropogenic pollutants and Kilauea's plume products.

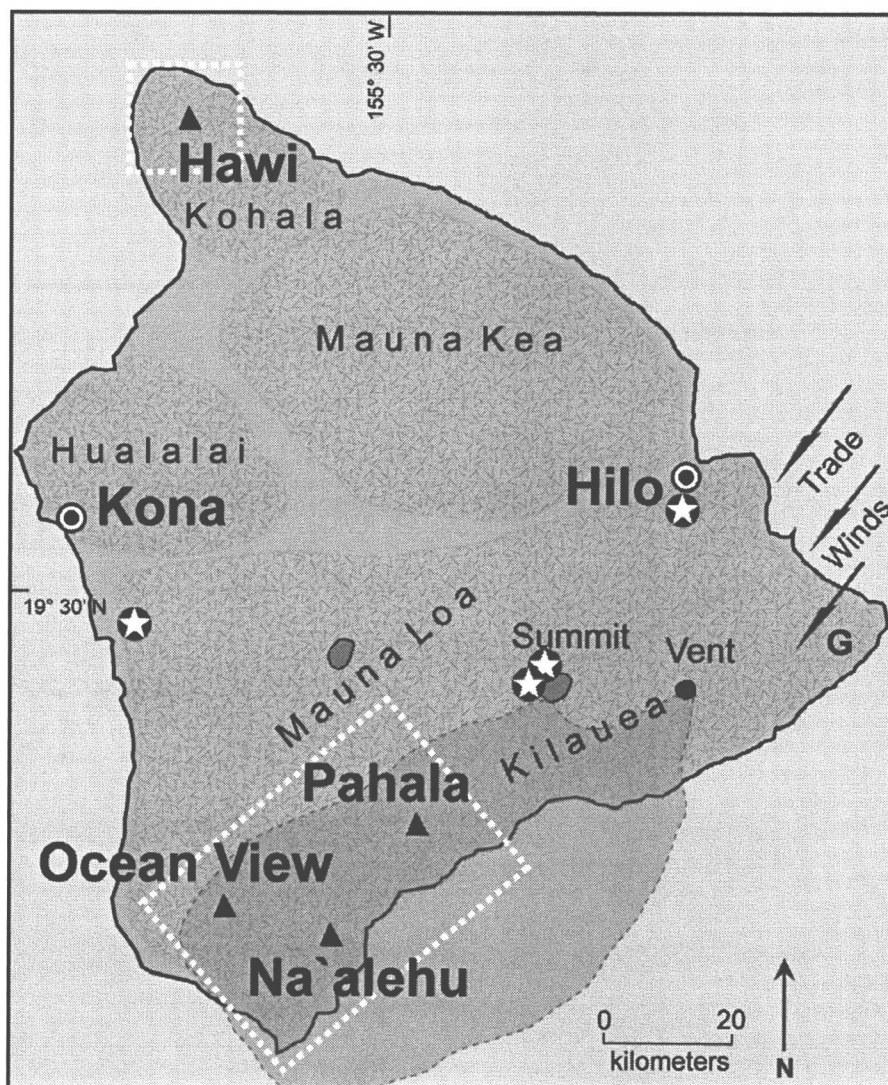


Figure 1.1 Map of the study location for the Big Island of Hawai'i, U.S.A. The cohort areas are outlined in white. Kau District has three population centers: Ocean View, Pahala, and Na'alehu. Five volcanoes are identified. Typical Pacific trade wind patterns are shown. Volcanogenic air pollution in Kau district is illustrated. Stars mark the current locations of air monitors in Kona, Hilo and two in Hawai'i Volcanoes National Park. The G marks the location of geothermal wells.

Volcanic emissions can accumulate near the summit of the volcano when trade winds are light, variable or absent (Figure 1.2). Light and variable winds are least common in summer months. In addition, occasional southwesterly “Kona” winds form from a low-pressure system that develops in winter, lasting for up to a week. Kona winds can carry volcanic emissions over nearby populated areas to the north, such as Hilo, and have been reported as far away as Honolulu on the island of Oahu. The State of Hawai‘i has fixed air monitors for SO<sub>2</sub> and particulate matter <2.5 μm (PM<sub>2.5</sub>) in Hilo and south of Kona. There are two fixed SO<sub>2</sub> monitors at the summit of Kilauea monitored by the Hawaiian Volcano Observatory (HVO) and the U.S. National Park System.

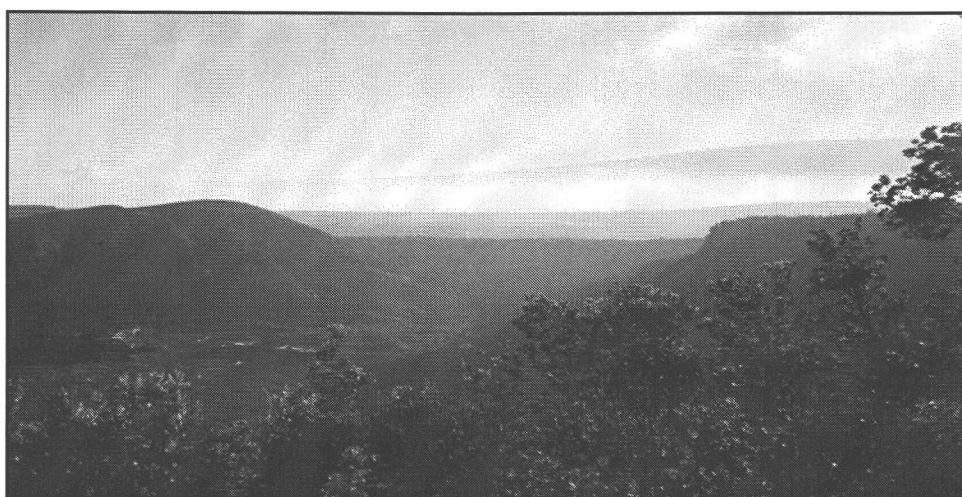


Figure 1.2 Photograph of volcanic air pollution accumulating at the summit of Kilauea Volcano during light trade wind conditions, May 2004. Orientation: looking south.

The Kau District and Hawi area share common histories of immigration and agricultural development. The first Hawaiians settled on the Big Island. Over the last 130 years, the Kau District and Hawi area served as prime agricultural land for sugar cane. More recently, both areas host macadamia nut orchards. Since the termination of sugar cane harvesting in the 1990s, these areas have undergone socioeconomic and agricultural changes. In Kau District, residents currently harvest macadamia nuts (seasonally) or travel to Kona for employment. Coffee crops have been introduced, ranching has increased, and subdivisions, such as Ocean View, continue to be built on the flanks of Mauna Loa Volcano in Kau. In the Hawi area, many residents travel to work at resorts on

the northern Kona Coast and old sugar cane land is being developed into subdivisions. Ranching continues on the flanks and summit of Kohala Volcano. Both areas are free currently from any major source of anthropogenic pollution, with the exception of mechanically-generated dust in the Pahala area from agriculture. During the years of sugar cane production, however, burning of fields occurred after each harvest.

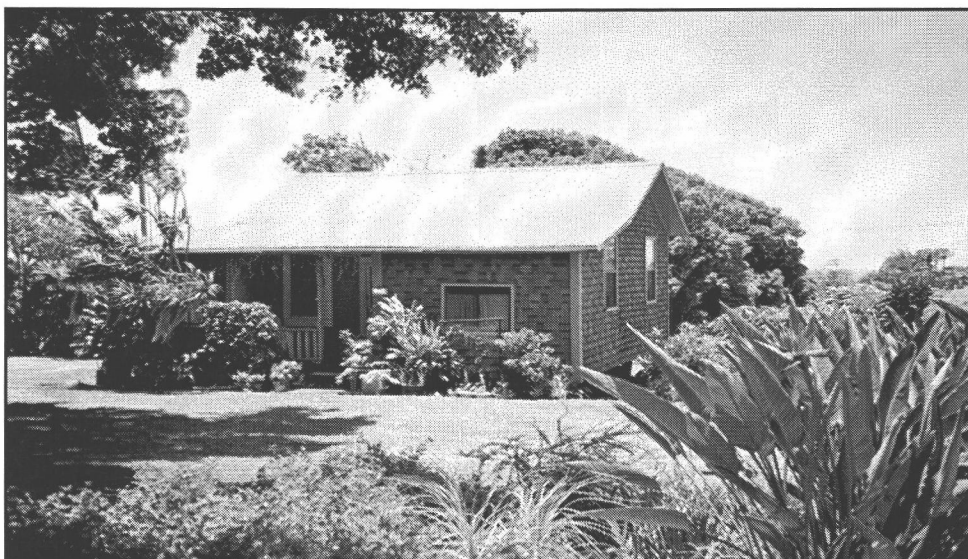


Figure 1.3 Photograph of a typical sugar plantation-style house on the Big Island.

The ethnicity of residents in these areas includes the native Hawaiians and those who migrated to Hawai'i since the 1800's. Filipinos migrated to work the sugar cane plantations. Japanese and Okinawans have created local businesses. Caucasians have mainly come from the U.S. mainland. Native Hawaiians are a people who have strived to live in harmony with sea and land. Volcanoes have always been an intricate part of their culture. Hawaiians believe the goddess "Pele" lives within Kilauea Volcano and rules the volcanoes of Hawai'i. Volcanic eruptions symbolize displeasure from Pele, and are related to a certain "Kapu" broken law, or forbidden action that has occurred (Nimmo, 1992). Eruptions both destroy and create new land, thereby transforming the environment. There is a strong cultural belief that Pele controls fate, and intervention into her actions is unnecessary and not advised (Warrick, 1979). The worship of Pele is still practiced in modern times.



## **The Principals of Volcanism**

### **Origins and styles of volcanism**

Plate tectonics on Earth is a dynamic process by which slabs of crust, or plates, are rafted on a conveyor belt of convection-driven molten material from the upper mantle (Windley, 1977; p. 225-240; Davies, 1999). The conveyor system is powered at plate margins by the push of hot upwelling mantle material at the spreading centers and the pull from the cooling slab sinking at subduction zones (Davies, 1999; Collins, 2003). This complex system of mantle-driven plate interactions is unique to our planet and produces many styles of volcanism (Table 1.1).

Most volcanism on Earth occurs under the oceans in spreading centers along mid-ocean ridges as a passive, effusive-style of eruption that forms the oceanic plates (Gregg & Fink, 1995). Magma generated at these spreading centers along divergent plate margins, produces low viscosity (low silica content) basaltic lavas and dominantly effusive axial volcanoes (shield-like accumulations of pillow lavas in rifted valleys) (Gregg & Fink, 1995). In contrast, at the subduction zones along convergent plate margins, andesite and dacite magma of higher viscosity develops high peaked stratovolcanoes that produce more commonly explosive style eruptions as evident in Oregon's Cascade volcanoes (Francis, 1993).

Volcanoes also develop within the plates over mantle plumes in the Earth's mantle known as hot spots (Davies, 1999; Courtillot et al., 2003). When intra-continental hot spots develop beneath the thick continental crust, crustal melts produce magma with a high silica content and high viscosity (Francis, 1993). These magmas produce volcanoes that become extremely explosive and erupt rhyolite magmas such as the Yellowstone Caldera. When hot spots develop beneath the ocean crust, however, basaltic magma is effusively erupted and gradually climbing shield volcanoes build and grow into islands such as the Hawaiian Islands (Davies, 1999).

Hawaiian shield volcanoes are the largest volcanic landforms on Earth (Francis, 1993). Shield volcanoes are commonly created as the oceanic plate moves over a hot spot, or a deep mantle plume of magma, that develops intraoceanic away from the spreading center ridges (Press & Siever, 2002; Courtillot et al., 2003). The Hawaiian-

Emperor volcanic chain forms a linear pattern of shield volcanoes, like beads on a necklace, which represent effusive volcanism produced over the Pacific hot spot for over 65 million years (Clague & Dalrymple, 1987; Leyrit & Montenat, 2000). Hawaiian volcanoes have a high frequency of eruption (Peterson & Moore, 1987) and are presently active on the Big Island of Hawai'i.

Explosive volcanism is a style associated with all types of volcanoes and is environmentally destructive and catastrophic (Table 1.1). Walker (1973) classified explosive volcanic eruptions based on the dispersal and degree of fragmentation of the primary volcanic fragments expelled during the eruption, and production of pyroclastic deposits. Six types of explosive eruptions styles are characterized that represent an increase in explosivity from hawaiian, strombolian, plinian, vulcanian, surtseyan, ultraplinian, and phreatoplinian (Wright et al., 1980) (Table 1.1).

According to Leyrit and Montenat (2000, p.21), explosivity is dependent on the silica and volatile content of the magma. An increase in the silica content increases the viscosity and the explosivity of an eruption. In general, volcanoes along convergent plate margins have displayed high magnitude explosive events with a low frequency of occurrence (Chester, 1993). Some geologists suggest that magmas evolve at subduction zones from the process of melting that is induced by the down-going oceanic slab (Defant & Drummond, 1990). They theorize that melts develop in the overlying mantle wedge, pond at the base of the crust, buoyantly rise upward into the crust, and mix with crustal melts (Gill, 1981 p. 56-59; Davidson et al., 1991). These magmas are rich in silica and produce magmas of higher viscosity relative to the Hawaiian types. They hold more dissolved gases, thereby creating buoyant magma plumes that can erupt violently with enormous ash-laden eruption columns ejecting gases and ash into the stratosphere (Francis, 1993; Leyrit & Montenat, 2000; Press & Siever, 2002).

There are, however, exceptions to the rule. Explosivity is also possible during Hawaiian style eruptions of low viscosity basaltic magma; when interacting with meteoric water (surtseyan), when bubbles of gas rise rapidly (Hawaiian fire fountains), or when the bubbles coalesce and burst (strombolian-like) (Cas & Wright, 1988) (Table

1.1). Surtseyan events are called phreatomagmatic eruptions that are locally explosive and often destructive.

### Products of volcanism

Two principal types of volcanic products are erupted from volcanoes: (1) lava flows, and (2) pyroclastic rocks (Fischer & Schmincke, 1984; Cas & Wright, 1988; Francis, 1993). Lavas are uniform flows of magma that emanate from effusive volcanoes, and in some basaltic eruptions they are fed by fire fountains characteristic of the eruptions of Kilauea Volcano (Cas & Wright, 1988; p.59-91). Pyroclastic rocks are deposits composed of primary volcanic fragments produced by the fragmentation of magma during explosive activity (Fisher & Schmincke, 1984; p. 8 and 89-127). Lava flows are the dominant effusive products of volcanism and pyroclastic deposits are the dominant explosive products of explosive eruptions. Both lava flows and pyroclastic rocks produce an array of textures and deposits influenced by chemical composition, volatile and crystal contents, and periods of cooling (Francis, 1993; Press & Siever, 2002).

Lava flows and pyroclastic accumulations from volcanoes are classified by chemical composition and mineralogy, and they vary considerably in size, shape, texture, vesicularity, explosivity, and tectonic setting (Table 1.1) (Cas & Wright, 1988; Francis, 1993). Viscosity is an important physical property of volcanic rocks and a measure of a liquids resistance to flow. Viscosity is proportional to the silica content and increases as silica increases (Table 1.1) (Press & Siever, 2002). The highly viscous felsic rocks such as dacite and rhyolite are rich in silica and alkali elements, but generally low in iron and magnesium (Press & Siever, 2002). Intermediate volcanic rocks such as andesite have moderate silica and alkali (K and Na) compositions (Press & Siever, 2002). In contrast are the mafic volcanic rocks, such as the Hawaiian basalts; low in silica and high iron and magnesium (Press & Siever, 2002). Thus, the effusive nature of basaltic eruptions is related to the ease of gas escape due to high temperature and a low viscosity or high fluidity (Cas & Wright, 1988).

## Hawaiian lava flows

Kilauea Volcano is an excellent site to witness basaltic lava flows in action. Characteristic features of basaltic lava flows at Kilauea include: 1) pahoehoe; 2) A'a; 3) tube-fed pahoehoe flows, 4) pillow lavas, and 5) entrail pahoehoe (Hazlett, 1987). Hawaiian lava flows typically begin as pahoehoe with a smooth to ropey surface texture that are fluid and fast moving, traveling downhill at 10-300 meters per hour (Press and Siever, 2001). In Hawai'i, a pahoehoe flow has been clocked at 30 kilometers per hour (Tarbuck & Lutgens, 2002). Further from the vent the pahoehoe flow begins to crystallize and become A'a lava (Cashman, 2001). A'a lava flows have jagged spinosed fragmented surfaces and are also termed clinkery because of the sound the fragments make when grinding against one another during flow. A'a flows are generally slower moving and average 5-50 meters per hour, with high steep flow fronts (Tarbuck & Lutgens, 2002; Cashman, 2001).



Figure 1.4. Photograph of pahoehoe lava texture from Kilauea Volcano, June 2004.

Lava tubes are characteristic features of shield volcanoes (Cas & Wright, 1988). Lava tube systems efficiently carry liquid basalt great distances at rapid speeds while preserving temperatures and consistency of the lava (Cashman, 2001). In Hawai'i, these tubes often carry lava directly to the ocean's coast, thereby creating new land when lava

enters the sea. Surface flows, or breakouts can occur when a tube system becomes plugged.

The superheated pahoehoe enters the ocean water from the tube system with violent phreatic explosions. The remaining lava pushes below the waves and forms pillow lavas (streams of overlapping lava toes with a cross section that resemble pillows) (Hazlett, 1987; Umino et al., 2002). When lava flows reach the ocean and mix with seawater, a hydrochloric acid aerosol (pH 1.5-2.5) called "laze" is formed and released in a plume at the site of entry (Kullman et al., 1994; Chuan, 1995; Sansone et al. 2000).

Entrail pahoehoe has a texture that resembles intestinal entrails (Hazlett, 1987). This flow type is common when A'a flows spill off the pali, or cliff, and the lava is forced to increase speed due to gravity (W. Rose personal communication, 1985). Entrail pahoehoe (lava toes) is also found when lava breaks out of a tube and travels over the surface some distance until it cools or is covered by other flows (Umino et al., 2002).

#### Hawaiian pyroclastic fragments

Explosive Hawaiian activity caused by the rapid release of volatiles in the magma produces fire fountaining and the famous curtains of fire (Francis, 1993). Hawaiian basalts contain small proportions of volatile gases, however these gases can collect in the upper part of the magma chamber so that when an eruption begins powerful fountains spray liquid basaltic lava outwards. Early on in Hawaiian eruptions, magma can move from the summit caldera down one of the volcanoes rifts (East or Southwest). As magma moves towards a distal flank eruption site, dramatic curtains of fire, or liquid lava squirting out of cracks along the rift (creating spatter ramparts), can be observed. Lava erupting simultaneously at the summit and down a rift has been observed at Kilauea. Once a site is established, lava release becomes localized. Fire fountaining releases pyroclastic fragments (ejecta) that accumulates and forms a cone. Explosive fire fountaining occurs concurrently with the effusive release of large amounts of lava at the cone or nearby fissures.

Pyroclastic fragments typical of Hawaiian eruptions include: 1) spatter at the vent, and 2) airborne material down wind of the vent called scoria (Cas & Wright, 1988). Spatter deposits develop as cone and ramparts that line the vent area (Hazlett, 1987). The

scoria consists of Pele's Tears (small glassy black droplets of lava), Pele's Hair (fragile threads of golden glass), and reticular pumice (highly vesiculated sponge-like golden glass) (Hazlett, 1987; Cas & Wright, 1988). During a fountaining event small pyroclastic fragments (<2mm ash) have been known to remain airborne for 10's of kilometers (Fisher and Schmincke, 1984). Although ash fall is a rare event at Kilauea, during a fountaining event at Pu'u 'O'o Cone in 1985, Kona winds blew and deposited fine ash in Hilo over 25 kilometers away (A. Longo, personal communication, 1985).

### The history of Kilauea Volcano

Kilauea Volcano is considered one of the world's most active shield volcanoes and one of the most researched. Kilauea is one of three active shield volcanoes (Mauna Loa and Hualalai) and two dormant shield volcanoes (Mauna Kea and Kohala) located on the Big Island of Hawai'i. The Hawaiian Islands were formed by volcanism from an active hot spot below the Pacific Plate. Two major trends of volcanism have been identified in the Hawai'i Emperor chain, the Loa trend which includes Mauna Loa and the developing Loihi submarine volcano (~ 40 Km SE of Pahala), and the Kea trend which includes Mauna Kea and Kilauea (Clague & Dalrymple, 1987). The age for Kilauea is estimated at 400,000 years (Peterson & Moore, 1987). According to the USGS (2003) about 90% of the surface of Kilauea is formed by lava flows less than 1,100 years old and 70% of the volcano's surface is younger than 600 years. Kilauea and other Hawaiian eruptions are categorized as low magnitude/high frequency events (Chester, 1993). Due to the mostly effusive nature of these volcanoes, few deaths have been attributed to Hawaiian eruptions.



Figure 1.5. Photograph of Kilauea Iki Lava Lake on the summit of Kilauea, May 2004.

A unique feature of Kilauea and Hawaiian eruptions is lava lakes (Francis, 2000). Long-lived activity during an eruption can form lava ponds or large lava lakes (Figure 1.5). Eruptions that lead to the formation of a lava lake have occurred at the summit such as Kilauea Iki (1959), or down a rift at Kupaianaha (1986). The geologic record reveals that during the 19<sup>th</sup> century activity at Kilauea focused mainly at summit lava lakes, however in the 20<sup>th</sup> century more flank eruptions became comparatively common (Symonds et al., 1994).

Loss of life may be comparatively minimal; however, destruction of human dwellings and the birth of new land is an evolving process at Kilauea. During the 20<sup>th</sup> century an East Rift eruption resulted in the destruction of the entire town of Kapoho, displacing all residents and leaving a flattened field of lava flows for new life to begin again.

The current Pu`u `O`o-Kupaianaha (POK) eruption, on the East Rift, began on January 3, 1983. The USGS considers this eruption the most destructive since A.D. 1790 and the longest-lived in the last 600 years (Heliker & Brantley, 2003). According to the USGS (Heliker & Brantley, 2003), the site of the initial eruption was down the East Rift 19 kilometers (km) away from the summit (Figure 1.6) where a cinder cone called Pu`u



'O'o was formed. Initially, eruptions were sporadic, monthly in sequencing, and produced fountaining episodes (up to 1,500 feet high) that spewed vast amounts of tephra locally and produced high SO<sub>2</sub> emissions. The Pu'u O'o Cone was built to a maximum height of 835 feet.

In July 1986 however, the volcano moved from intermittent episodes of eruption at Pu'u 'O'o into a continuous status from a new vent area 3 km away. The Kupaianaha lava pond formed over the vent. Large amounts of lava were released through tube systems. Surface flows and lava tube systems have destroyed 8 miles of highway, 189 structures including the town of Kalapana, and added 544 acres of land to the state of Hawai'i (Heliker & Brantley, 2003). In 1992, Kupaianaha stopped release of lava and shortly thereafter activity returned to Pu'u 'O'o. From 1992 to present day continuous effusive release of lava ( $\sim 0.0005 \text{ km}^3/\text{day}$  or  $500,000 \text{ m}^3$ ) and gases have been occurring at and near Pu'u 'O'o (Heliker & Brantley, 2003; Sutton et al., 2000).

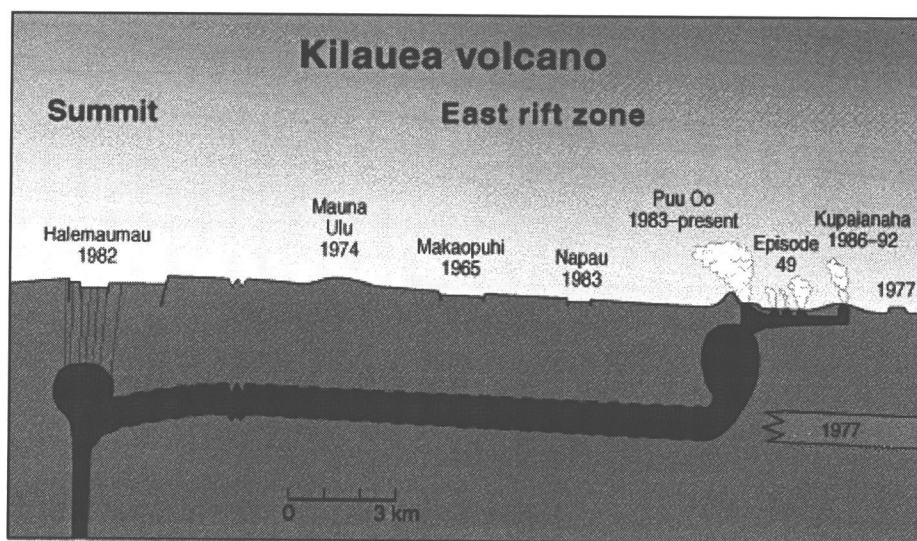


Figure 1.6. Schematic diagram of Kilauea Volcano: Summit and East Rift eruption. (Credit with permission from Mike Garcia, Hawai'i Center for Volcanology, 1998).

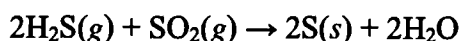
## Volcanogenic Air Pollution

### Sulfur chemistry

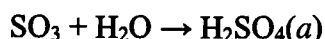
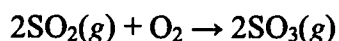
Various sulfur species can be found in volcanic environments (Table 2). In geothermal areas, hydrogen sulfide (H<sub>2</sub>S) is the dominant gas. Hydrogen sulfide slowly



reacts with atmospheric oxygen to form  $\text{SO}_2$  gas. During a volcanic eruption, magma releases sulfur dioxide ( $\text{SO}_2$ ) and hydrogen sulfide ( $\text{H}_2\text{S}$ ). These two gases ( $g$ ) can react with each other within minutes (reduction) to produce elemental sulfur particles ( $s$ ) and water vapor (USGS, 1996).

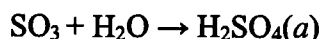
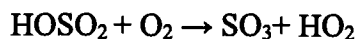


Using the natural sulfur cycle for air as a model,  $\text{SO}_2$  gas can directly oxidize to  $\text{SO}_3$  gas, which in turn, in the presence of moisture in the air will rapidly hydrate to a colloid of sulfuric acid aerosol ( $a$ ) ( $\text{H}_2\text{SO}_4$ ) (WHO, 1979).



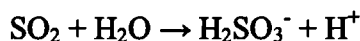
Direct oxidation, however, is a slow process. Other oxidation mechanisms include photo-oxidation with sunlight, reactions with the hydroxyl radical ( $\text{OH}\cdot$ ), and other reactions (with ozone ( $\text{O}_3$ ), bi-radicals, ground-state atomic oxygen and peroxy radicals  $\text{HO}_2\cdot$  and  $\text{RO}_2\cdot$ ) (Godish, 1997). The atmospheric lifetime of  $\text{SO}_2$  is about 2-4 days (Godish, 1997).

A major mechanism for conversion of  $\text{SO}_2$  is  $\text{OH}\cdot$ , which readily reacts in the gas-phase by photoinitiation to form  $\text{H}_2\text{SO}_4$  (ATSDR, 1998; Seinfeld 1986). Reaction with  $\text{OH}\cdot$  is as follows:



Sulfuric acid is a strong, stable diprotic acid that requires extreme heating to decompose to sulfur trioxide and water. Sulfuric acid may react further (nucleation) with compounds in the air to produce other aerosol sulfates (Godish, 1997). Aerosols are liquid droplets or crystalline solids suspended in air that range in size from ultra fine ( $<0.1 \mu\text{m}$ ) to fine ( $<2.5 \mu\text{m}$ ). The most common sulfate salts are the hydrogen sulfates ( $\text{HSO}_4^-$ ), such as ammonium sulfate ( $[\text{NH}_4]_2\text{SO}_4$ ), and the sulfates ( $\text{SO}_4^{2-}$ ) including sodium sulfate ( $\text{Na}_2\text{SO}_4$ ). The average atmospheric lifetime of aerosols is less than 3 days (Godish, 1997).

In addition, SO<sub>2</sub> can dissolve readily into atmospheric water (fog, rain droplets, clouds) where aqueous-phase oxidation occurs with (1) hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) to rapidly form H<sub>2</sub>SO<sub>2</sub> (Seinfeld & Pandis, 1998), or (2) sulfurous acid (H<sub>2</sub>SO<sub>3</sub>) that slowly oxidizes in the presence of air to form H<sub>2</sub>SO<sub>4</sub>.



Lastly, H<sub>2</sub>SO<sub>4</sub> can condense onto the surface area of an existing particle, such as adherence to particulate matter (Godish, 1997). All these chemical processes involving species of sulfur in ambient air are affected by the presence of sunlight (photoinitiation), amounts of humidity and rainfall (aqueous-phase reactions), the presence of competitors and other air pollutants (initiators or inhibitors), or other natural catalyzing agents (manganese and iron salts) (WHO, 1979; Pasiuk-Bronikowska, 2002).

The trophospheric fate of SO<sub>2</sub> from volcanic origin includes both unique and standard scrubbing mechanisms and sinks. In work on the eruption plume at Vulcan Fuego in Guatemala, Rose et al. (1978) found that 33% of the sulfur, along with chlorine, was absorbed onto tephra surfaces. Tephra, which contains iron, could act as a catalyzing agent with sulfur. Rain is a natural scrubbing mechanism of SO<sub>2</sub> that results in a lowering of pH in rainwater – hence acid rain. As seen in the findings of Nicholson et al. (1996) at Poas Volcano in Costa Rica, lower SO<sub>2</sub> exposure levels were found during the rainy season, rather than the dry season. At Kilauea, Sutton et al. (2000) found that local acid rain resulted in the leaching of toxic chemicals (i.e., lead) into drinking water from residential water catchment systems. At Masaya Volcano in Nicaragua, wet and dry deposition of SO<sub>2</sub> strongly affected the vegetation in areas of high concentrations and elemental sulfur accumulation was noted (Delmelle et al., 2002). The contemporary hypothesis is that SO<sub>2</sub> converts over time to sulfur aerosols, resulting in less SO<sub>2</sub> at distal sites from the volcanic source and more sulfur aerosols. According to HVO volcanologist A.J. Sutton (personal communication, Nov. 2002), by the time the plume reaches distant Kona, it is mostly aerosol with little primary SO<sub>2</sub> remaining. In fact, Dr. Ray Chuan (personal communication, Nov. 2002) believed the pollutant of most concern to health may not be SO<sub>2</sub>, but rather are the sulfur aerosols.

The atmospheric fate of volcanic SO<sub>2</sub> from effusive activity is also affected by the following: atmospheric air mixing, dispersion patterns, inversions that can trap pollutants, and topography. The volcanic plumes from the basaltic volcanoes Kilauea and Masaya are strongly affected by topography and meteorology. As described previously at Kilauea, normal trade wind direction results in the plume being carried out over the ocean, allowing mixing with seawater aerosols, and carried around the flank of Mauna Loa for long distances. Whereas at Masaya Volcano, the stable wind patterns carry the plume into the topographic low of the caldera where the population resides, causing air stagnation, and limiting the mixing/dispersion of the plume's gases with background air (Delmelle et al., 2002).

### Volcanic gases and aerosols of Kilauea Volcano

Gases emitted by volcanoes display geochemical signatures (constitution and emission rate) that correlate with magmatic processes during quiescence, pre-eruptive, and eruptive phases. At Kilauea Volcano, the non-eruptive baseline emission rate of SO<sub>2</sub> is 150 tons per day (USGS, 1996). According to Gerlach (2001) early pre-eruptive gases (H<sub>2</sub>O, CO<sub>2</sub>, and H<sub>2</sub>S) dominate, but as an eruption nears, the geochemical signature of the gases emitted changes (H<sub>2</sub>O, CO<sub>2</sub>, SO<sub>2</sub>, H<sub>2</sub>S, HCL, HF, CO, and H<sub>2</sub>). The total gas percent contribution of CO<sub>2</sub> decreases as SO<sub>2</sub> significantly increases, often signaling a possible eruption. When an eruption occurs, various species of sulfur, water vapor, and CO<sub>2</sub> become the major constituents of gas emissions from volcanoes.

Volcanic air pollution is a significant human health concern at Kilauea Volcano. Unlike explosive stratovolcano eruptions that send particles and gases into the stratosphere, more effusive Hawaiian style volcanoes release gases into the lower levels of the troposphere – the human habitat (Figure 1.7). Kilauea's SO<sub>2</sub> emission rate during the current POK eruption has averaged 1,800 - 2,000 tons/day being released into the lower troposphere mostly from the East Rift vent of the eruption, the summit, and along surface lava flows (Sutton et al., 2000). Of this amount, the summit caldera releases about 90 to 150 metric tons of SO<sub>2</sub>, leaving the majority of the emissions from the East Rift eruption site (USGS, 1998). Even higher SO<sub>2</sub> emissions have occurred. In 1983 during a fountaining episode, Kilauea emitted SO<sub>2</sub> at a rate of 32,000 tons/day for 15 hours

(Greenland et al., 1985). Compare the emission rates of Kilauea to the EPA's definition of a "major source" of contamination as that which emits 10-25 tons per year of a hazardous air pollutant(s) (EPA, 1994).

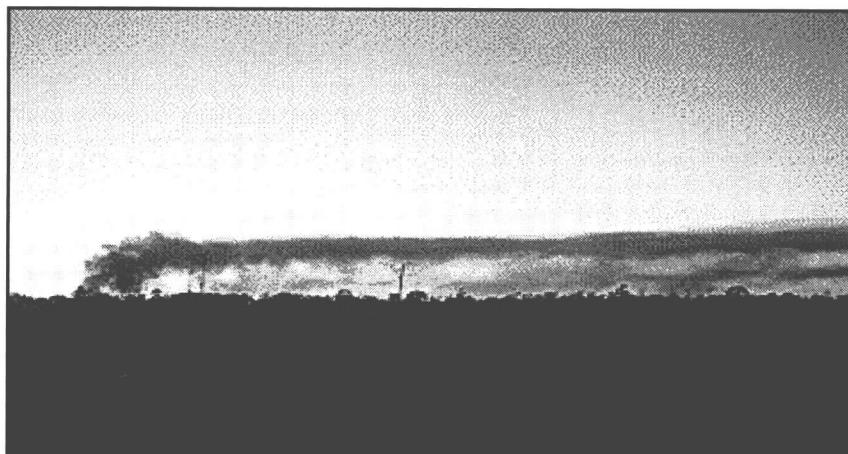


Figure 1.7 Photograph of the low altitude eruption plume of Kilauea Volcano (Credit: USGS, Volcano Hazards, R.W. Decker, March 1, 1983).

In the absence of strong winds, volcanic smog called "Vog" accumulates. According to the HVO the composition of Vog is primarily  $\text{SO}_2$ , water and aerosols containing sulfuric acid, sulfate compounds, selenium, mercury, arsenic and iridium (Sutton et al., 2000). The composition of Vog varies at locations from predominately  $\text{SO}_2$  near vent and summit areas, to  $\text{H}_2\text{SO}_4$  aerosol (less than one micron in size, centered  $\sim 0.3 \mu\text{m}$ ) and un-reacted  $\text{SO}_2$  at distal sites (Chuan, 1997, 1998; Sutton et al., 2000). Aircraft sampling of the plume, using a laser optical counter, measured the volcanic aerosol at  $\sim 0.3 \mu\text{m}$ , while background marine aerosol was larger at 2-3.0  $\mu\text{m}$  (Clarke & Peter, 1991).

Chuan (1995) proposed that volcanic  $\text{H}_2\text{SO}_4$  originates as both a primary and a secondary pollutant at Kilauea.  $\text{H}_2\text{SO}_4$  is formed in the eruption plume before it exits the vent and is dispersed in a range of sizes. In contrast,  $\text{H}_2\text{SO}_4$  formed by oxidation of  $\text{SO}_2$  in the plume away from the vent ranges in size from 0.1 to 1  $\mu\text{m}$  (Chuan, 1997).

Analysis of Vog from distal sites identifies the signature chemicals as sulfuric acid, elemental sulfur, and sulfate compounds, especially sodium sulfate (Chuan, 1995, 1997, 1998). Sodium sulfate is believed to originate from oceanic sodium chloride and

sulfuric acid (Chuan, 1997). Constituents of Vog at Kona have a typical bi-modal peak distribution of 1.7  $\mu\text{m}$  (sodium sulfate of volcanic origin and sodium chloride of marine origin) and 0.3  $\mu\text{m}$  (sulfuric acid of volcanic origin) (Chuan, 1997, 1998).

In contrast, Hilo, located 30 miles from the vent, receives Vog sporadically when Kona winds occur. Background, Vog-free air, is distinctly mono-modal at 1.7  $\mu\text{m}$  particle size with sodium chloride marine aerosol as the dominant species (Chuan, 1997). During Vog periods however,  $\text{SO}_2$  is present and sulfate aerosols comprise 57% of the  $\text{PM}_{10}$  (with 42% identified specifically as  $\text{H}_2\text{SO}_4$ ) (Morrow, 2000). Regardless, levels of  $\text{SO}_2$  in Hilo and Kona have remained below EPA's primary standards.

From 1986 to June 2000, there were 85 documented occurrences of high  $\text{SO}_2$  gases ( $>250$  ppbv) within Hawai'i Volcanoes National Park (HPNV) (Sutton et al., 2000). These episodes are related to changes in typical wind conditions. In early 2001, HVNP developed a health advisory plan related to volcanic air pollution. When  $\text{SO}_2$  concentrations reach or exceed 250 ppbv at the Kilauea summit monitoring stations, park personnel and HVO are informed, along with Hawai'i Civil Defense and the State Health Department (USGS, January 25, 2001). Park personnel inform visitors to the level of pollution. Nevertheless, at Halema'uma'u Crater, a common tourist site on the summit of Kilauea, typical trade winds blow volcanic air pollution on visitors most days. Figure 1.8 shows the warning sign located in the parking lot at Halema'uma'u Crater. A typical exposure is 30 minutes for the short round-trip walk to view the crater. The  $\text{SO}_2$  concentration at this site was measured in this study (Chapter 2).



Figure 1.8 Photograph of the warning sign at Halema'uma'u Crater visitor site on the summit of Kilauea in Hawai'i Volcanoes National Park. Subject is one meter tall.

### Methods for volcanic gas measurement

Measurements of atmospheric volcanic gases can be obtained from a variety of techniques and instruments involving both direct sampling, and remote sensing, as well as other distance techniques. An overview of these methods and application at volcanic sites provides scientific support for the techniques to be used in this study.

#### *Direct sampling techniques*

Direct sampling of volcanic gases requires safe access to fumaroles or vents. The goal is to obtain geochemical analyses of the gases without contamination from

background air. Purposes for direct sampling include obtaining a baseline description of gases at site, monitoring over time a geochemical pattern concurrent with volcanic activity, and assessing current risk to human and animal populations, or vegetation (Tedesco, 1995). Samples of inert gases from fumaroles are collected in Dewar or titanium tubes partially filled with sodium hydroxide and later analyzed for content in a lab. Additionally, thermal waters and soil measurements for gases can be directly obtained in volcanic areas.

Health researchers recommend direct sampling of soil and fumaroles gases in populated volcanic areas. Baxter et al. (1999) studied a population living inside the caldera at Furnas Volcano, Sao Miguel, Azores. Direct testing of gases inside houses, at fumaroles, and in soil, revealed correlations between radon and CO<sub>2</sub> levels, some of which were toxic for radon (150 Bq/m<sup>3</sup>) and at elevated CO<sub>2</sub> levels (≥15%) for lethal asphyxia. SO<sub>2</sub> levels measured by passive diffusion detector tubes were not elevated. Recommendations for safety were given to the local residents (Baxter et al., 1999).

Direct sampling of volcanic gases can be accomplished by flying through a plume. A small infrared carbon dioxide analyzer (LI-COR) has been used by the USGS, specifically for CO<sub>2</sub> (USGS, 2000). The Fourier transform infrared spectrometer (FTIR) is used for measuring CO<sub>2</sub> and SO<sub>2</sub> concentrations (USGS 2000; Delmelle et al., 1999).

### *Remote sensing techniques*

A far safer analysis of volcanic gases can be obtained by remote sensing techniques (Andres & Rose, 1995). Correlation Spectrometer (COSPEC) is the most widely used form of remote sensing spectroscopy of volcanic plumes and clouds. Originally designed for pollution studies in the 1960's, this instrument is the primary method for measuring SO<sub>2</sub> in volcanology since the early 1980's (USGS, 1998; Stoiber et al., 1983). COSPEC optically measures the amount of solar UV light (energy) absorbed by the SO<sub>2</sub> molecule, thereby assessing the proportion of SO<sub>2</sub> in the volcanic cloud and compares these data with an internal standard (background SO<sub>2</sub> in atmosphere) (McGuire et al., 1995; Sutton, et al., 1992; USGS, 1998).

The validity of COSPEC to measure SO<sub>2</sub> in volcanic gases can best be illustrated by the non-valid use of COSPEC to measure CO<sub>2</sub> (Andres & Rose, 1995). Background

air has very small amounts of SO<sub>2</sub> (0.1-70 ppb) whereas volcanic emissions have much greater quantity and can be distinguished optically. In contrast, both background air and volcanic gases have large amounts of CO<sub>2</sub> (both near 300 ppmv) making it more optically difficult to distinguish the origin of the CO<sub>2</sub> gas. Experts in the field of gas geochemistry regard the COSPEC as the best current instrument for ground based, air based and hand-held SO<sub>2</sub> measurements.

The reliability of COSPEC has greatly improved by usage worldwide at over 17 volcanoes and greater than 6300 measurements (McGuire et al., 1995). The volcanic plume can change direction and dilution due to wind. Wind speed is the greatest threat to the reliability of data collected by COSPEC. Therefore, wind speed is simultaneously collected by a hand-held anemometer and used in the calculation for emission rate (Sutton et al., 1992). Gerlach (2001) has found COSPEC's reliability affected by the presence of rain, which causes a scrubbing effect on the SO<sub>2</sub>.

The COSPEC has been used weekly at Kilauea during the current 22-year eruption, and emission rates are reported with uncertainties of +/- 20 to 30 percent, with precision at 10 percent. Measurements are taken from a vehicle traversing multiple times beneath the volcanic plume at right angles while pointing the instrument up through the plume. These measurements are averaged, and provide the SO<sub>2</sub> plume concentration and emission rate (McGuire et al., 1995; Sutton et al., 1992; USGS, 1998). Kilauea's large SO<sub>2</sub> data set and weekly measurements provided this study concurrent and historical gas emission data.

Other remote sensing instruments used for monitoring volcanic gases are satellite based. The total ozone mapping spectrometer (TOMS) instrument measures UV ozone and SO<sub>2</sub> in the stratosphere, but has not been found to be effective in detecting low tropospheric gases (Symonds et al., 1994), however, in 2003 at the basaltic volcano Mt. Niyiragongo (3,469 meters) in the Democratic Republic of the Congo, TOMS has been used to estimate the SO<sub>2</sub> emission rates during effusive eruption.

### *Techniques to monitor ambient air pollution*

The pulse fluorescence analyzer is the most commonly used fixed instrument for SO<sub>2</sub> gas; recording volume mixed concentrations in parts per billion volume (ppbv). An



ultraviolet light is used to irradiate SO<sub>2</sub> molecules that are measured when they fluoresce. It has been used in general air pollution monitoring and recently at Kilauea by HVO. On typical trade wind days, gases blow south away from the summit and the SO<sub>2</sub> levels are ≤5 ppbv on the summit. Whereas on days when the winds slow or the direction changes, the summit gases blow into park areas exposing local populations to much higher SO<sub>2</sub> levels (on March 26, 2001, 216 ppbv, recorded by author at HVO).

Standard air quality data monitoring uses an optical nephelometer, an instrument that measures ambient particulate matter (PM<sub>10</sub> or PM<sub>2.5</sub>). Another is the Gardner Counter, a primitive instrument that measures the mass concentration of the total number of particles per cubic centimeter in a sample of air (condensation nuclei counts) (Ryan, 2000). These two instruments can collect data on particulate matter pollution (dust, sulfates) from volcanic, anthropogenic, and other natural sources.

### *Detector tube techniques*

Measurements of SO<sub>2</sub> gas concentration by diffusion tubes, employing both active and passive methods, has been used at some volcanic sites. Active measurements involve the use of pumps to collect samples or devices to manually pull air through collecting media into a tube. The pump type and designated flow rate for the gas and situation are critical to collecting accurate active samples. In contrast, passive measurements work on the principal of gas diffusion along a concentration gradient (Fick's First Law of Diffusion). Passive diffusion measurements are made with the assumption that SO<sub>2</sub> is completely adsorbed into the indicating substance (gel or filter) and there is no back diffusion pressure. Changes in temperature, pressure, humidity, and air movement (wind) can have positive or negative effects on the reading or sampling rate by active and passive diffusion methods (Patnaik, 1997). Since detection tubes were first used for mine safety in 1920, advances have been made to decrease or eliminate these confounding effects, and improve the accuracy and usefulness of passive diffusion measurements.

SO<sub>2</sub>-specific detector tubes have been employed using a variety of methodologies at degassing volcanic areas worldwide. Poas is an andesitic stratovolcano in Costa Rica with degassing activity that has affected the local population and ecosystem since 1986. Researchers at Poas have used long-term passive diffusion detector tubes (exposed for a

two week measurement), short-term diffusion detection tubes (four hour measurement), and active pumped detection tube samples with impregnated filter paper for sulphates (Nicholson et al., 1996). These results, along with electrolytic sensor equipment, allowed researchers to determine geographic areas exposed to various levels of SO<sub>2</sub> at human occupation heights. In addition, there were demonstrated differences in SO<sub>2</sub> concentrations for the rainy and dry seasons.

Passive diffusion detector tubes (long-term 20-day exposure measurements) were used during two consecutive spring seasons (1998 and 1999) at Masaya to map SO<sub>2</sub> levels over a large geographic area (900 km<sup>2</sup>) outwards from the source to 50 kilometers away (Delmelle et al., 2002). Recurring gas episodes have affected the local population and ecosystem at Masaya since the mid 1800's. This method allowed researchers to map levels of SO<sub>2</sub> and correlate the dispersion of the plume to topography, thereby predicting geographic exposure on health risks to the population. No investigations have been conducted to date to confirm human health effects.

Harwell Scientifics SO<sub>2</sub> passive diffusion tube samplers have been used in urban air pollution and volcanology studies (Delmelle et al. 2002; Baxter et al., 1999; Nicholson et al., 1996). These SO<sub>2</sub> specific tubes are well suited and field verified in humid tropical climates with rainfall. They consist of a perfluoroalkoxy (PFA) tube, 7 cm x 1 cm, two end caps, with the inlet cap containing a porous polytetrafluoroethylene (PTFE) membrane to prevent dust contamination and wind-driven shortening of the diffusion path. Using the principal of gas diffusion, ambient SO<sub>2</sub> is trapped on a potassium hydroxide impregnated stainless steel grid (30 µl of a 0.1 M potassium hydroxide in 10% v/v glycerol solution) within the tube. In the lab, absorbed medium is removed from exposed tubes, and absorbed SO<sub>2</sub> is extracted with 0.1 volume hydrogen peroxide solution to oxidize all forms of sulfur to sulfate. Sulfate concentration is determined by using Ion Chromatography. The mass concentration of SO<sub>2</sub> (µg/m<sup>3</sup>) in each tube over the exposure time is calculated by assuming a diffusion coefficient for SO<sub>2</sub> in the air of  $1.18 \times 10^{-5} \text{ m}^2 \text{ s}^{-1}$ , presenting a time averaged SO<sub>2</sub> environmental exposure level comparable to a 24-hour average exposure amount. Laboratory precision is  $\pm 1\%$ . For an exposure time of 3 weeks, the SO<sub>2</sub> lower detection limit is 1.4 µg/m<sup>3</sup>.

Lastly, concentration of a gas measured by passive diffusion can be affected by changes in atmospheric pressure, therefore the altitude of a sample site should be considered. An altitude flux correction (Larson & Vong, 1990) should be made to mass concentration lab data ( $\mu\text{g}/\text{m}^3$ ) analyzed under laboratory (steady state) conditions. According to Larson and Vong (1990), the ratio of diffusivity (D), using an altitude's pressure (P) in millibars and temperature (T) in kelvin, to a value at standard laboratory conditions ( $D_0$ ) is:

$$D/D_0 = (P_0/P) \times (T/T_0).$$

Standardization to an altitude specific volumetric mixing ratio concentration (personal communication Richard Vong, March 2004) should also be made to data sampled at different altitudes. Standardization allows comparison between different sets of samples.

## **Ambient Air Pollution and Human Health**

There is limited research regarding human health effects from volcanic air pollution, however numerous studies have been conducted on the human health effects of anthropogenic air pollution. Burney (2000) points out that while the literature acknowledges negative health effects of air pollution on lung function, it has difficulty defining and quantifying these effects. This knowledge is limited by: 1) not knowing a dose-response over time component, 2) differentiating the component(s) of the air pollution responsible for the observed negative effect, and 3) the long term effects of air pollution exposure to human health (Burney, 2000). In addition, human health effects during acute exposure are different than effects from chronic exposure. Literature is presented from research applicable to this study, mainly gases and aerosols containing sulfur, and particulate matter (Table 1.2).

The major human exposure site for toxic air pollutants is the respiratory system. Numerous respiratory and systemic diseases can develop from direct exposure to air pollutant particles. For exposure to occur, however many factors are involved in the human-particle interaction. These factors include: 1) the amount of particles; 2) physio-chemical characteristics of the particle; 3) location of deposition; 4) removability of

particle 5) systemic absorption characteristics and fate; and 6) the human response to particle exposure (Lehnert, 1993). Furthermore, the size of the particle appears to be related to its location of deposition in the pulmonary system. Sizes of coarse particles can be upwards from 10  $\mu\text{m}$ , particles  $\leq 10 \mu\text{m}$  ( $\text{PM}_{10}$ ) are inhalable, fine particles  $< 2.5 \mu\text{m}$  ( $\text{PM}_{2.5}$ ) are respirable, and ultrafine  $< 0.1 \mu\text{m}$  are respirable and may potentially enter the circulatory system (Godish, 1997; Suwa, 2002).

Gases are the smallest particles and have the ability to travel to the deepest areas of the respiratory system (alveoli), and may diffuse (absorption) into the systemic system (blood) or be exhaled. Transport of gases, however, can be affected by their solubility. The higher the water solubility of the molecule, the easier it is to be trapped by the moist upper and middle respiratory system, thereby decreasing the amount of deep alveolar penetration. High velocity air flow, such as during labored breathing and exercise, can carry gases much deeper into the respiratory system than during regular depth breathing.

Deposition of particles can occur by inertial impaction, gravitational settling, Brownian diffusion, and interception (Lehnert, 1993). Impaction occurs mostly in the upper airway (nasopharyngeal and tracheobronchial areas) involving particles  $> 0.2 \mu\text{m}$  in size (Lehnert, 1993). Deposition by settling occurs at the middle and small bronchioles and involves particles 0.05 to 20  $\mu\text{m}$  in size (Lehnert, 1993). Deposition by Brownian diffusion, the displacement of particles by random bombardment with gases causing collision with surfaces along the air pathway, is the most prevalent mechanism for the smallest particles  $< 0.5 \mu\text{m}$  in size (Lehnert, 1993). Nasal breathing can increase the amount of Brownian diffusion of particles in the nasal region, thereby limiting the penetration of particles ( $< 0.2 \mu\text{m}$ ) into the lower regions (Lehnert, 1993). Furthermore, this deposition is directly proportional to the amount of residency time of the particle. Deposition by interception occurs mainly with elongated particle shapes, such as asbestos fibers.

The complex mechanisms of particle deposition in human lungs have been simplified and it should be noted that other particle factors should be considered with exposure to air pollutants such as particle shape and charge. Also, human factors involved in particle deposition include the individual's style of breathing (mouth verses

nasal) and effort (relaxed versus labored), presence of airway restriction from disease, and anatomical abnormalities (scoliosis and others).

The human response to foreign airway particles and gases is quite elaborate and complex. The goal is to protect and prevent cell damage, and to capture and remove the particle. Physical barriers include the airway shape, mucociliary apparatus (mucus and cilia), cough mechanism, and hydration. Some general defense mechanisms beyond the physical barriers include: 1) macrophages in alveoli that can also migrate and line the upper conducting airways; 2) neutrophils; 3) additional mucus production in upper and middle airways; 4) neutralizing compounds (ammonia); 5) detoxifying enzymes; and 6) bronchial constriction. The deposition of some aerosols in the conducting airways can result in a substantial increase in phagocytic activity (macrophages, polymorphonuclear leukocytes, and migrating blood monocytes) (Lehnert, 1993). Consider that phagocytosis involves not only the physical engulfing of foreign particles but also chemotaxis by reactive oxygen species or enzyme release that can inadvertently damage host cells. Becker and Soukup (2003) suggested that recognition of foreign particles by human alveolar macrophages involves receptors that recognize microbial structures that are mostly sized from 2.5 – 10  $\mu\text{m}$ . It is the entire human-particle interaction that ultimately results in either a healthful recovery or human disease.

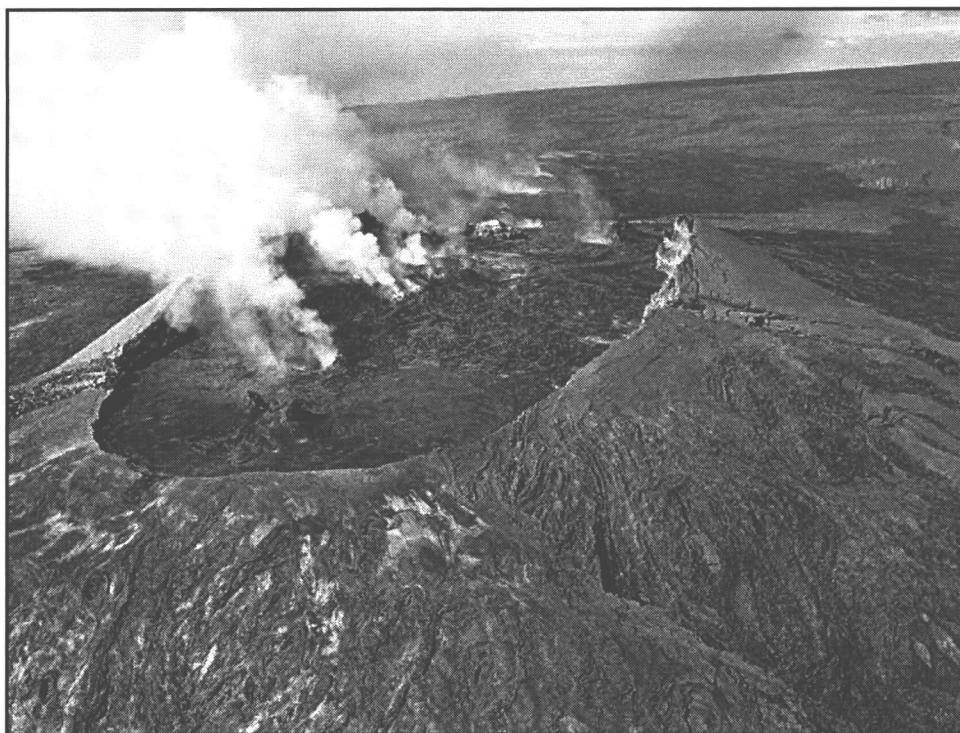


Figure 1.9 Photograph of the plume at Pu'u O'o in April 2004, during epidemiological data collection on the downwind Kau population (Credit: USGS, HVO, April 2004).

### Health effects from sulfur dioxide

Sulfur dioxide is mostly absorbed in the upper respiratory tract (40-90%) due to its high water solubility (WHO, 1979). In addition, nasal breathing and high concentrations of  $\text{SO}_2$  can increase the amount of absorption in the upper respiratory tract, thereby decreasing the amount that reaches the alveoli (Schlesinger, 1999; Amdur, 1966). Within the respiratory system,  $\text{SO}_2$  is neutralized by ammonia or forms sulfuric acid as it contacts moist surfaces. When  $\text{SO}_2$  reaches the alveoli it is dissolved into an aqueous phase, forming free radicals  $\text{SO}_2^-$  or being detoxified by oxidized glutathione, absorbed into the blood stream as bisulfite and sulfite ions, transported throughout the body, where it is metabolized by sulfite oxidase and excreted as sulfate via urine (Schlesinger, 1999; WHO, 1979; Gumuslu et al., 2001).

The major physiological effects from  $\text{SO}_2$  exposure are changes in the mechanical functions of the upper airways resulting in bronchoconstriction or increased pulmonary resistance (WHO, 1979; Godish, 1997; Koenig, 2000). Minor effects are irritation in the

nasal-pharyngeal area. In animal studies however, high SO<sub>2</sub> exposure shows an array of respiratory diseases, having been related to bronchial lesions in dogs (Drazen et al., 1982; Bhaskar et al., 1988); and bronchitis, tracheitis, and interstitial pneumonia in rats (Elifimova & Gusev, 1969) and guinea pigs (Bustueva, 1961).

### *Historical case studies of community exposures to SO<sub>2</sub>*

Historical world events identified SO<sub>2</sub> as an air pollutant causing death and disease in human populations. The Meuse Valley in Belgium in 1952 experienced five cold December days of stable air that accumulated air pollutants from the local steel mills, coke ovens, and smelters. More than 60 people died (10 times the local mortality rate) from accumulation of SO<sub>2</sub>, sulfuric acid mist and fluoride gas (Nemery et al., 2001). In the United States a small town called Donora experienced a similar tragedy; mixed air pollutants (SO<sub>2</sub> plus nitrogen oxides) killing 20 elderly or respiratory compromised individuals because of a multiple day atmospheric inversion (Anderson, 1999).

It was the deadly London Fog of 1952 that shocked the world about air pollution. Over a period of four days in cold December, burning coal resulted in SO<sub>2</sub> levels reaching a maximum of 690 ppbv (mean 570 ppbv) mixed with particulate matter measuring at 1,400 µg/m<sup>3</sup>; levels 5 to 19 times higher than today's standards (Bell & Davis, 2001) (Table 1.2). The official death rate attributed to those days in December is 3,000 more than normal (London Ministry of Health, 1954). Most affected were infants (≤4 weeks in age) who experienced a 67% increase in mortality and secondly were the elderly (+65 years) who experienced a 30% increase in mortality (Anderson, 1999). A reassessment of mortality by Bell and Davis (2001), however accounts for many more deaths from this air pollution incident. Using the current hypothesis that air pollution events can cause lagged-time health responses, such as higher susceptibility to infectious respiratory diseases and cardiac exacerbations, the excess death toll is newly estimated at 12,000 (Bell & Davis, 2001). Still today, London smog episodes continue. In urban areas of Great Britain, 3,500 annual deaths are attributable to elevated SO<sub>2</sub> levels (UK Department of Health, 1998).

Extremely high levels of SO<sub>2</sub> mixed with other pollutants are found in urban areas worldwide. Schwela (2000) reviewed world data on air quality for WHO and found that

70-80% of all surveyed cities, with populations of >500,000 residents, exceeded WHO air quality guidelines. While there was a declining trend in levels worldwide, populations in low-income countries still experience high SO<sub>2</sub> levels and an estimated excess burden of disease.

### *SO<sub>2</sub> exposure in individuals without respiratory disease*

In healthy non-asthmatic adults a dose-response relationship between SO<sub>2</sub> levels and symptomology has been measured (Koenig, 2000; WHO, 1979). Initial controlled chamber studies on healthy subjects using small sample sizes demonstrated there is no measurable effect on respiratory functioning at 370 ppbv, slight effects appear at 750 ppbv over 30 minutes [decrease in forced vital capacity (FVC) and forced expiratory volume over one second (FEV<sub>1</sub>)], increased pulmonary resistance and decreased nasal mucus flow at 1 ppmv, with additional effects of an increased pulse rate and respiratory rate at 1 to 8 ppmv (Bates & Hazucha, 1973; Anderson et al., 1974; Snell & Luchsinger, 1969; Amdur et al., 1953). More recent studies suggest that normal healthy individuals show minimal or no upper respiratory effects with exposures of SO<sub>2</sub> at 5.0 ppmv over 1-4 hours (Schlesinger, 1999; EPA, 1986). Exposure to >10.0 ppmv for a few minutes will result in irritation of the eyes, mucus membranes and throat (Schlesinger, 1999). Death from respiratory insufficiency can occur at levels of 60-100 ppmv (Schlesinger, 1999).

### *SO<sub>2</sub> exposure in sensitive populations*

Further studies on SO<sub>2</sub> have identified general population groups sensitive to SO<sub>2</sub> exposure at lower levels: 1) asthmatics; 2) children and adolescents; 3) respiratory and cardiac compromised individuals; and 4) a healthy but sensitive to SO<sub>2</sub> individuals (Koenig, 2000; Schwela, 2000; WHO, 1999; Dickey, 2000; Schlesinger, 1999). Individuals with asthma are the most sensitive group showing a non-threshold exposure dose-response relationship (WHO, 1999). Asthmatics and non-asthmatics with SO<sub>2</sub> sensitivity can experience acute responses within minutes after inhalation of 250 – 500 ppbv, such as bronchoconstriction and airway resistance that reduces the FEV<sub>1</sub> and FVC, shortness of breath (SOB), and wheezing (Schwela, 2000; Schlesinger, 1999). Even lower levels of SO<sub>2</sub> can elicit a response in asthmatics during exercising, mouth breathing, or cold air environments (Schlesinger, 1999). There is a bulk of literature



regarding asthmatic medications and their effects in asthmatics that are exposed to SO<sub>2</sub> (Koenig, 2000). Therefore, the magnitude of these responses varies in individual asthmatics and can be related to an individual's stage of disease progression, concomitant infection, medication regime and other disease factors (Schlesinger, 1999).

### *Epidemiological and community studies of SO<sub>2</sub> exposure*

Epidemiological field studies can offer further knowledge about the human-environmental air pollution interaction. A study conducted by Peters et al. (1996) during the winters of 1991 and 1992 in two of the most polluted cities in Eastern Europe (Erfurt and Weimar) revealed health effects in adult and pediatric asthmatics. The dominant air pollutant was SO<sub>2</sub>, which exceeded the WHO's standard of 125 µg/m<sup>3</sup> (40 ppbv) 250 times in Weimar and 134 times in Erfurt during the two winter study. For example, in Weimar during winter 1991, SO<sub>2</sub> averaged 236 µg/m<sup>3</sup> and reached 1,018 µg/m<sup>3</sup> on one day. Of the 257 subjects, prolonged exposure to SO<sub>2</sub> was associated with decreases in pulmonary function in both adults and pediatric subjects; with pediatric subjects having decreases three times that of the adult subjects (Peters et al., 1996).

More recently, Herbarth et al. (2001) reviewed the findings of many epidemiological studies from Germany. A study by Bredel et al. (1980) found a clear association of elevated SO<sub>2</sub> and bronchitis without fever in ≤3 year-old infants. Herbarth et al. (2001) conducted two cross-sectional studies in 1994 and 1998 in Germany to further test this hypothesis. The prevalence of bronchitis was related to the child's lifetime exposure to SO<sub>2</sub>. In fact, there was a positive correlation between SO<sub>2</sub> and bronchitis ( $r = 0.96$ ,  $p < 0.001$ ) in the 3,816 pediatric subjects. These studies suggest the association of pediatric bronchitis with chronic SO<sub>2</sub> exposure.

Substantial literature is devoted to adverse birth effects from urban air pollution. A population based case-control study conducted in Georgia, U.S.A, found an association between mothers exposed to high levels of SO<sub>2</sub> with TSP to very low birth weight babies (<1,500 grams) (Rogers et al., 2000). An adjusted odds ratio (OR) of 2.88 [95% confidence interval (CI) = 1.16, 7.13] was determined for babies from mothers with high exposures (95<sup>th</sup> percentile) of the combined pollutants. Other levels for pollutants (below 95<sup>th</sup> percentile) did not produce significant odds ratio confidence intervals. It should be

noted that a small sample size (143 cases, 202 controls) could have resulted in a lack of power in the analysis to distinguish significance at lower ratio levels.

A recent finding suggests that SO<sub>2</sub> levels as low as 11.4 ppbv have a 26% increased odds of an infant being <2500 grams at birth compared to an exposure of < 7.1 ppbv (Lin et al., 2004). In another study on infant births from 1985-1998 in Vancouver, Canada, an increase of 5 ppbv of SO<sub>2</sub> was associated with a 9% increased odds for pre-term birth, and a 7% increased odds for intrauterine growth retardation (Liu et al., 2003). The average SO<sub>2</sub> concentration during the study was 4.9 ppbv, with an average maximum of 13.4 ppbv (Liu et al., 2003) (Table 1.2).

The aforementioned studies however are in settings that have mixed air pollutants. Few studies exist that can single out SO<sub>2</sub> from other pollutants. In a small cohort study of power-station workers (N = 72), who worked 8-hour shifts in an exposed area (average concentration = 800 ppbv of SO<sub>2</sub>) for at least 2 years, researchers found an increase in cough (OR = 5.8; 95% CI = 1.8, 20.6) and sputum production compared to non-exposed cohort workers (Froom et al., 1998). Within the exposure cohort members, those who were smokers reported an even higher prevalence of cough. There were no measurable differences in FEV<sub>1</sub> between exposed and non-exposed cohorts (Froom et al., 1998).

A large cross-sectional study, along the Russian-Norwegian border, studied the exposure effects on pulmonary function in nearby populations occurring from a nickel smelter with an annual release of 180,000 tons of SO<sub>2</sub> in 1995 (Smith-Sivertsen et al., 2001). In comparison, the average annual release at Kilauea is estimated at 730,000 tons. Serious damage to area vegetation was attributed to the high SO<sub>2</sub> emissions. Using a large randomized sample (>5,000), researchers failed to measure a reduction in lung function when SO<sub>2</sub> would increase above a reference level (Smith-Sivertsen et al., 2001). Disease prevalence of respiratory illnesses was not measured in subjects. Most importantly, measured SO<sub>2</sub> levels were within acceptable WHO levels in one population and exceeded these levels only 20 days in the other population.

## Health effects from sulfur Aerosols – H<sub>2</sub>SO<sub>4</sub> and others sulfates

H<sub>2</sub>SO<sub>4</sub> is a corrosive irritant to the eyes and upper respiratory tract in some individuals at 0.5 to 2 mg/m<sup>3</sup> and coughing at 6-10 mg/m<sup>3</sup> (Schlesinger, 1999). Death caused by high H<sub>2</sub>SO<sub>4</sub> exposure would most likely occur from laryngeal or bronchial spasm (Schlesinger, 1999) (Table 1.2).

### *Sulfate aerosol exposure in individuals without respiratory disease*

Exposure to high levels of SO<sub>2</sub> are needed before there are significant changes in pulmonary defenses, but exposure to lower levels of acidic sulfates will alter airway reactivity, alveolar particle clearance, and mucociliary transport in even normal individuals (Holgate et al., 1999). In studies on healthy individuals there appears to be no effect on pulmonary function measurements of H<sub>2</sub>SO<sub>4</sub> aerosol <350 µg/m<sup>3</sup> (Utell et al., 1985). In healthy, non-smoking subjects exposed to 100 and 1000 µg/m<sup>3</sup> of H<sub>2</sub>SO<sub>4</sub> aerosol, there was however a significant slowing in bronchial mucociliary clearance, but no effects on pulmonary function (Leikauf et al., 1984). In addition, alveolar macrophages in human subjects exposed to 1000 µg/m<sup>3</sup> H<sub>2</sub>SO<sub>4</sub> aerosol have a significant depression in oxygen-dependent killing mechanisms (Zelikoff et al., 1997). Despite the results in the above studies, the U.S. EPA (1989) considers that an exposure to H<sub>2</sub>SO<sub>4</sub> at ≤1000 µg/m<sup>3</sup> does not produce evidence of inflammatory responses in animals or humans.

### *Sulfate aerosol exposure in sensitive populations*

There is some evidence that asthmatics are more sensitive to acidic sulfur aerosols than healthy individuals, however these effects are not consistent, nor as dramatic as the asthmatic response to SO<sub>2</sub> (Schlesinger, 1999). Studies on adolescents with asthma conducted by Koenig (1980, 1983) showed decrements in FEV<sub>1</sub> when exposed to SO<sub>2</sub> with NaCl aerosol (similar to Vog) and H<sub>2</sub>SO<sub>4</sub> aerosol (Koenig, 1980, 1983). In these studies, the adolescent asthmatics had a 23% decrease in FEV<sub>1</sub> when exposed to 1.0 ppmv SO<sub>2</sub> with NaCl aerosol, and an 8% decrease in FEV<sub>1</sub> when exposed to 100 µg/m<sup>3</sup> H<sub>2</sub>SO<sub>4</sub> aerosol (Koenig, 1980, 1983). Hanley et al. (1992) found significant decrements in asthmatic adolescents to an even lower level of H<sub>2</sub>SO<sub>4</sub> aerosol (70 µg/m<sup>3</sup>). Linn et al. (1997) however, found no significant results in pulmonary function or symptoms during a

chamber study of adolescents (9-12 years) with and without asthma exposed to an average of  $101 \mu\text{g}/\text{m}^3$   $\text{H}_2\text{SO}_4$  aerosol.

Most interesting is that there appears to be an unexplainable adaptive response to longer exposures of  $\text{H}_2\text{SO}_4$  aerosols in adolescent asthmatics. Further work by Koenig (1992) and confirmed by Morrow et al. (1994), found greater decrements in  $\text{FEV}_1$  when exposed to the same levels of  $\text{H}_2\text{SO}_4$  aerosol for shorter time (35-45 minutes) periods than for longer time periods (60-90 minutes). Additionally, in healthy non-asthmatic adolescents with allergies, an exposure to  $\text{SO}_2$  with NaCl aerosol (similar to Vog) resulted in an 18% decrease in  $\text{FEV}_1$  (Koenig, 2000).

In contrast to adolescents with asthma, is the human response to  $\text{H}_2\text{SO}_4$  aerosol in the elderly. Koenig et al. (1993) found that exercising elderly subjects (60-75 years) with asthma had similar pulmonary function changes as those in healthy non-asthmatic elderly subjects, neither of which showed significant changes from baseline after being exposed to  $70 \mu\text{g}/\text{m}^3$  sulfuric acid. It should be noted that the previous study used a small sample size. More work on the human response to  $\text{H}_2\text{SO}_4$  aerosol in the elderly population is warranted.

### *Epidemiological and community studies of exposure to sulfates*

In addition to the literature on experimental studies, Ostro et al. (1991) demonstrated in an epidemiological study the association of airborne acid aerosols with increased respiratory symptoms in asthmatics. In a panel of 207 asthmatics in Denver, Colorado, airborne acid aerosols (including sulfates) were found to be significantly associated with several indicators of asthma.

Dockery et al. (1996) conducted a large study involving 24 communities in the USA and Canada to determine whether long-term exposure to acid aerosols was associated with respiratory symptoms in a population of children. The 24 cities included: 11 cities in the sulfate belt of the Eastern US, six cities in the transport belt, three cities in the Simi Valley of California, and four control cities. Subjects were comparable Caucasian children from 8 to 12 years of age ( $N = 13,369$ ). Human response data were collected by a parental questionnaire. With control for potential confounders, these data suggested that children living in a community of high levels of strong acidic particles had

66% higher odds of reporting bronchitis than those from a low pollution area (OR = 1.66; 95% CI = 1.11, 2.48). In addition, these subjects reported more bronchitis with high levels of fine particle sulfate (OR = 1.65; 95% CI = 1.12, 2.42). Reports of asthma, a persistent wheeze, chronic cough or phlegm, however, were not associated with higher levels of aerosols. These results were consistent with the Harvard Six-Cities Study (Damokosh et al., 1993) findings, which noted higher rates for bronchitis but not for asthma or wheeze in association with total suspended particles, PM <15 m in size, or strong acidic aerosols.

In addition, Raizenne et al. (1996) collected pulmonary function data on 10,251 subjects from the 24 communities concurrently with Dockery et al. (1996). Significant findings included an association between acid aerosol exposure and a decrement in FEV<sub>1</sub> (OR = 3.1%, 95% C.I.: 1.6, 4.6), FEV (OR = 3.5%, 95% C.I.: 2.0, 4.9) and a relative odds for low lung function (OR = 2.5, 95% C.I.: 1.8, 3.6).

Moreover, in a Canadian cohort study, an ambient sulfate level of 6.6 µg/m<sup>3</sup> was associated with decrements in pediatric lung function (Stern et al., 1989). Thurston et al. (1997) proposed that a sulfate threshold in humans likely exists <5 µg/m<sup>3</sup>. These findings suggest that long-term exposure to ambient particles of a strong acidic nature negatively affects pediatric lung growth, function and development.

### Health effects from a combined exposure to SO<sub>2</sub>, sulfate aerosols and PM

The literature continues to support evidence that particulate matter (PM) air pollution, specifically particles smaller than 10 µm, is associated with adverse human health effects: exacerbation of asthma, chronic obstructive pulmonary disease (COPD), cardiopulmonary disease, and mortality from myocardial infarction (MI) and cerebrovascular accidents (CVA) (Pope, 2000; Holgate et al., 1999; Pope & Dockery, 1999; Thurston, 1996). What can an exposure of 50 µg/m<sup>3</sup> of PM<sub>10</sub> do to human populations? In a review of the literature by Godish (1997) human health effects include: 1) 30-40% higher relative risk of mortality in the elderly and respiratory compromised individuals; 2) 6-25% increase in hospital admissions for pneumonia and COPD exacerbations; and 3) short term pulmonary peak-flow decrements of 30-40 ml/sec.

There continues to be debate over the size of particles ( $PM_{10}$ ,  $PM_{2.5}$ , and ultra fine) and their relationship to human disease. Early studies used measurements of total suspended particles (TSP) or  $PM_{10}$ ; however, recent studies now focus on the relatively smaller  $PM_{2.5}$  and ultra fine particles. Schwartz and Neas (2000) reanalyzed three longitudinal diary studies on lung function effects and reported symptoms in school-aged children exposed to PM. Findings revealed that  $PM_{2.5}$ , especially sulfate particles, had stronger respiratory effects on the children than coarse  $PM_{10}$  (Schwartz & Neas, 2000).

### *Epidemiological and community studies of mixed pollutants*

Historical events provide a supportive basis for modern hypotheses. London's continued smog episodes suggest that elevated levels of combined  $SO_2$ , PM, and  $H_2SO_4$  aerosol concentrations of  $400 \mu g/m^3$  are associated with excess mortality, and risk continues during non-smog episodes when  $H_2SO_4$  aerosol levels are  $\geq 30 \mu g/m^3$  (Godish, 1997).

Researchers conducting a large-scale prospective study over seven years, involving 550,000 subjects from 151 U.S. cities, found a 17% increase in mortality in subjects exposed to fine  $PM_{2.5}$  (Pope et al., 1995). There was an association to cardiac disease in subjects not dependent on smoking status; specifically sulfates in the  $PM_{2.5}$  were related to hospital admissions for cardiac disease (Pope et al., 1995). Sulfate compounds account for 40-50% of  $PM_{2.5}$  in urban areas of the U.S. (Godish, 1997).

Peters et al. (1997) found a significant increase in blood plasma viscosity in a random sample of men and women during a pollution episode of elevated  $SO_2$  and PM. Pope et al. (1999) found an increase in pulse rate, but no hypoxemia, in a daily time-series panel study of the elderly. Further studies, using 24-hour ambulatory cardiac monitoring, showed positive associations of heart rate increase with ambient  $PM_{10}$  increases.  $PM_{10}$  pollutant events were associated with changes in the heart rate variability as measured by rhythms and EKG. Results showed a lower autonomic cardiac control and a decrease in normal cardiac variability in exposed subjects. (Pope et al., 1999). Peters et al. (1999) noted increased electrocardiogram-measured hearts rates in 25-64 year olds and a faster rate in females during pollution episodes with  $SO_2$  and TSP. An increase of  $70 \mu g/m^3$  of  $SO_2$  was associated with an increase in heart rate of 1.75

beats/minute (Peters et al., 1999). In addition, elevated systolic BP has been found to be associated with exposure to SO<sub>2</sub> and TSP in mixed pollutant environments (Ibald-Mulli et al., 2001; Linn et al., 2001).

Recent hypotheses focus on the relationship between PM and cardio-circulatory diseases suggesting that PM induces cytokine responses, increased viscosity of the blood, and progression of atherosclerosis, all of which can be precursors of acute cardiac events (Suwa, 2002; Pope, 2000). In 1996, Thurston suggested that PM<sub>10</sub> studies confirmed an acute pollution-mortality association at routine ambient air levels, especially in susceptible populations. Pope et al. (2004) recently identified small but significant relationships between long-term exposure of fine PM<sub>2.5</sub> and cardiac mortality from ischemic heart disease, dysrhythmias, heart failure, and MI.

It should be noted that PM in volcanic settings has varying constituents, some of which are sulfurous or volcanic ash, and will vary geographically. Furthermore, pathogenic effects can be related to gases or to substances that adsorb onto the surface area of particles (Godish, 1997). The unmet challenge remains distinguishing the culprits and the interactions at play in the etiology of human disease.

In the 1980 eruption of Mt. St. Helens, air samples from Washington state peaked at 30,000 µg/m<sup>3</sup> TSP (90% sized PM<sub>10</sub>); containing mostly silica (Anderson, 1999). Baxter (1981) investigated related health effects and found a four-fold increase in ER admissions for asthma and a two-fold increase for bronchitis during the volcanic air pollution episode.

A study conducted in Japan at Mt. Sakurajima examined the association of neonatal mortality and ambient SO<sub>2</sub> levels between years 1978-1988 (Shinkura et al., 1999). Mt. Sakurajima is a frequently erupting volcano that spews both ash fall and gases on local urban populations. Researchers found the monthly average of SO<sub>2</sub> was positively associated with neonatal mortality ( $p = 0.002$ ) using a Poisson regression model. When relative risks of four groups were compared, using the lowest exposure group as a control comparison, the highest exposure group had a 120% (CI 95%: 1.2, 4.1) increased risk for neonatal mortality. Individual variables not showing an association to neonatal mortality included: number of eruptions, amount of ash fall, or suspended PM. A limitation of this

study is that the source of SO<sub>2</sub> (volcanic versus anthropogenic) could not be clearly determined. Further research at this urban-volcanic site is warranted (Shinkura et al., 1999).

### *Air pollution standards and recommendations*

The present body of knowledge suggests that certain sensitive populations are potentially at risk in communities where SO<sub>2</sub> levels are at current EPA NAAQS levels (1990). Numerous studies have demonstrated that asthmatics (pediatric and adult) are more sensitive to develop respiratory responses at lower levels of SO<sub>2</sub> than non-asthmatic individuals (Koenig, 1980; Holgate et al., 1999). WHO (1997) suggests that asthmatic individuals should not be exposed to levels of 175 ppbv of SO<sub>2</sub> for more than 10 minutes.

There is emerging data, however, that low levels of SO<sub>2</sub> in combination with PM, is pathogenic for all human lungs (Godish, 1997; Schwela, 2000). There are known respiratory responses in sensitive groups exposed to 250 µg/m<sup>3</sup> of SO<sub>2</sub> in the presence of PM, and a lowest-observed-adverse-effect level (LOAEL) at 100 µg/m<sup>3</sup> of SO<sub>2</sub> in the presence of PM (WHO, 1999). Therefore, WHO considered the interactive effects of SO<sub>2</sub> and PM. Hence, the acceptable level of SO<sub>2</sub> (125 µg/m<sup>3</sup> 24 hour average; 50 µg/m<sup>3</sup> annual mean) was determined after applying an uncertainty factor of two to the LOAEL (WHO, 1999) (Table 1.2).

In the weight of emerging evidence, EPA revised the National Ambient Air Quality Standards (NAAQS) for PM in 1997 to more conservative values and regards PM exposure as having non-threshold health effects. In addition, WHO (1999) considers that available information about PM pathology does not allow a judgment to be made of concentrations below which no effects would be expected. Current experimental studies are now focusing on health effects from fine and ultra fine PM, especially with diesel components.

### **Research on Volcanic Health Effects conducted in Hawai'i**

According to the Hawai'i State Department of Health (1997) respiratory disease is a common and frequently occurring disease in Hawai'i. The most recent prevalence of asthma in adults was 6.9% for the state of Hawai'i and 7.9% for the Big Island (Hawaii



BRFSS, 2002). Hawaiians had the highest adult asthma prevalence at 11.5%, followed by Filipinos (11.0%), Caucasians (7.9%) and Japanese (3.7%) on the Big Island (Hawaii BRFSS, 2002). In the districts of Puna and Kau that surrounds the volcano, the combined prevalence of asthma in adults was 9.0%, and a higher 16.2 % in children (Hawaii BRFSS, 2002). The Kona District had adult and pediatric asthma prevalence, measured independently, at 9.4% (Hawaii BRFSS, 2002).

After only a few years into the eruption, Hallenborg et al. (1991) expressed that many local physicians and patients believed the increase in mortality and morbidity of asthma was due to the volcano. Data from existing human health studies support a hypothesis of negative health outcomes from Kilauea's present eruption. The body of knowledge, however, is severely limited in the quantity of data, the populations studied, and the research methods employed.

### Hydrogen sulfide air pollution

At the start of the current eruption in 1983, there was public concern over volcanic emissions generated from man-made geothermal wells (Figure 1.1). Since 1976, local residents had been complaining of adverse health effects from being exposed to the emissions, primarily hydrogen sulfide ( $H_2S$ ) (Table 1.2). In 1984, a cross-sectional survey was conducted in neighborhoods near six geothermal stations located on the lower East Rift of Kilauea (the POK eruption is uphill from this site) (Anderson and Oyama, 1987). A door-to-door health survey was conducted on an exposed population, capturing 88% of residents ( $n = 350$ ) and 93.2% of a nearby control population ( $n = 604$ ). No significant differences in prevalence were found. In addition, no correlations were found between ambient  $H_2S$  data and prevalence data. A notable limitation of this study was the extremely close geographic proximity of the control population. There was higher prevalence in various health illnesses however, when compared to prevalence measures for the entire Big Island and state. After adjustment for age, prevalence of the common cold in the exposed population (14.3%) was over twice the prevalence of the county (6.4%) and the state (7.4) (Anderson et al., 1987). In addition, there were significantly higher prevalence ratios of the following: upper respiratory conditions, digestive disorders, diseases of the eyes/ears, non-allergic skin diseases, malignant neoplasms,

nervousness or depression, hay fever, and sinusitis. There was not an increased prevalence of asthma.

In early 1990, the survey was repeated in the exposed community and a more distal control community (Roper, 1992). These data revealed higher prevalence for the exposed community when compared to the 1984 data and again higher prevalence when compared to county and state measures. Within the exposed community from 1984-90, prevalence of bronchitis and emphysema doubled, sinusitis rose from 4.9% to 14%, and asthma increased from 4.0% to 5.0% (Roper, 1992). All these prevalence values were above county and state prevalence measures (Roper, 1992).

### Air pollution from Kilauea's eruption

A preliminary retrospective study was conducted by the Centers for Disease Control (CDC) at the start of the current Kilauea eruption to assess respiratory hazards associated with the volcanic emissions (Bernstein et al., 1984). Data were collected from outpatient and admitting departments at Hilo and Kona hospitals for a time period of two weeks prior to eruption thru two weeks into eruption. Diagnostic criteria were asthma, bronchospasm, or acute/chronic bronchitis. Volcanic gas emissions ( $\text{SO}_2$  and particles) were obtained from Hilo, Kona and HVNP. No increases in respiratory diagnoses due to increased volcanic emissions were detected in this study. Authors reported that these data were inadequate to assess the research objective (Bernstein et al., 1984).

Another study conducted for the CDC by Mannino et al. (1996), demonstrated that volcanic emissions might have affected the respiratory health of Big Island residents. Data were collected retrospectively from the medical records of four hospitals: Hilo, Kona, Kau and Kohala. Geographically, Kohala is the only hospital that could be a control for this study, virtually Vog-free. Emergency room (ER) admissions were reviewed from 1981 to 1991 for the diagnoses of asthma or chronic obstructive pulmonary disease (COPD). Total sample size was 12,539 with nearly equal numbers of male and female subjects. Findings revealed outcome rates for ER subjects with asthma or COPD: 80% discharged home; 15.6 % admitted; 3.6% ICU admitted; 0.3% transferred; 0.1% died. Inevitably, the Hilo and Kona hospitals demonstrated significantly higher rates of ER admissions ( $p < .05$ ). For the Hilo hospital alone, the

mean weekly visits for asthma rose from 11.4 in 1981 to 15.2 in 1991 (adjusted for population increase). Furthermore, these weekly ER visits were positively correlated to weekly percentages of hourly westerly winds ( $r = .21$ ,  $p = .002$ ) and low temperatures ( $r = .20$ ,  $p = .003$ ). Seasonal variations were observed in these data with higher rates in winter as compared to summer. Limitations of this study included: 1) a possible diagnostic bias in the ER records; 2) an absence of actual air quality data; 3) no notable step in the data that would reflect the volcanoes activity of intermittent eruptions (1983-1986) to continuous eruption in 1986 through 1991. Researchers recommended that future studies include measurements of volcanic emission levels to correlate with occurrences of respiratory illness (Mannino et al., 1996).

Chen (2000) re-analyzed these data using re-calibrated meteorological data. The new meteorological data were compared to data from 1981-1991 for Hilo Hospital ER admissions for respiratory distress complaints. There were no strong relationships found for wind conditions and ER admissions. There continued to be a seasonal relationship to these data, and moderate relationships between ER visits and winds coming from both Kilauea and the northern Hamakua coast (a non-volcanic area with high rainfall, abundant vegetation and mold growth).

Smith (1998; 1999) and the Hawai'i Preparatory Academy (HPA) conducted two studies into the health effects of Vog. The initial study focused on health effects in 325 students over 16 months. Total particle measurements were collected at HPA three times daily by Gardner counters. Upper respiratory health data was obtained from school health records. No significant correlations were found (Smith, 1998). HPA is located in Waimea, a northern area of the island not regularly exposed to Vog.

The second study again attempted to correlate human health effects and Vog by using regional air data and health care visits in the general population (Smith, 1999). Data were collected from over three years time at Waimea, Kona, and Kau. Total particulates were again measured by Gardner counters at the three locations. Respiratory health data were collected from medical records. Analysis showed no correlations of health data to particle concentrations at the research sites. The Kau data showed the highest number of particles ranging from 90/cc to 330,000/cc (Smith, 1999). Significant limitations to these

studies occurred. Air data collected from these sites were inconsistent. Health data were not limited to specific diagnoses at all locations. Therefore, significant bias was introduced. All healthcare sites were not included. Nonetheless, these two studies identified the Kau area as a site of high particle exposure.

The Vog-net project in the high schools around the island continues to date. There is a sampling site at Pahala High School in Kau district. These data suggest that particle concentrations change over the 24-hour period, with the highest particle concentrations in the early sunlight morning hours. It has been suggested that the inversion lowers at night, thereby concentrating the air mixing, and rises during the day (Steve Ryan, NOAA, email communication on April 11, 2003).

Michaud et al. (2000) conducted a pilot longitudinal panel study to investigate possible associations between Vog and acute respiratory effects in 67 asthmatic grade school children. Hilo, Hawai'i, was chosen as the study site because the geographical area was prone to variable Vog occurrences and meteoroidal equipment was in place. Asthmatic subjects were selected to investigate repeated acute pulmonary insults on development of chronic pulmonary disease. Subjects were from grades 4<sup>th</sup> through 6<sup>th</sup>, ages that have the ability to perform effective spirometry and still less likely to smoke. Dependent variables of SO<sub>2</sub> and sub-micron aerosol particulate matter were collected. Numerous health variables were collected: subject diary entries, subject-performed peak flow meter readings, use of bronchodilator medication, parental questionnaire, and periodically collected spirometry data. Data collection, however, failed to capture health data concurrent to actual Vog occurrences. Only two Vog episodes occurred during the three-month data collection period of March-May, 1999.

Notwithstanding, no statistically significant relationships were found using multiple regression techniques. Descriptive analysis revealed a profile of common asthma symptomology in subjects (coughing, wheezing, medication use). The study did identify possible confounders for future investigations with asthmatics (burning of yard material, or large scale burning of fields, and construction activity).

Michaud et al. (2004) found positive associations ( $p < 0.01$ ) for ER visits in Hilo from 1997 through 2001 for asthma/COPD with Vog-contaminated air. ER visits with a

3-day lag rose 6.8% for every 10 ppbv increase in SO<sub>2</sub>, and rose 13.8% (1-day lag) per 10 µg/m<sup>3</sup> increase in PM<sub>1.0</sub> (Michaud et al., 2004). Despite these significant findings, stronger associations were found between ER visits and the month of the year, suggesting other influential variables to these data. Due to the minimal occurrences of Kona winds, and therefore minimal occurrences of human exposure to Vog, these studies demonstrate that Hilo is unsuitable for future studies that focus on chronic pathologies or chronic exposure to volcanic air pollution.

Lastly, a study led by the University of Hawai'i-Manoa is underway investigating the longitudinal effects of Vog on pediatric lung development. Various elementary school sites on the Big Island, including Pahala and Hawi, are being used to follow pediatric health prospectively for 3 years since 2002. Environmental sampling is being conducted periodically for SO<sub>2</sub> and PM. No data has been released to date (December 2004).

Throughout the 22-year eruption of Kilauea Volcano, the Hawai'i Department of Public Health did not separate the Kau district (downwind exposure during usual trade wind patterns) from the Puna district (upwind from the volcano) in bi-annual epidemiology surveys or health surveillance. Therefore, there were no available historical or current health data on the Kau population for use in this study. Currently, there is an absence of specific health monitoring for adverse effects from volcanic air pollution. Air monitors are at Hilo, Kona, and the summit of Kilauea (upwind of the summit degassing). In summary, the work conducted in Hawai'i prior to this study focused on measuring air quality in the urban population centers of the island, exploring acute reactions to volcanogenic air pollution, specifically with pediatric asthmatics, and measuring acute illness cases (ER visits) of respiratory disease.

**Table 1.1. Description of Extrusive Volcanic Rocks**  
(Walker, 1973; Wright et al., 1980; Cas & Wright, 1988; Baxter, 1990; Press & Siever, 2002; Tarbuck & Lutgens, 2002)

Type of Volcanic ROCK	Composition & Viscosity rating	Gas content & Temperature	Vent Type & Explosivity	Tectonic Setting & Dominant Style of Volcanism	Human Health Hazards
<b>Rhyolite</b>	Felsic (70% silica)  <i>Highest viscosity, slow moving</i>	Most (4-6%) 800-1000°C	Multi-vent volcano-tectonic depressions, calderas, domes, pumice cones, tuff rings.  <i>Highly explosive</i>	Extensional tectonics, convergent plate margins, intracontinental hot spots, rifts. <i>Styles: Ultraplinian, phreatoplinian</i>	Explosive ejecta, High-altitude ash columns for air travel, Regional ash fall, lahars and mud flows, Pyroclastic (hot ash) flows
<b>Dacite</b>	Intermediate (65% silica)  <i>Intermediate viscosity</i>		Mature strato-volcanoes domes, tuff rings, maars <i>Explosive to effusive</i>	Convergent plate margins <i>Styles: Plinian, vulcanian</i>	
<b>Andesite</b>	Intermediate (60% silica) <i>Intermediate viscosity</i>		Strato-volcanoes <i>Effusive to explosive</i>	Convergent plate margins <i>Styles: Plinian, vulcanian</i>	
<b>Basalt</b>	Mafic (50% silica)  <i>Low viscosity, fast flowing</i>	Least (1-2%) 1000-1200°C	Shield volcanoes, scoria cones, maars, tuff rings & cones, axial & composite volcanoes. <i>Mostly Effusive with some explosive (surtseyan, fire fountains)</i>	Mid-ocean ridges, Hot spots, mid-continental rifts. <i>Styles: Hawaiian, Strombolian,</i>	Gas releases, Lava flows, Phreato-magmatic eruptions, Localized ash
<b>Komatite</b>	Ultramafic <i>Lowest viscosity</i>		<i>Effusive</i>	Pre-Cambrian	Pre-hominoid

Table 1.2. Description of Air Pollutants and the Associated Human Health Effects.  
 [U.S. EPA National Ambient Air Quality Standards (NAAQS), 1990, 1997; WHO, Air Quality Guidelines, 1999; Godish, 1997; EPA IRIS database; California OEHHA, 2002; Agency for Toxic Substances & Disease Registry (ATSDR), 1998]

Compound	Description	Acceptable Limits	Health effects from compound
<b>Sulfur dioxide (SO<sub>2</sub>)</b>	Colorless gas, taste/odor detected at 0.38-1.15 ppmv, pungent irritant odor >3.0 ppmv.  <u>Background level</u> in remote areas: <5 ppbv. (0.005 ppmv)	<b>U.S. EPA standards:</b> <u>annual arithmetic mean:</u> 0.03 ppmv (80 µg/m <sup>3</sup> ), 30 ppbv <u>24-hour average:</u> 0.14 ppmv (365 µg/m <sup>3</sup> ), 140 ppbv <b>WHO recommendations:</b> <u>annual arithmetic mean:</u> (50 µg/m <sup>3</sup> ) <u>24-hour average:</u> (125 µg/m <sup>3</sup> ) <b>ATSDR:</b> Minimal risk level: 10 ppbv, or 27 µg/m <sup>3</sup> , acute exposure of <14 days	Upper respiratory tract (URT) and eye irritant, bronchoconstriction, respiratory tract inflammation (chronic rhinitis & pharyngitis), cough, bronchitis, exacerbation of existing asthma and restrictive diseases, neonatal mortality
<b>Hydrogen sulfide (H<sub>2</sub>S)</b>	Colorless gas, taste, the odor threshold is 25 ppbv (0.035 mg/m <sup>3</sup> ) <u>Background level:</u> 30-100 pptv.	<b>EPA standard:</b> NOAEL: 42.5 mg/m <sup>3</sup> (30.5 ppmv) (IRIS, 07/01/1995) <b>WHO:</b> <u>24-hour average:</u> 0.15 mg/m <sup>3</sup>	Asphyxiant, URT irritant, bronchitis, asthma, pulmonary edema, death from acute respiratory distress syndrome due to pulmonary edema
<b>Sulfuric acid (H<sub>2</sub>SO<sub>4</sub>)</b>	Colorless aerosol, oily, burning irritant	<b>EPA:</b> Not available <b>WHO:</b> Not available <b>California:</b> Chronic reference exposure level (1-year) (REL) = 1 µg/m <sup>3</sup>	Skin, eye and URT irritant; decrease pulmonary functioning; decreases mucociliary transport, alveolar clearance, and alveolar phagocytosis; restrictive pulmonary diseases, chronic bronchitis, asthma exacerbation
<b>Particulate matter PM<sub>10</sub></b> coarse basic pH Diameter of ≤10 µm <i>Inhalation range</i>	Colorful, mass, solid or liquid forms, suspended or deposited	<b>EPA:</b> <u>annual arithmetic mean:</u> 50 µg/m <sup>3</sup> <u>24-hour average:</u> 150 µg/m <sup>3</sup> <b>WHO:</b> no judgment is made of concentrations below which no effects would be expected.	Restrictive pulmonary diseases (COPD), decrease pulmonary functioning, chronic bronchitis, pneumonia, exacerbation of existing asthma
<b>Particulate matter PM<sub>2.5</sub></b> fine acidic pH Diameter of <2.5 µm <i>Respirable range</i>	Colorful, mass, solid or liquid forms, suspended or deposited. <u>Background levels:</u> remote areas: 1-2 µg/m <sup>3</sup> ; non-urban continental: <10 µg/m <sup>3</sup>	<b>EPA:</b> <u>annual arithmetic mean:</u> 15 µg/m <sup>3</sup> <u>24-hour average:</u> 65 µg/m <sup>3</sup> <b>WHO:</b> no judgment is made of concentrations below which no effects would be expected.	Same as PM <sub>10</sub> plus: alveolar damage, cardiac disease, mortality from cerebral vascular accident, myocardial infarction or other cardiac diseases

**SO<sub>2</sub> AND FINE AEROSOL DISPERSION FROM THE KILAUEA  
PLUME, KAU DISTRICT, HAWAII, USA**

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## Abstract

Proximal ground-level assessment of sulfur dioxide gas and fine aerosol in the volcanic gas plume downwind from Kilauea volcano indicates high levels of both species. SO<sub>2</sub> and fine aerosol data were collected in the Kau district, from 37 to 74 km downwind from the eruption, and at a nonexposed control site in Hawi, Hawai'i. Typical trade winds and effusive eruption occurred during sampling in August–September 2003. Ambient SO<sub>2</sub> concentrations, measured with diffusion tubes, ranged from 6 to 34 ppbv and correlated negatively with altitude. In contrast, fine aerosol ( $\leq 0.3 \mu\text{m}$  size) concentrations, measured with a cascade impactor, ranged from 0.61 to 11.82  $\mu\text{g}/\text{m}^3$  and correlated positively with altitude. We attribute decrease of SO<sub>2</sub> with altitude to rapid oxidation as diurnal wind patterns blow the plume from oceanic terrain landward to more abundant oxidation sources. Aerosol increase with altitude likely reflects emission of H<sub>2</sub>SO<sub>4</sub> from Kilauea, supplemented by oxidation of SO<sub>2</sub> in atmospheric hydrosols. Kau residents are exposed to volcanogenic pollutants at concentrations that warrant concern for adverse health effects.

## Introduction

Effusive eruption at Kilauea volcano, Hawai'i, has persisted since 1986, releasing an average of 1600 tons of sulfur dioxide ( $\text{SO}_2$ ) per day into the troposphere (Sutton and Elias, 2002). Unlike Plinian eruptions that send particles and gases into the stratosphere, effusive Hawaiian eruptions release gases into the lower troposphere where humans reside. An estimated 500 million humans live near active volcanoes worldwide (Baxter et al., 1999). The World Health Organization (WHO, 2001) has called for action to assess regional and local air pollution and the associated burden of disease. At the basaltic shield volcano Masaya, Nicaragua, Delmelle et al. (2002) mapped an increase in  $\text{SO}_2$  in the gas plume with proximity to the vent, and concentrations were above WHO guidelines. Prevailing winds and local topography strongly affect plume dispersion. At Kilauea, little is known about the downwind dispersion of the plume's volcanic air pollutants or their effects on the human population. We present results from the first regional ground-level assessment of downwind air in the Kau district of the Big Island, Hawai'i (Figure 2.1), which was conducted as part of a study to measure chronic health effects.

## Setting

Kilauea volcano's ongoing Pu'u 'O'o-Kupaianaha East Rift eruption has been releasing a gaseous plume primarily composed of water,  $\text{SO}_2$ , and sulfate aerosols (Sutton et al., 2000). Degassing of  $\text{SO}_2$  also occurs at Kilauea's summit both during eruption and in periods of quiescence (USGS, 1998).

The gas plume travels from the vent with prevailing northeastern Pacific trade winds through the marine boundary layer that is capped by a temperature inversion at ~1800 m above sea level (Figure 2.1). Below the inversion, humidity is high (70%–80%) and well distributed (WRCC, 2002). The plume travels southwesterly over the ocean around sloping Mauna Loa volcano and over South Point and eventually reaches the Kona Coast. In the Kau district, the trade winds blow to the southwest in the mornings and move inland and upslope by midday, bringing the plume over populated areas. Orographic effects produce afternoon rains in the upslope areas (Giambelluca &

Schroeder, 1998). Nighttime downslope winds carry the plume back over the ocean. The terrain in Kau is bare lava flows mixed with forest and farming and grazing lands.

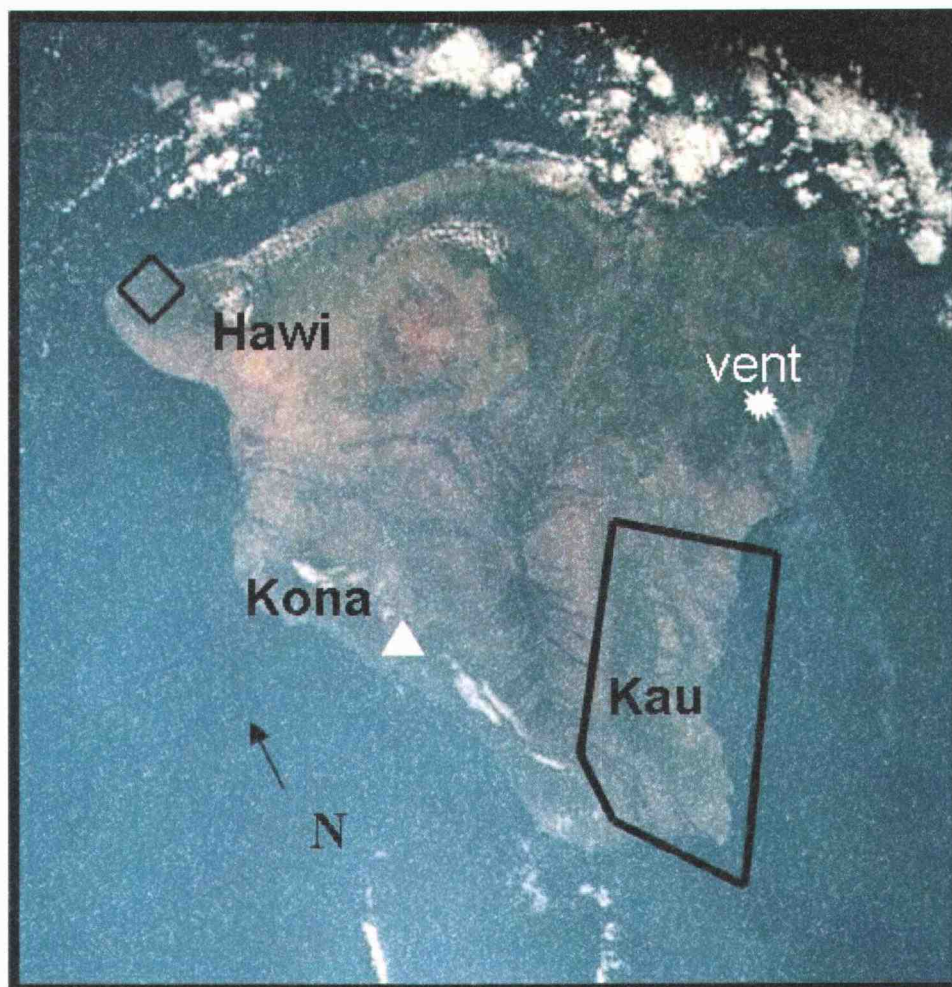


Figure 2.1 Location map of the Big Island ( $19^{\circ}30'N$ ,  $155^{\circ}30'W$ , 129 km wide east-west), Hawai'i, U.S.A. Exposed Kau cohort and unexposed Hawi cohort areas are outlined. The vent and plume are shown during trade-wind conditions. The white triangle marks the Kona air-monitor. Photograph credit: National Aeronautical and Space Administration, taken on STS-85 Space Shuttle mission August 1997.

Aircraft sampling of Kilauea's plume, using a laser optical counter, identified volcanic aerosol at  $\sim 0.3 \mu m$ ; background marine aerosol was much larger at  $2\text{--}3.0 \mu m$  (Clarke & Porter, 1991). Chuan (1995, 1997, 1998) characterized ground-based volcanic aerosols near the vent and at distal sites by using scanning electron microscopy and energy-dispersive X-ray analysis. Aqueous sulfuric acid aerosol ( $H_2SO_4$ ), with geometric

mean diameters of 0.3  $\mu\text{m}$  and 0.1  $\mu\text{m}$ , comprised >80% of the total aerosol mass, and lesser elemental sulfur and silicates were measured at the Pu'u 'O'o vent (Chuan, 1995). In the Kona Coast area, 130 km away, bimodal mass concentrations of volcanic aerosols peaked at 0.3  $\mu\text{m}$  ( $\text{H}_2\text{SO}_4$  and elemental sulfur) and at 1.7  $\mu\text{m}$  ( $\text{Na}_2\text{SO}_4$  and other sulfates), with background aerosols of NaCl (0.8  $\mu\text{m}$ ) and various silicates (>2  $\mu\text{m}$ ) (Chuan 1995, 1997, 1998). Aqueous HCl (0.8  $\mu\text{m}$ ) has been detected in Kona samples when lava-sea entry occurred (Chuan, 1995).  $\text{SO}_2$  levels, monitored by the State of Hawai'i at a fixed site on the Kona Coast (Figure 2.1), are low. The lack of information on air quality in the vast Kau district, between the volcano and Kona, motivated this investigation.

### **Sampling Methodology and Conditions**

$\text{SO}_2$  and fine volcanic aerosol data were collected during August and September 2003. We used a cohort model of exposed and unexposed areas with comparable geography and human populations. The exposed cohort lies 37–80 km downwind of the vent (Figure 2.1). The unexposed control cohort, Hawi, lies on the north side of the island. The sites have comparable rainfall, temperature, vegetation, and crops. Weather patterns during the three-week sampling period included typical Pacific trade winds ( $\leq 16$  km/h), with the exception of a 48-h-long tropical storm that brought rains and winds up to 38 km/h in the Kau district. Inland areas were subject to afternoon rain showers on most days.

Lava flows erupted continuously during the sampling period from the base of Pu'u 'O'o and tube breakouts. Lava did not enter the sea. Gas emissions for  $\text{SO}_2$ , by correlation spectrometer, averaged  $1173 \pm 335$  t/d (metric tons per day) at the eruption site and  $126 \pm 25$  t/d at the summit, in keeping with the recent 2 yr average of  $1440 \pm 470$  t/d and  $110 \pm 30$  t/d, respectively (USGS, unpublished HVO data from A.J. Sutton).

#### **Sulfur dioxide sampling**

$\text{SO}_2$  was measured with 70  $\text{SO}_2$ -specific, passive diffusion tubes, made by Harwell Scientifics (Table 2.1). These tubes are used in urban air pollution and volcanology studies (Delmelle et al., 2002; Baxter et al., 1999; Nicholson et al., 1996)

and are field verified for tropical climates. Cross sections of the vent plume and summit degassing paths were captured by stratified sampling, using five gradient lines along the flank of Mauna Loa (Figure 2.2A) and six random sites. Six outdoor tubes served the control cohort. Outdoor tubes were placed 1.5–2.5 m above the ground, away from contamination sources or busy roads; exposure time ranged from 19 to 23 days. The method cannot resolve temporal variation of exposure.

Indoor tubes ( $N = 11$ ) were placed in homes and facilities void of tobacco smoke, without air conditioning, and away from windows, doors, or stoves to measure indoor penetration of  $\text{SO}_2$  on sensitive populations, such as children and medical patients. Two tubes for accuracy were placed for 20 days next to  $\text{SO}_2$  pulse fluorescence monitors, and three sets of paired tubes in the cohorts were used to measure reliability ( $\pm 5.5\%$ ). Outside the cohorts, two tubes were deployed near-source for 90 hours to capture  $\text{SO}_2$  in the vent plume and summit path for mapping purposes.

### Fine aerosol sampling

Aerosol samples (Table 2.2) were collected using a quartz crystal microbalance cascade impactor (QCM) with four geometric mean-diameter size ranges:  $\geq 1.6 \mu\text{m}$ ,  $0.8 \mu\text{m}$ ,  $0.3 \mu\text{m}$ , and  $0.1 \mu\text{m}$  (Chuan, 1995, 1997). Samples were collected at 1.5 m above ground for between 10 and 30 min; the QCM provides particle-size distribution and mass concentration of each size fraction with a precision of  $\pm 1 \mu\text{g}/\text{m}^3$  within 5 min (Chuan, 1998). The method does not detect long-term exposure.

Concurrent with the  $\text{SO}_2$  study, 54 fine aerosol samples were collected in the exposed cohort and 4 samples from the control cohort to use for background adjustment of exposed samples. To assess for a temporal pattern of plume migration with trade winds, samples were collected every 2 h for a 24 h period in the town of Pahala. During light variable wind conditions, 14 additional samples were collected in the exposed area.

## Results

Both  $\text{SO}_2$  and fine aerosol concentrations were elevated relative to background in the exposed cohort. Ambient  $\text{SO}_2$  average concentration in the Kau district (17.8 ppbv,  $45.2 \mu\text{g}/\text{m}^3$ ) was 25 times that in control Hawi (0.7 ppbv,  $1.8 \mu\text{g}/\text{m}^3$ ) and ranged from 6



ppbv at Ocean View to 34 ppbv in the Na'alehu area (Table 2.1, Figure 2.2A). Aerosol findings reveal that adjusted concentrations of particles of  $\leq 0.3 \mu\text{m}$  size ranged from 2 to 40 times greater in the Kau district ( $0.61\text{--}11.82 \mu\text{g}/\text{m}^3$ ; Figure 2.2B) than in control Hawi ( $0.31 \mu\text{g}/\text{m}^3$ , Table 2.2). During variable winds, relatively higher concentrations of particles of  $\leq 0.3 \mu\text{m}$  size were measured in Kau (Table 2.2).

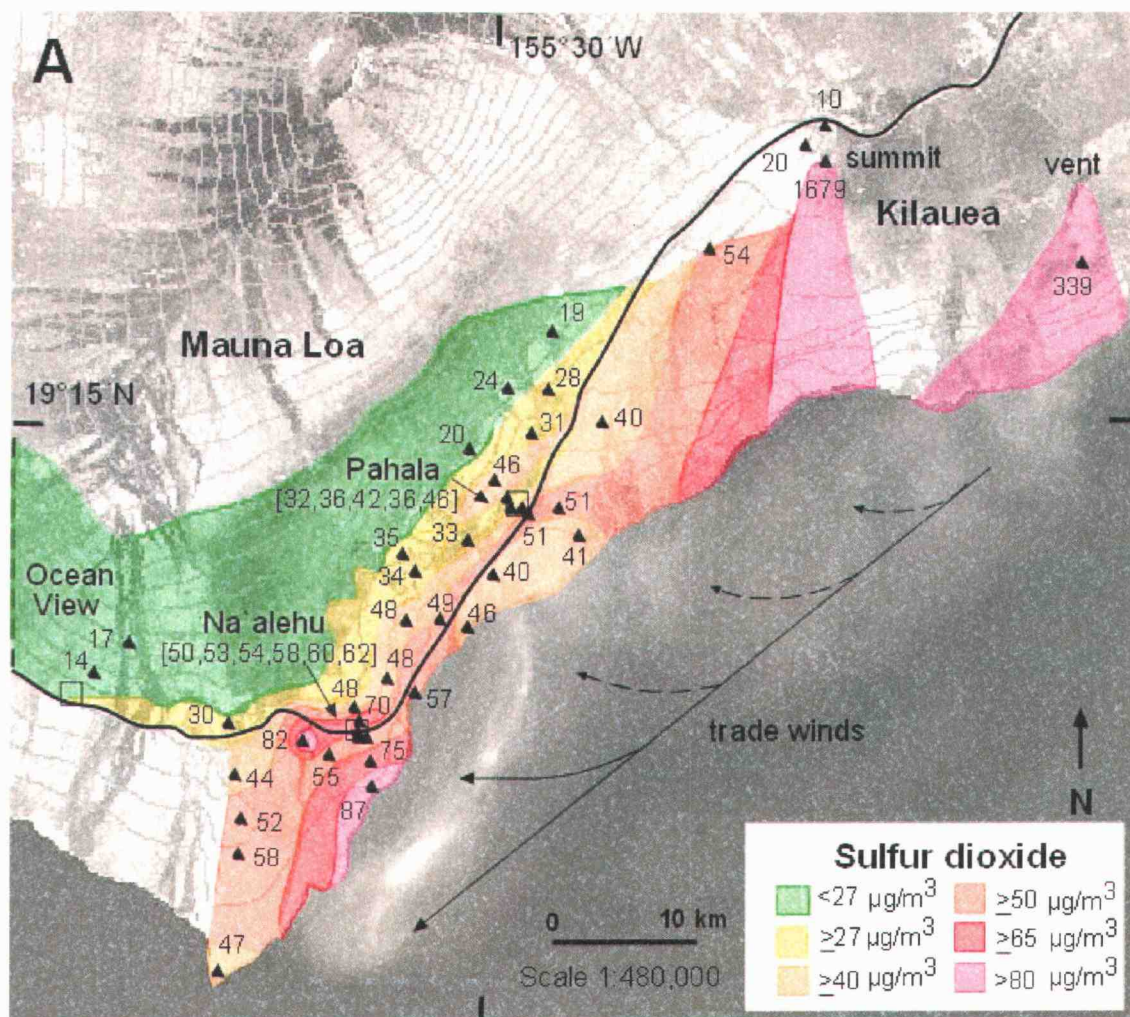


Figure 2.2A. Atmospheric  $\text{SO}_2$  concentration shown via contour map of Kau District, Hawai'i, U.S.A. Colors and specific mass concentrations (in  $\mu\text{g}/\text{m}^3$ ) are based on three-week exposure measurements taken in August–September 2003. Black triangles represent outdoor sample sites with corresponding concentration value. Base map uses elevation markers in 500 foot (152 m) intervals, provided by State of Hawai'i GIS (Geographic Information System) Program I-Map.

The concentration of SO<sub>2</sub> strongly decreases with altitude throughout the exposed cohort area ( $n = 43$ ; Figure 2.2A) with and without accounting for distance from source (partial correlation  $r_{12,3} = -0.73$  and  $r = -0.70$ ,  $p < 0.001$ , respectively). We considered instrument effects on SO<sub>2</sub> concentrations and used diffusion-coefficient corrections (up to -10% at highest altitude) and ppbv standardization for altitude (+2% to 4%); even for corrected data, altitude and concentration of SO<sub>2</sub> remained highly correlated. A high SO<sub>2</sub> measurement in the Na`alehu area (32 ppbv) occurs at a sharp topographic gradient, suggesting topographically induced stagnation of the plume (Figure 2.2A). Comparison of indoor and outdoor SO<sub>2</sub> testing yielded indoor/outdoor ratios ranging from 0.15 to 0.71, with a high value of 16.8 ppbv (Table 2.3). Locations with amounts >10 ppbv included the community hospital, grade school, and two residences of asthmatic individuals.

Volcanic aerosol has a pattern of peak concentrations at 0.3 and 0.1  $\mu\text{m}$ ; background aerosol has decreasing concentrations with decreasing size, as also noted by Chuan (1997, 1998). During trade winds ( $n = 52$ ), concentration of aerosols ( $\leq 0.3 \mu\text{m}$ ) and altitude were positively correlated,  $r = +0.69$  ( $p < 0.01$ ), even with accounting for distance,  $r_{12,3} = +0.65$  ( $p < 0.001$ ). Pahala was subject to an afternoon peak in  $\leq 0.3 \mu\text{m}$  particles; the diurnal pattern of volcanic fine aerosol followed the trade-wind pattern for the windward slope of the Big Island (National Weather Service, 2003). Distal Ocean View had the highest concentrations of fine aerosols during dry and wet conditions, morning and afternoons, as well as during trade and variable wind periods. Aerosol sampling along coastal areas detected no volcanic aerosols during trade winds, similar to Chuan's finding in 1997.



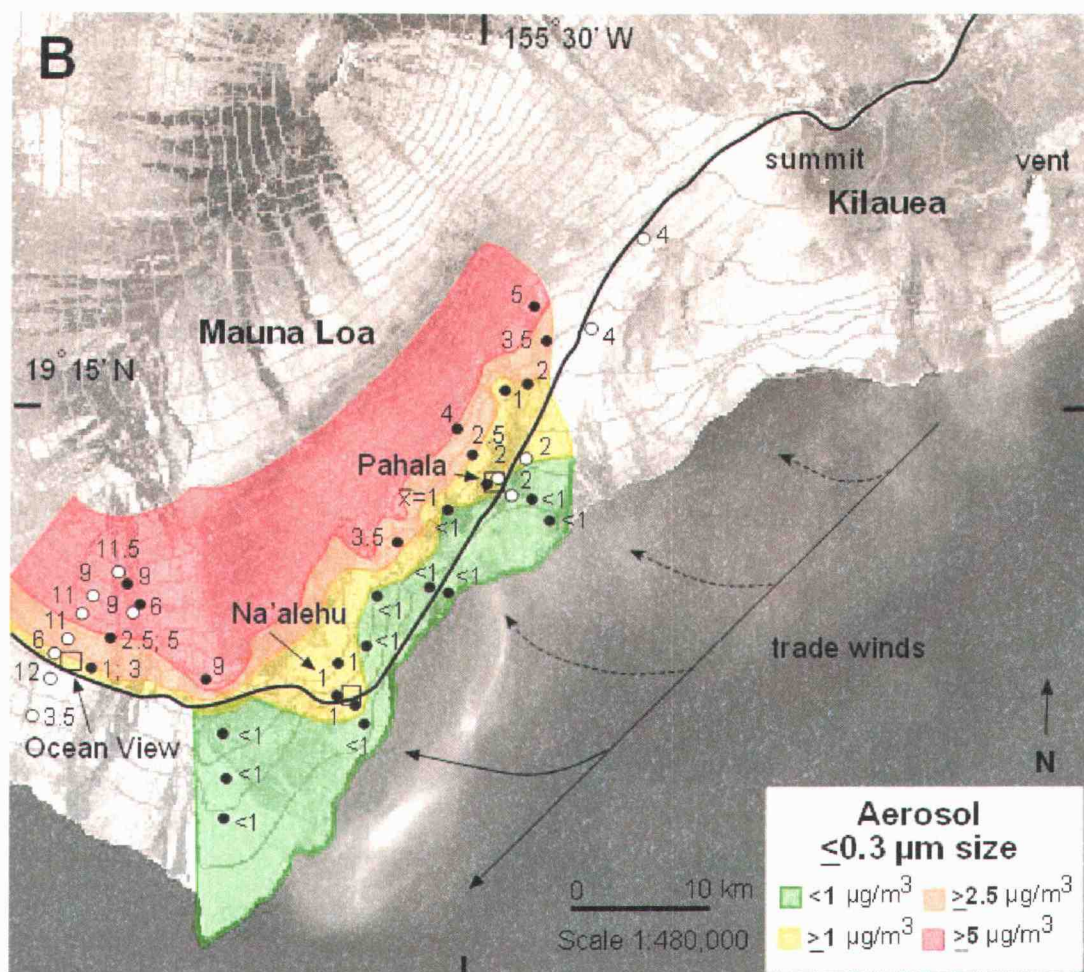


Figure 2.2B. Atmospheric fine aerosol ( $\leq 0.3 \mu\text{m}$ ) concentration shown via contour map of Kau District, Hawai'i, U.S.A. Colors and specific concentrations (in  $\mu\text{g}/\text{m}^3$ ) are based on grab sample measurements taken in September 2003. Black circles denote samples collected during trade-wind conditions. White circles denote samples during variable winds. Base map uses elevation markers in 500 foot (152 m) intervals, provided by State of Hawaii GIS Program I-Map.

## Discussion

The results of  $\text{SO}_2$  and fine aerosol measurements in the Kau district, downwind of Kilauea volcano, indicate that the area and population are exposed to volcanic air pollution. We first address the applicability of the study, the implications on human health, and finally the altitudinal variations of these data.

### Applicability of the study

Sampling was done during conditions representative of typical weather and volcanic activity at Kilauea, thereby laying the groundwork for public health



investigation. Assessment of the  $\text{SO}_2$  concentrations is conservative, because there was rainfall, which scrubs  $\text{SO}_2$  (e.g., Poas volcano, Nicholson et al., 1996), and no explosive lava fountaining occurred, which would increase  $\text{SO}_2$  emissions. Similarly, aerosol assessment is conservative because we emphasize the exposure burden of fine aerosols ( $\leq 0.3 \mu\text{m}$ ) and not total exposure of particulate matter ( $< 2.5 \mu\text{m}$ ,  $\text{PM}_{2.5}$ ). Ambient air likely also carries larger volcanic sulfates, between  $0.8 \mu\text{m}$  and  $2.5 \mu\text{m}$  (Chuan, 1997, 1998), thereby further exposing the population. High humidity at lower elevations may also hygroscopically enlarge fine aerosols beyond the range of the QCM instrument.

### Air quality and health

Sulfur dioxide is a known respiratory irritant to sensitive populations (e.g., people with respiratory disease, asthma, or advanced in age; WHO, 1979, 2000). Sulfate aerosols, however, can negatively affect both sensitive and general populations by inducing respiratory tract irritation that can alter lung defenses, such as airway reactivity and mucociliary transport (Koenig, 2000; Holgate et al., 1999).

The two decades of eruption at Kilauea exceed the seven-year time frame used to define a chronic exposure. Comparisons to long-term exposure standards are justified, because typical exposure conditions prevailed during our study. Only two samples in the Na`alehu area exceeded the U.S. Environmental Protection Agency (EPA, 1990) annual standard for  $\text{SO}_2$  ( $80 \mu\text{g}/\text{m}^3$ ), but using the WHO annual guideline for  $\text{SO}_2$  of  $50 \mu\text{g}/\text{m}^3$  (WHO, 2000), the Na`alehu area and most coastal areas are at or above the guideline (Figure 2.2). The summit tube placed 0.5 m above the ground at a common visitor's site recorded  $1679 \mu\text{g}/\text{m}^3 \pm 100 \mu\text{g}/\text{m}^3$ , well above the WHO 10 minute  $\text{SO}_2$  guideline of  $500 \mu\text{g}/\text{m}^3$  (WHO, 2000). Although specific for  $\text{SO}_2$ ,  $\text{H}_2\text{S}$  gas may have contaminated the tube. Further work is warranted because small children may be most at risk.

Many populated areas in Kau, even indoors, are above the acute minimal risk level (MRL) for  $\text{SO}_2$  (Fig. 2.2). The  $\text{SO}_2$  MRL is 10 ppbv, or  $\sim 27 \mu\text{g}/\text{m}^3$ , set by the U.S. Agency for Toxic Substances and Disease Registry (ATSDR, 1998) for an acute exposure of  $< 14$  days, a time frame covered by these data. An MRL is a public health guideline, set for sensitive members of the population, that estimates the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse

health effects over a specific time period. Exposure to concentration levels above an MRL do not necessarily mean that adverse health effects will occur.

In Ocean View, amounts of  $\leq 0.3 \mu\text{m}$  aerosol as high as  $9 \mu\text{g}/\text{m}^3$  in trade winds and  $11 \mu\text{g}/\text{m}^3$  in variable winds were recorded (Figure 2.3). Lioy and Waldman (1989) defined a sulfate aerosol pollution "event" as a concentration of  $\geq 5 \mu\text{g}/\text{m}^3$ . If  $\text{H}_2\text{SO}_4$ , at  $\leq 0.3 \mu\text{m}$ , is assumed to be the major aerosol component of these samples (Chuan, 1995, 1997, 1998), then higher-altitude downwind areas may be undergoing concentrations above the 1 yr chronic reference exposure level of  $1 \mu\text{g}/\text{m}^3$  (California OEHHA, 2001).

### Geographic variations of Kilauea's plume

The Kilauea plume affects the Kau district and is not restricted to oceanic terrain.  $\text{SO}_2$  concentrations did not decrease strongly with distance from source as expected. Instead, the highest  $\text{SO}_2$  concentrations were in the Na`alehu area, ~68 km from the source, where the plume passes over oceanic terrain. Sampling of a lateral part of the plume partially accounts for this  $\text{SO}_2$  pattern. At Masaya volcano, Nicaragua,  $\text{SO}_2$  concentrations are negligible at down-plume distances comparable to the distance Na`alehu is from the source, despite similar emission rates (Delmelle et al., 2002). Although wind-speed differences would influence this comparison, we argue that marine vs. land effects dominate.

A major mechanism for conversion of  $\text{SO}_2$  is the hydroxyl radical ( $\text{OH}\cdot$ ), which readily reacts in the gas phase by photoinitiation to form  $\text{H}_2\text{SO}_4$  (ATSDR, 1998; Seinfeld, 1986). Availability of  $\text{OH}\cdot$  in ambient air depends on the presence of competitors. Oceanic terrain is not a major source for available  $\text{OH}\cdot$  (Seinfeld & Pandis, 1998) because dimethyl sulfide reacts predominantly with  $\text{OH}\cdot$ , limiting availability of  $\text{OH}\cdot$  for oxidation of  $\text{SO}_2$ .

Aerosol increased with altitude regardless of distance from source. We propose that primary release of  $\text{H}_2\text{SO}_4$  from the eruption site contributes to the downwind aerosol burden to combine with  $\text{SO}_2$  oxidation mechanisms enhanced on land. At the Pu`u `O`o vent, Chuan (1995) measured  $\text{H}_2\text{SO}_4$  in ambient air samples. Moreover, primary released sulfates have been measured at other degassing basaltic volcanoes (Andres et al., 1993; Allen et al., 2002). At Kilauea, Porter et al. (2002) estimated the  $\text{SO}_2$  oxidation half-life

to be 6.0 hours, under the assumption that no  $\text{H}_2\text{SO}_4$  was released. Therefore, an unknown percentage of primary sulfates are likely released.

When the diurnal trade winds move inland and upslope, dissipating the plume horizontally and vertically into new air masses under the 1800 m inversion,  $\text{SO}_2$  oxidation may be enhanced with atmospheric water processes and available  $\text{OH}\cdot$  fed by the photolysis of nitroxy anions  $\text{NO}_2^-$  and  $\text{NO}_3^-$  and  $\text{H}_2\text{O}_2$  (Pasiuk-Bronikowska, 2002). Within clouds and fogs, hydrogen peroxide ( $\text{H}_2\text{O}_2$ ) is an effective and fast oxidant of  $\text{SO}_2$  independent of pH (Seinfeld & Pandis, 1998).  $\text{SO}_2$  is absorbed into hydrosols (clouds, fog, or rain droplets) where aqueous-phase oxidation occurs by  $\text{H}_2\text{O}_2$  producing  $\text{H}_2\text{SO}_4$  (Seinfeld & Pandis, 1998). Concentrations of  $\text{H}_2\text{O}_2$  are low near the surface and rise to a maximum at the top of the boundary layer (Seinfeld & Pandis, 1998), where cloud formation is common in Hawai'i.

## Conclusions

After 22 years of eruption at Kilauea, this environmental investigation found volcanogenic  $\text{SO}_2$  and fine aerosol at concentrations that justify public health concern for communities along the plume path. We posit that the striking decrease of  $\text{SO}_2$  with altitude results as wind sweeps the plume landward to react with land-derived oxidants and upward to interact with atmospheric hydrosols. Increase of aerosol with altitude is likely from a primary emission from Kilauea and oxidation of  $\text{SO}_2$ . The strong geographic variability of pollutant concentrations underscores the need for regional sampling prior to point monitoring in exposure assessments. Further work is warranted to investigate oceanic vs. land interactions with the plume and potential adverse effects on the human population.

## Acknowledgements

We thank the residents of Kau and Hawi, the Hawaiian Volcano Observatory, the Kau district schools, and the Kau Hospital for permission to sample. We thank the following contributors: P. Baxter, M.D., R. Bibilone, T. Elias, J. Green, M.D., A.J. Kimerling, A. Longo, C. Neumann, W.I. Rose, A.J. Sutton, R. Vong, and J.K. Yun.

Table 2.1. Atmospheric SO<sub>2</sub> Concentration Data. These data were obtained from Harwell Scientifics passive samplers during a three-week exposure in August - September, 2003. Field reliability tested by paired samples was  $\pm 5.5\%$ . An altitude flux correction (Larson and Vong, 1990) was made to the mass concentration lab data ( $\mu\text{g}/\text{m}^3$ ); thereby considering the effects of changes in pressure and temperature on the tube diffusivity at each sample site. The estimated relative error was + 8% to 11% to the criterion instrument (pulse fluorescence monitor) (U.S. National Park Service, 2004).  $\text{ppbv} = [(8.314\text{E}-2) * T / p\text{MSO}_2] * (F_g / \text{m}^3 * 103)$

<b>SO<sub>2</sub> Diffusion Tube Data</b>				<b>Diffusivity ratio</b>		<b>mass concentration</b>		<b>Gas mixing ratio ppbv</b>		
<b>Location</b>	<b>Altitude meters/feet</b>		<b>temp k</b>	<b>pres. mb</b>	<b>D/D0</b>	<b>Lab <math>\mu\text{g}/\text{m}^3</math></b>	<b><math>\mu\text{g}/\text{m}^3</math> corrected</b>	<b>ppbv altitude adjusted</b>	<b>Lab ppbv 1 atm. /20C</b>	<b>ppbv % change</b>
Park SO <sub>2</sub> monitor (3.88 ppbv)	1215	3,986	289.28	872.06	1.10	11.3	10.26	4.42	4.3	2.76
HVO SO <sub>2</sub> monitor (7.69 ppbv)	1123	3,684	290.00	882.73	1.09	21.8	19.95	8.51	8.2	3.69
Summit plume – Halemaumau Crater	1110	3,642	290.12	884.24	1.09	1833.0	1679.08	715.67	688.0	3.87
Eruption plume – Chain of Craters Rd.	518	1,700	294.86	952.91	1.04	354.0	339.42	136.43	133.0	2.52
Mauna Iki's summit	924	3,032	291.61	905.82	1.08	58.4	54.30	22.71	22.0	3.12
<b>Exposed cohort</b>										
Kapapala trend	981	3,220	291.15	899.20	1.08	20.1	18.60	7.83	7.5	4.16
Kapapala trend	668	2,192	293.66	935.51	1.05	29.3	27.78	11.33	11.0	2.91
Kapapala trend	509	1,670	294.93	953.96	1.04	41.3	39.63	15.91	15.5	2.60
Wood Valley	750	2,460	293.00	926.00	1.06	25.5	24.03	9.88	9.6	2.81
Pahala/Wood Valley	549	1,800	294.61	949.32	1.05	32.0	30.61	12.34	12.0	2.77
Pahala trend	792	2,600	292.66	921.13	1.06	21.6	20.29	8.37	8.1	3.28
Pahala trend	500	1,640	295.00	955.00	1.04	47.7	45.80	18.38	17.9	2.59
Pahala trend house	366	1,200	296.07	970.54	1.03	32.9	31.89	12.64	12.3	2.68
Pahala trend school	287	940	296.70	979.71	1.03	37.1	36.16	14.23	13.9	2.30
Pahala trend house	274	900	296.81	981.22	1.02	42.9	41.85	16.45	16.1	2.11

Table 2.1. Atmospheric SO<sub>2</sub> Concentration Data (Continued).

Location (exposed)	Altitude meters / feet		temp k	pres. mb	D/D0	Lab µg/m <sup>3</sup>	µg/m <sup>3</sup> corrected	ppbv altitude adjusted	Lab ppbv 1 atm. / 20C	ppbv % change
Pahala trend house	274	900	296.81	981.22	1.02	36.7	35.81	14.07	13.8	1.92
Pahala trend hospital	250	820	297.00	984.00	1.02	46.7	45.64	17.89	17.5	2.21
Pahala trend hospital	244	800	297.05	984.70	1.02	52.0	50.84	19.92	19.5	2.12
Pahala trend	229	750	297.17	986.44	1.02	51.9	50.79	19.88	19.5	1.90
Pahala trend	144	473	297.85	996.30	1.02	41.9	41.25	16.02	15.7	1.99
Pahala/Puna	396	1,300	295.83	967.06	1.03	34.3	33.18	13.18	12.9	2.16
Pahala/Puna	122	400	298.02	998.85	1.01	40.3	39.73	15.40	15.1	1.95
Punalu'u trend	549	1,800	294.61	949.32	1.05	36.5	34.92	14.08	13.7	2.68
Punalu'u trend	396	1,300	295.83	967.06	1.03	35.1	33.95	13.49	13.2	2.17
Punalu'u trend	90	296	298.28	1002.56	1.01	49.1	48.51	18.75	18.4	1.86
Punalu'u trend	42	139	298.66	1008.13	1.01	46.6	46.19	17.78	17.5	1.56
Punalu'u area	390	1,280	295.88	967.76	1.03	49.6	48.00	19.06	18.6	2.43
Honu'apo area	443	1,452	295.46	961.61	1.04	50.1	48.30	19.28	18.8	2.48
Honu'apo	60	197	298.52	1006.04	1.01	57.6	57.03	21.98	21.6	1.73
Na'alehu trend	572	1,877	294.42	946.65	1.05	49.7	47.46	19.18	18.6	3.01
Na'alehu trend house	232	760	297.14	986.09	1.02	70.8	69.28	27.12	26.6	1.91
Na'alehu trend house	213	700	297.30	988.29	1.02	51.0	49.97	19.53	19.1	2.19
Na'alehu trend house	207	680	297.34	988.99	1.02	60.9	59.69	23.31	22.8	2.21
Na'alehu trend school	195	640	297.44	990.38	1.02	63.3	62.10	24.23	23.7	2.17
Na'alehu trend school	195	640	297.44	990.38	1.02	54.0	52.97	20.67	20.2	2.26
Na'alehu trend clinic	195	640	297.44	990.38	1.02	55.2	54.15	21.13	20.7	2.02
Na'alehu trend clinic	195	640	297.44	990.38	1.02	59.4	58.27	22.73	22.3	1.91
Na'alehu trend	183	600	297.54	991.77	1.02	76.2	74.81	29.16	28.6	1.91
Na'alehu trend	152	500	297.78	995.37	1.02	88.0	86.58	33.65	33.0	1.93
Waiohinu Village	305	1,000	296.56	977.62	1.03	84.6	82.36	32.46	31.8	2.02
Kipuka Nahuaopala	238	780	297.10	985.39	1.02	56.2	54.97	21.53	21.1	1.99

Table 2.1. Atmospheric SO<sub>2</sub> Concentration Data (Continued).

Location (exposed)	Altitude meters / feet		temp k	pres. mb	D/D0	Lab µg/m <sup>3</sup>	µg/m <sup>3</sup> corrected	ppbv altitude adjusted	Lab ppbv 1 atm. / 20C	ppbv % change
South point trend	742	2,434	293.06	926.93	1.06	31.4	29.61	12.16	11.8	2.97
South point trend	506	1,660	294.95	954.30	1.04	45.7	43.86	17.61	17.1	2.89
South point trend	378	1,240	295.98	969.15	1.03	54.0	52.30	20.75	20.0	3.61
South point trend	277	909	296.78	980.87	1.03	59.3	57.84	22.74	22.3	1.92
South point trend	15	50	298.88	1011.26	1.01	46.9	46.57	17.88	17.6	1.57
Ocean View	1244	4,082	289.05	868.70	1.10	18.4	16.67	7.21	7.0	2.85
Ocean View	866	2,842	292.07	912.54	1.07	14.8	13.82	5.75	5.6	2.57
<i>Exposed cohort Average</i>					1.03	46.48	45.21	17.84	17.43	2.35

Control Cohort Location	Altitude meters/feet		temp k	pres. mb	D/D0	Lab µg/m <sup>3</sup>	µg/m <sup>3</sup> corrected	ppbv altitude adjusted
Hawi - north	146	480	297.83	996.06	1.02	<1.2		
Hawi - west	195	640	297.44	990.38	1.02	1.8	1.77	0.69
Hawi - south	268	880	296.86	981.91	1.02	1.2	1.17	0.46
Hawi - downtown	183	600	297.54	991.77	1.02	3.0	2.95	1.15
Hawi - east, pair	158	520	297.74	994.67	1.02	1.2	1.18	0.46
Hawi - east, pair	158	520	297.74	994.67	1.02	<1.2		
<b>Control cohort Average</b>						1.8	1.77	0.69

Table 2.2. Atmospheric Fine Particle Aerosol Data September, 2003. Precision is  $\pm 1 \mu\text{g}/\text{m}^3$ .

AEROSOL SAMPLE LOCATION & ALTITUDE	DATE /TIME	SAMPLING CONDITIONS	Count total	0.8 $\mu\text{m}$	0.3 $\mu\text{m}$	0.1 $\mu\text{m}$	$\leq 0.3 \mu\text{m}$	Adjusted $\leq 0.3 \mu\text{m}$
		(wind, speed, climate, visual)						
<b>Exposed Cohort Area</b>			$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$
Kapapala 981 m	9/15/03, 1645	trades 5 swirls, overcast, pre-rain, haze	5.87	0.69	3.52	1.66	5.18	4.87
Kapapala 792 m	9/15/03, 1445	trades <5, overcast, pre-rain, haze	4.55	0.75	2.56	1.24	3.80	3.49
Kapapala 668 m	9/15/03, 1400	trades 5-10, overcast	2.78	0.63	1.75	0.38	2.13	1.82
Wood Valley 750 m	9/11/03, 0737	trades <5, clear sun	1.64	0.73	0.49	0.42	0.91	0.60
Pahala Trend 144 m	9/20/03, 1520	trades 5, overcast	1.42	0.69	0.57	0.14	0.71	0.40
Pahala Trend 229 m	9/20/03, 1422	trades <5, overcast	1.70	1.04	0.41	0.24	0.65	0.34
Pahala Trend Hospital 244 m	9/12/03, 2037	trades <5, partial clouds, dark	13.61	0.94	8.81	3.86	12.67	12.36
Pahala Trend Base 274 m	9/9/03, 0600	trades <5, sunrise, clear	1.61	0.47	1.10	0.03	1.13	0.82
	0815	trades <5, sun, clear	1.97	0.66	0.70	0.60	1.30	0.99
	1005	trades <5, partial clouds	1.46	0.58	0.32	0.56	0.88	0.57
	1400	trades 5-10, overcast, pre-rain	1.61	0.92	0.47	0.20	0.67	0.36
	1600	trades 5, overcast, post-rain	3.30	2.12	0.85	0.32	1.17	0.86
	1800	trades 5, overcast, spotty sun, pre-rain	2.17	1.23	0.61	0.32	0.93	0.62
	2000	trades <5, overcast, dark, no rain	1.11	0.46	0.42	0.22	0.64	0.33
	2200	trades 5, overcast, dark, no rain	4.67	0.75	3.44	0.47	3.91	3.60
	2300	trades <5, overcast, dark, no rain	2.15	0.00	1.45	0.70	2.15	1.84
	0300	trades <5, overcast, dark, no rain	1.95	1.19	0.51	0.24	0.75	0.44
<b>Average for 24-hour sample</b>		low trades with rain	<b>2.20</b>	<b>0.84</b>	<b>0.99</b>	<b>0.37</b>	<b>1.35</b>	<b>1.04</b>
	9/10/03, 2008	trades calm, overcast, dark, post-rain	5.41	0.92	3.34	1.14	4.48	4.17
	9/10/03, 2210	trades calm, moonbeams, partial cloud	1.87	0.71	1.00	0.16	1.16	0.85
	9/12/03, 1530	trades 5-10, overcast, pre-rain	1.53	0.78	0.57	0.17	0.74	0.43
	9/12/03, 1838	trades 5-10, overcast, sunset, post-rain	6.41	1.75	3.43	1.22	4.65	4.34
	9/12/03, 2002	trades <5, partial clouds, dark	6.71	0.92	3.56	2.21	5.77	5.46

Table 2.2. Atmospheric Fine Particle Aerosol Data, September 2003 (Continued).

AEROSOL SAMPLE LOCATION & ALTITUDE	DATE /TIME	SAMPLING CONDITIONS	Count total	0.8 $\mu\text{m}$	0.3 $\mu\text{m}$	0.1 $\mu\text{m}$	$\leq 0.3 \mu\text{m}$	Adjusted $\leq 0.3 \mu\text{m}$
		(wind, speed, climate, visual)						
<b>Exposed Cohort Area</b>			$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$
Pahala (continued) 274 m	9/13/03, 2308	trades <5, moon & stars, clear night	4.27	1.72	2.21	0.34	2.55	2.24
	9/14/03, 1830	trades 5-10, overcast, sunset, no rain	0.95	0.25	0.40	0.30	0.70	0.39
	9/14/03, 2030	trades <5, overcast, dark, no rain	2.69	1.58	0.76	0.34	1.10	0.79
	9/15/03, 2030	trades calm, stars clear, dark	1.96	0.56	0.96	0.44	1.40	1.09
	9/16/03, 1800	trades <5, overcast, no rain	1.34	0.65	0.51	0.18	0.69	0.38
	9/17/03, 1949	trades <5, clear night	3.11	1.05	1.21	0.84	2.05	1.74
<b>Average of evening samples</b>		trades, some rain, typical evenings	3.30	0.99	1.63	0.67	2.30	1.99
Pahala Trend 500 m	9/11/03, 0837	trades 5, sun, haze	3.25	0.58	1.25	1.42	2.67	2.36
	9/11/03, 1345	trades 5-10, overcast, clear	1.35	0.11	1.11	0.11	1.22	0.91
Pahala Trend Upper 792 m	9/18/03, 1007	trades, <5, overcast, pre-rain, haze	5.41	1.30	2.40	1.71	4.11	3.80
Pahala/Punalu'u 396 m	9/11/03, 1157	trades <5, overcast	1.04	0.20	0.51	0.32	0.83	0.52
Punalu'u Trend 42 m	9/10/03, 0950	trades <5, partial clouds	<.01	0.00	0.00	0.00	0.00	0.00
Punalu'u Trend 90 m	9/10/03, 1025	trades 5-10, sun, clouds area	0.50	0.23	0.06	0.21	0.27	0.00
Punalu'u Trend 396 m	9/11/03, 0944	trades 5-10, partial sun, haze	4.48	0.75	1.79	1.93	3.72	3.41
Punalu'u area 390 m	9/11/03, 1132	trades <5, partial clouds, pre-rain, haze	1.29	0.57	0.41	0.29	0.70	0.39
Honu'apo area 443 m	9/11/03, 1029	trades 10, clear sun	1.61	0.52	0.63	0.44	1.07	0.76
Na'alehu Trend 152 m	9/18/03, 1236	trades 10, overcast	1.69	1.04	0.41	0.23	0.64	0.33
Na'alehu Trend school 195 m	9/12/03, 1708	trades <5, overcast	3.31	1.95	0.59	0.76	1.35	1.04
Na'alehu (end of town) 259 m	9/12/03, 1637	trades 5, overcast	2.75	1.45	0.70	0.60	1.30	0.99
	9/22/03, 1515	trades <5, partial clouds	1.05	0.95	0.06	0.02	0.08	0.00
Na'alehu Trend Upper 572 m	9/11/03, 1053	trades 10, sun, partial clouds	1.64	0.52	0.67	0.44	1.11	0.80
South point Trend 277 m	9/10/03, 1526	trades 15, partial clouds, clear	1.75	0.79	0.48	0.47	0.95	0.64
South point Trend 378 m	9/10/03, 1452	trades 10, partial clouds, clear	1.25	0.63	0.29	0.32	0.61	0.30
South point Trend 506 m	9/10/03, 1426	trades 5-10, sun, partial clouds, clear	1.09	0.48	0.21	0.40	0.61	0.30



Table 2.2. Atmospheric Fine Particle Aerosol Data, September 2003 (Continued).

Table 22: Aerosols from the Pacific Area Data, September 2003 (Continued).									
AEROSOLSAMPLE LOCATION & ALTITUDE		DATE /TIME	SAMPLING CONDITIONS	Count total	0.8 μm	0.3 μm	0.1 μm	≤ 0.3 μm	Adjusted ≤ 0.3 μm
			(wind, speed, climate, visual)						
Exposed Cohort Area				μg/m3	μg/m3	μg/m3	μg/m3	μg/m3	μg/m3
South point Trend	742 m	9/17/03, 1739	trades 5-10, overcast, haze	9.31	0.31	8.09	0.89	8.98	8.67
		9/20/03, 1041	trades 5, partial clouds, haze	9.16	0.72	4.03	4.40	8.43	8.12
Ocean View	634 m	9/10/03, 1645	trades 5-10, pre-rain, clear	2.12	0.68	1.07	0.37	1.44	1.13
		9/12/03, 1017	trades 5-10, partial clouds, clear	3.79	0.40	1.66	1.72	3.38	3.07
Ocean View	930 m	9/10/03, 1720	trades 5-10, pre-rain	4.93	2.00	2.33	0.59	2.92	2.61
		9/12/03, 1045	trades 5, partial clouds, clear	5.71	0.58	3.07	2.06	5.13	4.82
Ocean View	1302 m	9/10/03, 1805	trades 5-10, overcast, pre-rain, haze	8.54	6.37	1.95	0.20	2.15	1.84
		9/12/03, 1115	trades 5-10, partial clouds, haze	6.88	0.30	2.88	3.70	6.58	6.27
Ocean View	1393 m	9/12/03, 1140	trades 5 circular, partial clouds, haze	10.70	1.23	5.63	3.84	9.47	9.16
Exposed Cohort Area samples during variable winds									
Ocean View	1487 m	9/23/03, 1524	Variables 5, upslope, sun, haze	13.04	1.21	10.75	1.07	11.82	11.51
Ocean View	1244 m	9/23/03, 1502	Variables 5, trade-like, overcast, haze	10.39	1.04	8.06	1.28	9.34	9.03
Ocean View	1243 m	9/23/03, 1347	Variables 5, upslope, sun, clouds, haze	10.35	1.36	7.58	1.40	8.98	8.67
Ocean View	1049 m	9/23/03, 1607	Variables 5, upslope, sun, clouds	12.33	1.07	9.72	1.53	11.25	10.94
Ocean View	866 m	9/23/03, 1658	Variables <5, sun, distal haze	12.81	1.39	10.4	1.02	11.42	11.11
Ocean View	698 m	9/23/03, 1632	Variables <5, upslope, sun, wispy, haze	7.32	1.07	5.47	0.77	6.24	5.93
		9/23/03, 1807	Variables calm, sun, haze & sunset color	10.50	1.59	8.09	0.81	8.90	8.59
Ocean View	561 m	9/23/03, 1720	Variables 5, trade-like to west, sun, haze	12.95	1.39	10.71	0.83	11.54	11.23
Ocean View	305 m	9/23/03, 1742	Variables 5-10, trade-like, sun, haze	4.62	0.69	3.32	0.59	3.91	3.60
Kapapala Highway	762 m	9/24/03, 1306	Variables 5 inland, overcast	5.43	0.94	3.39	1.10	4.49	4.18
Kapapala trend Hwy	549 m	9/24/03, 1240	Variables 5 inland upslope, overcast	5.03	0.79	3.58	0.66	4.24	3.93
North of Pahala Hwy	305 m	9/24/03, 1341	Variables 5-10 inland, overcast	3.03	0.52	1.87	0.64	2.51	2.20
Pahala Trend Hwy	244 m	9/24/03, 1408	Variables <5 upslope & northerly, traffic	3.57	1.15	1.27	1.13	2.40	2.09
Pahala Trend (Base)	274 m	9/24/03, 1152	Variables 5 inland, overcast, haze	2.79	0.52	1.79	0.48	2.27	1.96

Table 2.2. Atmospheric Fine Particle Aerosol Data, September 2003 (Continued).

AEROSOL SAMPLE LOCATION & ALTITUDE	DATE /TIME	SAMPLING CONDITIONS	Count total	0.8 $\mu\text{m}$	0.3 $\mu\text{m}$	0.1 $\mu\text{m}$	$\leq 0.3\mu\text{m}$	Adjusted $\leq 0.3\mu\text{m}$
		(wind, speed, climate, visual)						
Hawi – downtown 183 m	9/19/03, 1107	trades 10-15, clear sun	1.10	0.69	0.25	0.15	0.40	
Hawi - end of town 195 m	9/19/03, 1419	trades 10-15, clear sun	1.02	0.63	0.20	0.18	0.38	
Hawi - north street 146 m	9/19/03, 1155	trades 10-15, clear sun	0.77	0.46	0.14	0.16	0.30	
Hawi - south high elev. 268 m	9/19/03, 1232	trades 10-15, patchy clouds	0.70	0.56	0.02	0.12	0.14	
<b>Average for Control site</b>			<b>0.90</b>	<b>0.59</b>	<b>0.15</b>	<b>0.15</b>	<b>0.31</b>	
<b>Kona Area</b>								
Captain Cook 561 m	9/17/03, 1547	trades <5, overcast, pre-rain, haze	3.55	0.63	2.13	0.78	2.91	<b>2.60</b>
Kipahoe reserve	9/17/03, 1656	trades <5, overcast, pre-rain, haze	7.62	1.04	5.50	1.07	6.57	<b>6.26</b>

Table 2.3. Indoor/Outdoor (I/O) Ratios of SO<sub>2</sub> in the Kau District of the Big Island, Hawai'i, U.S.A. Sulfur Dioxide concentration was measured by Harwell Scientifics passive diffusion tube samplers exposed for three weeks in August-September, 2003.

<b>I/O RATIO</b>	<b>INDOOR μg/m<sup>3</sup></b>	<b>OUTDOOR μg/m<sup>3</sup></b>	<b>INDOOR ppbv</b>	<b>LOCATION DESCRIPTION</b>
<b>0.69</b>	43.0	62.1	16.8	School cafeteria
<b>0.59</b>	31.4	53.0	12.3	School classroom
<b>0.56</b>	33.6	59.7	13.1	Plantation house
<b>0.68</b>	34.0	50.0	13.3	Plantation house
<b>0.15</b>	8.1	55.0	3.2	House
<b>0.71</b>	36.1	50.8	14.1	Hospital dayroom
<b>0.65</b>	29.8	45.6	11.7	Hospital clinic
<b>0.69</b>	24.9	36.2	9.8	School classroom
<b>0.23</b>	7.2	31.9	2.8	House
No data				2 tubes disturbed

## **Field Observation Notes**

**August – September 2003**

Visible harmful effects attributed to the sulfurous air pollution were observed in the environment. Street signs in Ocean View had increasing corrosion with a rise in altitude. Rusting of metal roofs and machinery was noted throughout the exposed cohort area. Leaf damage was observed on coffee plants in the upper Pahala area.

Interviews with local residents revealed conflicting experiences. A farmer in Pahala reported abundant crops from a variety of vegetables, whereas the macadamia nut production in the area was felt to have a decreased yield since the eruption. Other farmers of vegetables and coffee described high yields and no detrimental effects on crops. Residents from the Na`alehu area stated that flora was affected by a decreased ability to produce or early drop of fruit or nuts, chlorosis on foliage, and parasite infestation in certain areas. A long-time resident of Kau District described indicator plants as yellow/white ginger and blackberries. The author confirmed this by field observation.

Local ranchers in the Pahala area described the plume's behavior as moving inland and swirling every afternoon during typical trade winds. Effects of burning eyes and breathing irritation were reported by ranchers, whose outdoor exposure averaged 12 hours a day. Farmers in the upper Punalu`u area described the plume as passing westerly through high altitude hill areas each day, rapid rusting of their machinery as compared to other Big Island areas, and significant respiratory and eye irritation when lava-sea entry occurs (likely HCl aerosol from laze).

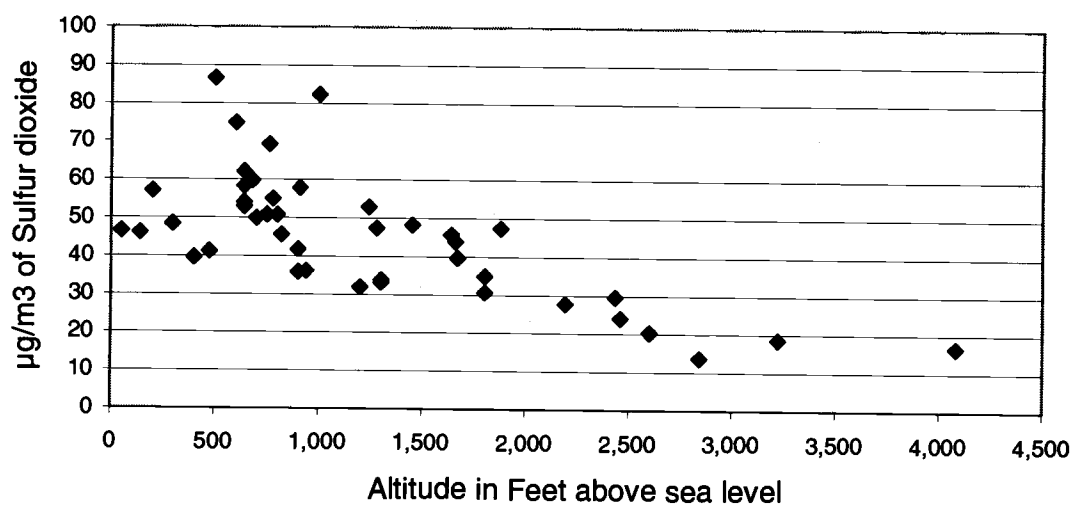


Figure 2.3. Correlation scatter plot of  $\text{SO}_2$  mass concentration ( $\mu\text{g}/\text{m}^3$ ) and altitude. ( $r = -.70$ ,  $p < 0.01$ ,  $N = 43$ )

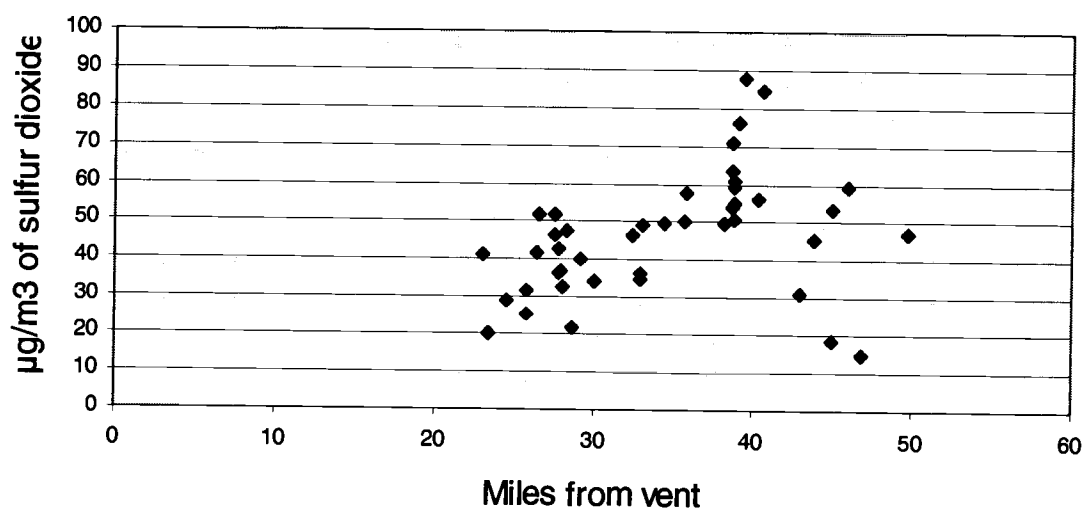


Figure 2.4. Correlation scatter plot of  $\text{SO}_2$  mass concentration ( $\mu\text{g}/\text{m}^3$ ) and distance from source. ( $r = +.35$ ,  $p < 0.05$ ,  $N = 43$ )

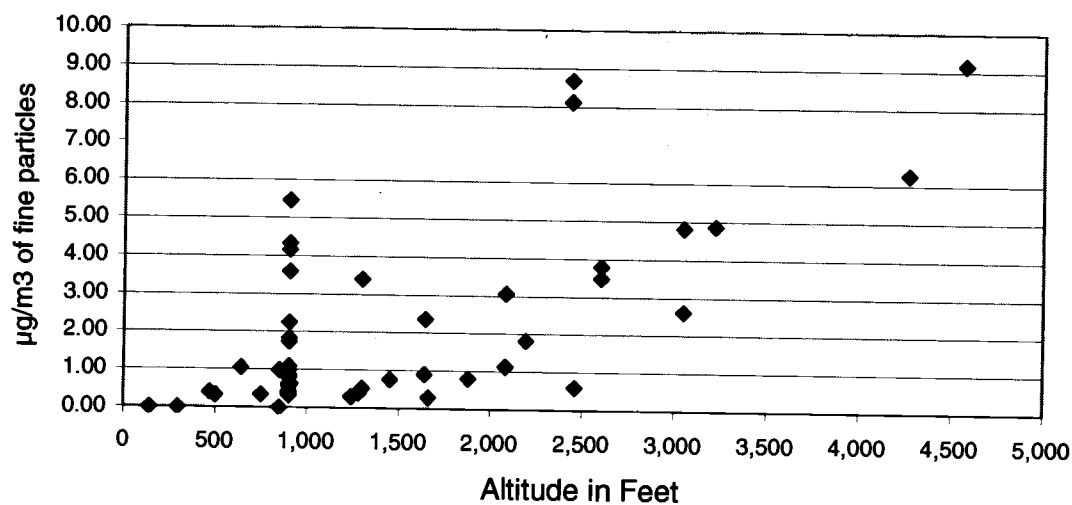


Figure 2.5. Correlation scatter plot for fine particles ( $\leq 0.3 \mu\text{m}$ ) and altitude during trade winds. ( $r = +.69$ ,  $p < 0.01$ ,  $N = 52$ )

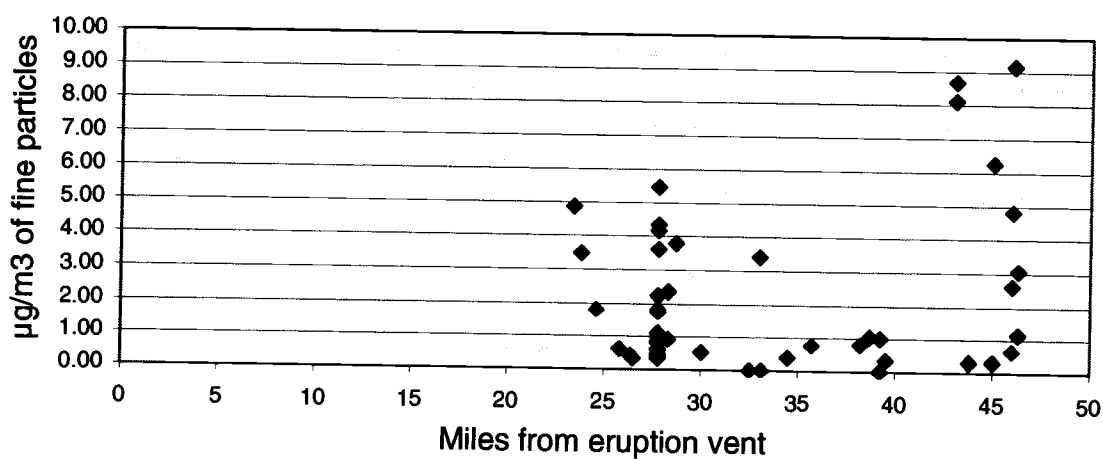


Figure 2.6. Correlation scatter plot of fine particles ( $\leq 0.3 \mu\text{m}$ ) and distance from source during trade winds. ( $r = +.30$ ,  $p < 0.05$ ,  $N = 52$ )

**PREVALENCE OF CARDIO-PULMONARY HEALTH EFFECTS  
FROM VOLCANIC AIR POLLUTION,  
KAU DISTRICT, HAWAII, USA**

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Pending submission 2005

**Abstract**

This cohort study assessed adult human health effects associated with chronic exposure to volcanogenic air pollution. Exposed cohorts of sulfur dioxide gas and fine sulfate aerosol were located in the Kau District, Hawai'i, downwind from erupting Kilauea Volcano. The control cohort was from an unexposed area on the island. Cardio-pulmonary effects were compared revealing substantially increased prevalence of cough, phlegm, throat irritation, rhinorrhea, sinus congestion, and eye irritation. Field-measured blood pressure and respirations were significantly elevated. SO<sub>2</sub> and sulfate exposed elderly non-smokers had averages of 8 and 7.4 beats/minute faster pulse rates than controls. Long-term residency in active and degassing volcanic areas may influence cardio-pulmonary health.



## Introduction

An estimated 500 million humans worldwide live near active volcanoes (Baxter et al., 1999). Eruption at Kilauea Volcano on the Big Island of Hawai'i, has occurred nearly continuously since 1983, emitting an average of 1,600 tons of sulfur dioxide (SO<sub>2</sub>) per day (Sutton & Elias, 2002); the largest point source for SO<sub>2</sub> in the United States. Unlike explosive plinian eruptions such as Mt. St. Helens, that send particles and gases into the stratosphere and ash fall in the region, Hawaiian eruptions effusively release gases and aerosols into the lower troposphere where humans reside, thereby creating an environment that may be detrimental to cardio-pulmonary health.

Knowledge on the health of populations in active volcanic areas is sparse but growing. Harmful health effects from air borne ash have been identified (Baxter, 1981; Shinkura et al., 1999; Allen et al., 2000; Berube, et al., 2004). Degassing volcanic areas can have associated health risks to local populations from SO<sub>2</sub>, carbon dioxide, radon, and hydrogen sulfide (Nicholson et al., 1996; Baxter et al., 1999; Demelle et al., 2002). In Hawai'i, limited work has been conducted on health effects during Kilauea's current 22-year long eruption. Several studies however, have identified increased hospital visits for respiratory conditions (Bernstein et al. 1984; Mannino et al., 1996; Michaud et al., 2004) and respiratory effects in pediatric asthmatics related to the eruption (Morrow, 2000). In addition, a prospective study into effects on pediatric lung development currently is underway on the Big Island (Sutton et al., 2003).

The objective of this study was to conduct a cohort investigation of adult residents in Kau District (Figure 3.1), the first downwind area from Kilauea, focusing on prevalence measures of respiratory and cardiac symptoms, disease, and abnormal vital signs. These results identify an epidemiology-based volcanic SO<sub>2</sub> and sulfate syndrome in adults.

### Background information: Exposure levels

SO<sub>2</sub> is released from Kilauea in a plume from a vent at the eruption site, and diffusely from the summit. As the plume travels at various wind speeds, oxidation to sulfuric acid aerosol (H<sub>2</sub>SO<sub>4</sub>) and then to other sulfates occurs in the atmosphere at various rates depending on sunlight, meteorology, and availability of oxidants. A visible

build-up of sulfate pollution, called “Vog” by local residents, can occur with light variable winds. Therefore, exposure is not the same in all downwind areas, as different areas experience different species of sulfur, at varying concentrations.

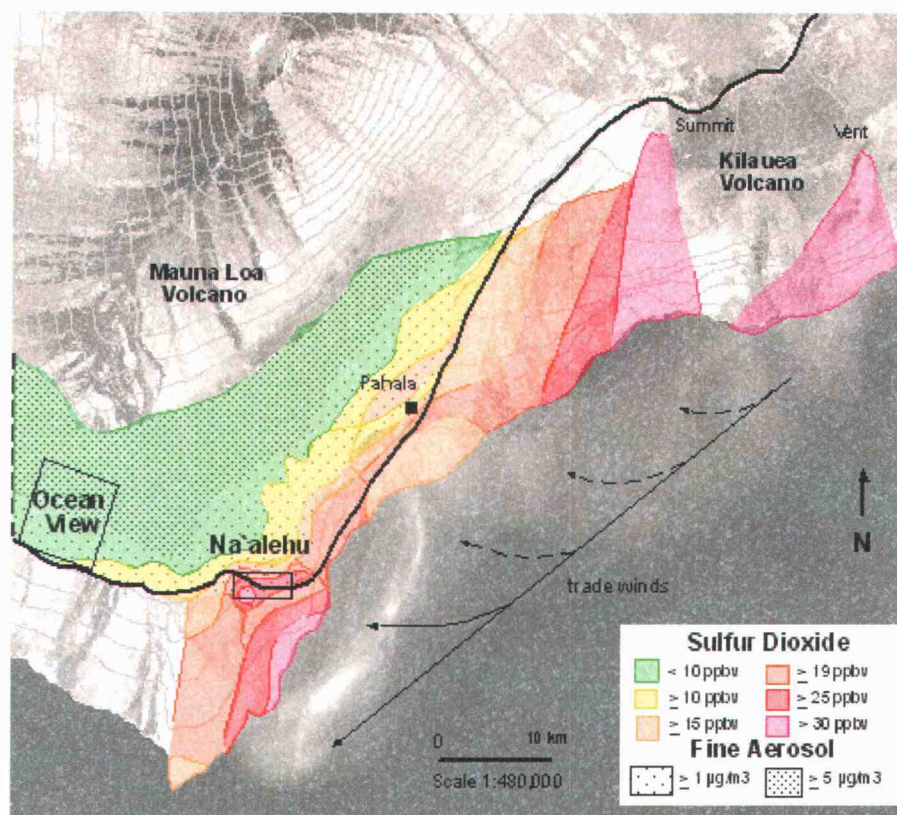


Figure 3.1 Contour map of volcanic air pollution measured in Kau District, Hawai'i, September 2003. Base map provided by State of Hawaii GIS Program I-Map.

Residents of Kau District have been exposed to volcanogenic air pollution at concentrations that warrant concern for adverse health effects (Figure 3.1). In 2003, the first environmental sampling for  $\text{SO}_2$  and fine aerosol pollution ( $\leq 0.3 \mu\text{m}$ ) was conducted in a down-wind region of Kilauea and in a control site (Longo et al., 2005). The average three-week ambient  $\text{SO}_2$  concentration in the Kau District was 17.8 ppbv; an amount 25 times greater than in the control site (0.7 ppbv). The ambient  $\text{SO}_2$  concentrations ranged from 6 ppbv in Ocean View, to 34 ppbv ( $87 \mu\text{g}/\text{m}^3$ ) in the Na'alehu area (Longo et al., 2005). Ambient air and indoor concentrations of  $\text{SO}_2$  were detected above the acute Minimal Risk Level (MRL) of 10 ppbv for an acute exposure of <14 days (ATSDR,

1998), and above the World Health Organization's (WHO, 1999) guideline of 50  $\mu\text{g}/\text{m}^3$  for an annual exposure (Longo et al., 2005).  $\text{SO}_2$  was found to decrease in concentration with rise in altitude ( $r = -0.70$ ,  $p < .001$ ), resulting in the highest exposures along coastal areas of the district.

In contrast to  $\text{SO}_2$ , during the same three-week period fine particles ( $\leq 0.3\mu\text{m}$ ) increased in concentration with altitude regardless of distance away from the volcano (partial  $r_{12,3} = +0.65$ ,  $p < .001$ ). Concentrations of  $\leq 0.3\mu\text{m}$  particles ranged from 2 to 40 times greater in Kau District (0.61 to 11.82  $\mu\text{g}/\text{m}^3$ ) than in the control site (0.31  $\mu\text{g}/\text{m}^3$ ) (Longo et al., 2005). In Ocean View, amounts as high as 9  $\mu\text{g}/\text{m}^3$  during typical trade winds and 11  $\mu\text{g}/\text{m}^3$  in variable winds were measured. Chuan (1995, 1997, 1998) has characterized the particles in Kilauea's plume as containing sulfuric acid aerosol ( $\text{H}_2\text{SO}_4$ ) and elemental sulfur ( $\leq 0.3\mu\text{m}$ ), sulfates (1.7  $\mu\text{m}$ ), and background aerosols of sodium chloride (0.8  $\mu\text{m}$ ), and silicates ( $>2\mu\text{m}$ ). Aqueous  $\text{H}_2\text{SO}_4$  comprised more than 80% of the total aerosol mass (Chuan, 1995, 1998). With continual eruption at Kilauea Volcano, residents in high altitude downwind areas are likely exposed to concentrations of  $\text{H}_2\text{SO}_4$  above the one-year chronic reference exposure level (REL) of 1  $\mu\text{g}/\text{m}^3$  (California OEHHA, 2001).

## Toxicology

Sulfur dioxide is a water-soluble chemical irritant. The major physiological effect from  $\text{SO}_2$  exposure is bronchoconstriction or increased pulmonary resistance (WHO, 1979; Koenig, 2000). In healthy non-asthmatic adults, a dose-response relationship exists starting at various thresholds for different exposure durations and different activity levels (Koenig, 2000; Schlesinger, 1999; WHO, 1979). Further studies, however, have identified general population sub-groups sensitive to  $\text{SO}_2$  at lower concentrations: 1) asthmatics; 2) children and adolescents; 3) respiratory and cardiac compromised individuals; and 4) healthy but sensitive to  $\text{SO}_2$  individuals (WHO, 1999; Schlesinger, 1999; Koenig, 2000; Schwela, 2000; Dickey, 2000). Asthmatics are most sensitive, showing a non-threshold, dose-response relationship with exposure to  $\text{SO}_2$  (WHO, 1999). The Minimal Risk Level is a public health guideline that considers the sensitive sub-groups of the population (ATSDR, 1998). Chronic exposure to  $\text{SO}_2$  is associated with

increased morbidity and mortality worldwide (WHO, 1999; Shinkura et al., 1999; Schwela, 2000; Venners et al. 2003).

In addition, sulfuric acid is a respiratory irritant. Sulfate aerosols can negatively affect both sensitive and general populations by inducing respiratory tract irritation that can alter lung defenses, such as airway reactivity, alveolar particle clearance, and mucociliary transport (Holgate et al., 1999; Koenig, 2000). Asthmatic children and adolescents are thought to be the most sensitive groups to  $\text{H}_2\text{SO}_4$  exposure (California OEHHA, 2001; Schlesinger, 1999). Ambient sulfate levels of  $6.6 \mu\text{g}/\text{m}^3$  have been associated with decrements in pediatric lung function (Stern et al., 1989). Thurston et al. (1997) proposed that a sulfate threshold likely exists  $<5 \mu\text{g}/\text{m}^3$ .

## Methods

### Study sites

A cohort study was conducted in populated areas of Kau District that are exposed to volcanogenic air pollution. Based on results from 2003 environmental data (Longo et al., 2005), exposed cohorts of  $\text{SO}_2$  and of sulfate were selected and compared to an unexposed control cohort (Figure 3.1, Table 3.1). The  $\text{SO}_2$  cohort was in the Na`alehu area located 68 km downwind of the eruption at altitudes between 600 to 1,000 feet above sea level (a.s.l.). The Na`alehu area averaged 24.5 ppbv ( $62.6 \mu\text{g}/\text{m}^3$ ) of  $\text{SO}_2$  and was low in fine particles. The sulfate cohort was in the Ocean View area, located 74 km downwind between altitudes of 2,000 to 4,800 feet a.s.l. Ocean View averaged  $4.1 \mu\text{g}/\text{m}^3$  of particles  $\leq 0.3 \mu\text{m}$  during typical trade winds,  $9.4 \mu\text{g}/\text{m}^3$  during variable winds, and was  $<10$  ppbv in  $\text{SO}_2$ .

The unexposed control cohort was from the Hawi area on the north side of the island at altitudes between 500 to 900 ft a.s.l. The exposed and control sites have comparable meteorology, vegetation, grazing lands and crops (macadamia nuts and old sugarcane land), and no major sources of anthropogenic air pollution. The Na`alehu and Hawi areas share similar histories, both formerly old sugar plantations that brought immigration from the Philippines, Japan, and the U.S. mainland. Ocean View is a relatively new and remote subdivision of the Big Island located on an active rift of

Mauna Loa volcano (Figure 3.1), with fewer long-time residents than Na`alehu and Hawi. Most residents from Na`alehu and Ocean View work within Kau District or along the Kona coast, another area that experiences volcanic air pollution. Pahala town, the other population center in Kau, was not studied because it had agricultural production that produced visible ambient particulate matter, potentially interfering with volcanogenic health findings.

### Sampling and data collection

Health data were randomly collected between April and June 2004, during typical conditions of effusive continuous eruption at Kilauea with high SO<sub>2</sub> emissions. The total projected sample size was 330 participants, 110 per cohort, to find a doubling of risk. Participants were randomly selected by every 3<sup>rd</sup> household in the Na`alehu and Hawi areas. In Ocean view, random selection of up to two households every 3<sup>rd</sup> mile-squared block was employed below 4,000 ft. a.s.l. Inclusive sampling was used above 4,000 ft due to the sparse population. Sampling in all areas was restricted to no more than two adults per household.

Table 3.1 describes the demographics of the study participants. We restricted participation to adults aged 20 years or older with a minimum of seven years residency, thereby allowing assessment for chronic exposure pathology. The participation rate among eligible residents was 92% in Na`alehu, 98% in Ocean View, and 96% in Hawi. Assessment for migration out of the exposed area or immigration into the control area specifically for health purposes was conducted by asking exposed participants about neighbors or by asking Hawi participants directly.

Data were collected on a questionnaire by interview or by self-administration, followed by measurement of vital signs. The content of the questionnaire primarily was yes/no questions on respiratory and cardiac symptoms, diseases and illnesses that included questions from the National Health and Nutrition Examination Survey (NHANES) survey of 9,965 persons (1999-2000) (U.S.DHHS, 2003) and the American Thoracic Society (Ferris, 1978). In order to assess volcanic exposure effects, participants were instructed to answer questions for the time period of volcanic eruption (since 1983), instead of their lifetime, and while in residence in the area. For example, a case of

childhood pneumonia in a 45 year old would not be counted for pneumonia prevalence. Additional descriptive information was also collected on medication use, perceptions of health effects from the eruption, along with demographic data. The questionnaire was provided in English and Tagalo (Appendix III, IV). In Ocean View, altitude was recorded for each participant's household.

An experienced registered nurse took field vital sign measurements, at rest (5-15 minutes), of blood pressure (BP), radial pulse, and respiratory rate for each participant. For BP measurement, calibrated aneroid sphygmomanometers, placed at heart level, with proper cuff size to arm size, were employed. BP was recorded using the slow-deflation auscultory method; systolic BP was the first Korotkoff sound and diastolic pressure was the disappearance of Korotkoff sounds (U.S. National Institute of Health, 2004). For elevated systolic pressures  $\geq 160$ , two measurements were taken approximately 5 minutes apart. Radial pulses were taken for 30 seconds with regular rhythm, and a full minute with irregular rhythm.

### Statistical analysis

Data were analyzed with PEPI version 4.0 (Abramson & Gahlinger, 2001) and SPSS version 8.0 (SPSS Inc, Chicago, IL). All analyses employed a 5% probability of type I error ( $\alpha = 0.05$ ). For the cohorts, crude prevalence (P), prevalence ratio (PR) and prevalence difference (PD), with 95% confidence intervals (CI), were calculated for dichotomous variables. Measures of central tendency were calculated on ratio variables. *T*-tests were conducted on BP, pulse, and respirations with directional hypotheses between the control and exposed cohorts. Vital sign measures also were dichotomized into normal/abnormal categories and analyzed for risk ratios.

PR and PD measures, with 95% CI's, were stratified into a priori specific tables to analyze for confounding and effect modification. Stratified-specific tables included: gender, age groups (20-39 years, 40-59 years,  $\geq 60$  years), race, smoking (never, former, current), occupational dust exposure, and length of residency ( $< 20$  years versus  $\geq 20$  years, i.e. entire eruption). In Na'alehu, race was analyzed for Hawaiian, Filipino, and other. We further analyzed for any effects from style of home (plantation verses modern) based on environmental sampling findings that indicated plantation homes penetrate up to

70% of ambient SO<sub>2</sub> as opposed to modern homes with lower penetration ratios (Longo et al., 2005). In Ocean View, race was analyzed for Caucasians and others. We analyzed the cohort as a whole and then considered any altitude effects, as the environmental findings and field observation noted higher levels of fine particles at higher altitudes (Longo et al., 2005). Sub-cohorts of low altitude (2,000 to 2,950 ft.) and high altitude (3,000 to 4,500 ft.) were compared individually to the control cohort. Correlations were analyzed on BP, pulse, and respirations with altitude. For determining public health needs, a burden of additional cases was calculated for each exposed cohort area based on the 95% CI for each PD and an estimated adult population (age category:  $\geq 21$  yrs) obtained from the U.S. Census data (2000).

Three variables (cough, dry cough and sinus/nasal congestion) have known effects from certain medications that could cause or potentate these symptoms (Physician Desk Reference, 2004). Therefore, each questionnaire was screened for use of any medication with known side effects of cough (such as Angiotension-Converting Enzyme inhibitors) or sinusitis, and excluded from the PR and PD measures for these variables.

## Results

### Sulfur dioxide cohort

Exposed participants reported significantly increased prevalence of upper and lower respiratory symptoms and eye irritation (Table 3.2), diagnosed hypertension (Table 3.3), elevated blood pressure (BP), and respirations (Tables 3.4 and 3.5), relative to control participants. Rhinorrhea had an increased adjusted for length of residency prevalence of 180%. The crude estimate for sinus congestion was not significant, although significant effect was found in participants not exposed to dust [PR 3.2 (95% CI: 1.4, 7.5)]. Reported cough on most days and nocturnal dry cough were 210% and 330% more prevalent, along with phlegm on most days (160%). A negative trend ( $p_2 < 0.00$ ) with cough was identified, decreasing with increased age, and significant in all age groups. Phlegm had a positive trend ( $p_2 < 0.00$ ) with smoking status, and was significant only for former and current smokers. The largest increase in prevalence was frequent sore or dry throat (830%).

Headaches were experienced predominately by male participants [PR 5.0 (95% CI: 1.6, 16.1)] and by those with  $\geq 20$  years residency in the area [PR 2.4 (95% CI: 1.4, 4.2)]. All outdoor symptoms (cough, eye irritation, rhinorrhea, sinus congestion, SOB and wheezing) had an elevated prevalence; SO<sub>2</sub> concentration was found to be higher outside than indoors (Longo et al., 2005). Burning eyes were reported by 32.5 % of participants, a 300% increase in prevalence.

Asthma had a prevalence of 22%, not significant in this study but higher than the adult average of 7.9% for the Big Island (Hawaii BRFSS, 2002). Significant effect was identified for SO<sub>2</sub> exposed male participants with asthma [PR 3.7 (95% CI: 1.1, 12.3)] and shortness of breath (SOB) without exertion [PR 6.0 (95% CI: 1.4, 25.3)]. Wheeze most days and nights was reported twice as often and had a positive trend with increased age ( $p_2 = 0.04$ ), but was not significant. Wheeze with a cold was significant at 63% more prevalence (adjusted for style of home) as was chronic hay fever (110%).

A significant increased prevalence of 41% for diagnosed hypertension was found and supported by field-measured, elevated BP at 52 % (adjusted for style of home). Although not significant, nearly twice as many exposed smokers (50%) had elevated BP relative to controls (28%). Tables 3.5 and 3.6 present statistical differences of vital sign means between the cohorts. There was a significantly elevated mean systolic BP in the SO<sub>2</sub> cohort overall ( $p_1 = 0.04$ ), and in Filipinos ( $p_1 = 0.005$ ). Mean systolic BP was significantly elevated for exposed participants with a BMI  $< 25$  ( $p_1 = 0.001$ ). Specific comparison of SO<sub>2</sub> cohort participants to controls within the normal weight and overweight ranges were significant, however, there was no difference between those who were obese. Male SO<sub>2</sub> participants had the highest mean systolic BP at 144 mg/Hg. Elderly (65 years and older) and non-smoking elderly mean pulse rates were significantly faster by 8 beats per minute. Mean respiration rate also was significantly faster than was the control ( $p_1 = 0.04$ ).

### Sulfate aerosol cohort

Sulfate cohort participants reported similar symptoms as reported by the SO<sub>2</sub> cohort but with an overall increase in prevalence (Tables 3.2, 3.3, 3.4). Some differences between altitude sub-cohorts were identified (Tables 3.7 and 3.8). Regardless of altitude,



indoor and outdoor rhinorrhea had significantly elevated prevalence ratios (840% adjusted for length of residency and 980% crude, respectively), as did sinus congestion (160%). Prevalence of cough on most days was increased by 310%, with a slight increase in the lower altitude area. Nocturnal dry cough had increased prevalence only in the upper altitude area (240%). Phlegm, adjusted for length of residency, showed a positive trend ( $p_2 < 0.00$ ) with smoking status, and was significant in former and current smokers. The high altitude had a 380% increase compared to a 170% increase at lower altitude, both adjusted for length of residency. Frequent sore or dry throat had an increased prevalence in this cohort, at 500% for low altitude and 700% for high altitude sub-cohorts.

Outdoor burning or irritated eyes had a significant increased crude prevalence (430%) with slightly increased prevalence in low altitude participants, and weak effect modification ( $p_2 = 0.06$ ) for participants with  $\geq 20$ -year residency. Outdoor cough in the sulfate cohort was 420% more prevalent than in the control cohort. Unique to the sulfate cohort was an increased prevalence of 570% when rhinorrhea, sinus congestion and eye irritation occurred together within participants. Headache had an increased age adjusted prevalence of 57%. Chest pain was found to have increased prevalence for current smokers [PR 3.3 (95% CI: 1.1, 10.5)], those exposed to dust [PR 2.2 (95% CI: 1.1, 4.4)], non-Caucasians [PR 2.3 (95% CI: 1.2, 4.3)], and those at higher altitude (70%).

The prevalence of asthma (16.4%) was equal to the control area, yet relatively higher than the Big Island as a whole. Within the asthmatics, only 22% had a known hereditary component, half that of the control, along with a 65% prevalence of adult-onset asthma. The occurrence of wheeze within the last year was significantly more prevalent (110%), specifically at higher altitude (170%). Wheeze with a cold increased 60% relative to controls, and increased 70% at high altitude. Wheeze most days/night was significantly increased by 170%, adjusted for race, and showed a positive trend ( $p_1 = 0.03$ ) for increased age. The outdoor wheezing crude prevalence was significant (330%).

Bronchitis had an increased adjusted for race prevalence (160%) compared to the control, with the highest adjusted increase (210%) in the low altitude sub-cohort. The chronic bronchitis PR was nearly significant [PR 2.6 (95% CI: 1.0, 7.1)]. Other

respiratory and cardiac diseases were non-significant, most notably pneumonia and hay fever.

Reported diagnosed hypertension in the sulfate cohort was elevated relative to the control by 60%, adjusted for race, and 80% unadjusted in the low altitude sub-cohort. Field measured elevated BP also was more prevalent by 60%, adjusted for race, and 100% adjusted for high altitude participants. Nearly 25% of sulfate cohort participants had undiagnosed elevated BP. A difference in mean BP was found (Table 3.5 and 3.6) for those with a BMI <25 ( $p_1 = 0.02$ ). Similar to the SO<sub>2</sub> cohort, pulse rates among elderly participants 65 years and older were faster on average than controls ( $p_1 = 0.009$ ), even among non-smokers ( $p_1 = 0.01$ ). Mean respirations were faster ( $p_1 = 0.001$ ) than the control. No significant correlations between vital signs and altitude were found.

### Exposure burden

Thirty percent of the SO<sub>2</sub> cohort participants and forty percent of the sulfate cohort participants reported that the volcanic eruption had affected their health. In addition, some participants from the Hawi control area described physical reactions when visiting “Vog”-affected areas.

As reported by study participants, adult Kau residents are experiencing more health symptoms and illnesses than adult Hawi residents. Table 3.9 presents significant prevalence differences (PD) and the estimated public health burden of additional adult cases within the populations, potentially related to the long-standing volcanic eruption. Most notable are the burden estimates for increased cases of cough, bronchitis and elevated BP among Ocean View’s adult residents.

There were no reports of health-related migration out of the SO<sub>2</sub> cohort area of Na’alehu and <5 reports of neighbors leaving the sulfate cohort area of Ocean View because of the “Vog.” The control cohort of Hawi had a 4.6% reported health-related immigration.

## Discussion

Exposure to volcanogenic SO<sub>2</sub> and fine sulfate was associated with increased prevalence of adult respiratory and cardiac symptoms and diseases, along with

statistically elevated vital sign measurements. The relevancy of this study, the limitations, findings, and public health impacts are discussed, respectively.

### Relevance of the study

WHO (2001) calls for action to assess regional air pollution and the associated burden of disease, especially on vulnerable populations identified as children, fetuses, elderly, or the sick. If indeed air pollution is a major world health problem with associated increased morbidity and mortality, then populations living in active volcanic areas warrant investigation. Environmental study results (Longo et al., 2005) demonstrated poor air quality in parts of Kau district, SO<sub>2</sub> concentrations above the acute MRL (ATSDR, 1998) and the WHO annual standard (1999), along with fine sulfate aerosol concentrations above the REL for H<sub>2</sub>SO<sub>4</sub> (California OEHHA, 2001) and sulfate threshold (Thurston et al, 1997). Continued fieldwork is warranted because these air data were limited to short-term sampling, and to fine particles sizes  $\leq 0.3\mu\text{m}$  and not to particulate matter  $\leq 2.5\mu\text{m}$  (PM<sub>2.5</sub>).

Urban populations in both high and low income countries already are vulnerable to health effects from air pollution (Schwela, 2000). Many active or degassing volcanoes worldwide are proximal to urban population centers. These volcanoes potentially add to the local pollution burden. In addition, volcanic air pollution can travel great distances (1400 km) within the troposphere (Durand & Grattan, 2001), affecting even distal populations. Therefore, these health findings are applicable not only to Hawai'i, but for other degassing volcanic areas.

### Limitations of the study

The study should be interpreted within the context of several limitations including a relatively small sample size, generalizability, bias, and lack of comparability between the exposed and unexposed sites. Because of the relatively small sample size and small control prevalence, a PR less than  $\sim 2.0$  may likely have been missed, especially with the cardio-pulmonary diseases that often have lower ratio measures yet affect large numbers of individuals. Generalizability of these data is limited to chronic exposure of SO<sub>2</sub> or fine sulfate aerosol (a minimal seven years residency). Enrolling participants based on geographical exposure status and not disease status minimized selection bias. Participant

recall bias may have affected these results minimally because of memory lapse or misunderstanding of disease status. There was no medical record confirmation for these data, however, the nurse researcher did review medications, frequently by visual inspection. Prevalence may have been underestimated minimally due to a bias of healthy survivors: those with a less severe form of illness are still alive or have not moved out of the exposed area. Lastly, Ocean View is different from Hawi in ecosystem (more bare lava) and altitude, potentially affecting cardio-pulmonary findings. Altitude effects were not apparent in these data. There were no positive correlations found between vital signs and rise in altitude, as participants were long-time residents with apparent acclimatization.

### Significant findings

These results suggest that cardiac status may be affected by SO<sub>2</sub> and fine sulfate aerosol in degassing volcanic areas. Increased adjusted prevalence for field-measured high BP was found. An increase of 4.8 mm Hg in mean systolic BP was found in the SO<sub>2</sub> cohort. Examining race and gender differences, only female Filipinos were different ( $p_1 = 0.01$ ) than control counterparts with a large mean increase of 16.7 mm Hg in systolic BP. In contrast, no race or gender differences in mean BP were detected in the sulfate cohort participants. In both cohorts, an elevated systolic BP was found in the normal weight participants relative to controls, suggesting an association of BP to sulfurous air pollution unrelated to BMI cardiac risk. Elevated systolic BP has been found to be associated with exposure to SO<sub>2</sub> and particulate matter in mixed pollutant environments (Ibald-Mulli et al., 2001; Linn et al., 2001). The BP data could not directly diagnose hypertension, nor differentiate between the types of hypertension (essential verses secondary) or the underlying etiology. Furthermore, there was no assessment for pulmonary hypertension (increased pulmonary arterial pressure), a condition associated with advanced COPD (Fishman et al., 2004).

Short-term exposure to PM<sub>10</sub> has been associated with a 1-5 day delayed increase in pulse rate within a panel of elderly subjects (Pope et al., 1999). We found significant differences in resting mean pulse rate with chronically exposed elderly ( $\geq 65$  years old) participants, with and without controlling for effects from smoking (Table 3.5). SO<sub>2</sub> and

sulfate exposed elderly non-smokers had averages of 8 and 7.4 beats/minute faster pulses compared to controls.

In addition, Peters et al. (1999) noted increased electrocardiogram-measured hearts rates in 25-64 year olds and a faster rate in females during pollution episodes with SO<sub>2</sub> and suspended particles. An increase of 70 µg/m<sup>3</sup> of SO<sub>2</sub>, near Na`alehu's average, was associated with an increase in heart rate of 1.75 beats/minute (Peters et al., 1999). In the current study, SO<sub>2</sub> cohort females and non-smoking females, had increased average pulse rates relative to controls of 3.6 beats/minute and 3.9 beats/minute, respectively. No differences were found when restricting the analysis to 25-64 year olds or only females.

Figure 3.2 describes the biologically credible findings from this study, illuminating the similarities and differences between the SO<sub>2</sub> exposed and sulfate exposed cohorts. Increased prevalence of respiratory symptoms was noted in both exposed cohorts. The location of airway exposure to SO<sub>2</sub> or sulfates is dependent on nasal verses mouth breathing. High prevalence of rhinorrhea indicates the use of nasal defense mechanisms to the irritants. Breathing SO<sub>2</sub> through the nose filters out up to 99% of the gas, but it also increases nasal congestion (Koneig, 2000). If the nose becomes blocked by vascular congestion, mouth breathing may occur exposing the larynx and lower respiratory tract to the irritants (Widdicombe, 1999). The substantial prevalence of sinus/nasal congestion and rhinorrhea with sulfate exposure reflects an overload of these defense mechanisms. Increased prevalence of cough and phlegm on most days is likely related to reflex mechanisms effective at absorbing and neutralizing the irritant, and clearing the large airways (Widdicombe, 1999). A frequent sore/dry throat may reflect irritation and a drying effect on the airway. All outdoor symptoms (cough, wheeze, SOB, rhinorrhea, and eye irritation) had elevated prevalence as would be expected given SO<sub>2</sub> or sulfate aerosol exposure. Eye irritation suggests a dermal reaction to both sulfur species.

Specific to chronic SO<sub>2</sub> exposure were increased prevalence of dry cough and hay fever in participants, indicating possible gas sensitivity reactions. Specific to chronic sulfate exposure were increased wheeze symptoms and bronchitis in the participants, suggesting repeated insults to lower airways with bronchial defenses and inflammatory processes. Most intriguing was the high prevalence, relative to controls, of rhinorrhea,

sinus congestion and burning eyes together in sulfate participants, suggesting the presence of a volcanic “Vog” sulfate syndrome.

<b><u>HEALTH EFFECTS</u></b>		
<b><i>TOTAL EXPOSED:</i></b>		
<b>SYSTEMIC</b>	headache hypertension ↑ blood pressure ↑ pulse in elderly	<b><i>SO<sub>2</sub> SPECIFIC:</i></b> dry cough hay fever
<b>DERMAL</b>	eye irritation burning	
<b>UPPER RESPIRATORY</b>	rhinorrhea cough sore/dry throat phlegm	<b><i>SULFATE SPECIFIC:</i></b> sinus congestion wheezing bronchitis
<b>LOWER RESPIRATORY</b>	wheeze with colds SOB outdoors ↑ respirations	

Figure 3.2. Health effects associated with chronic exposure to volcanogenic air pollution. These effects were reported by study participants with  $\geq 7$  year residency in Kau District.

Current and past smokers may experience more detrimental health effects than non-smokers from volcanic air pollution. In a cohort study of power-station workers, with an averaged shift exposure of 800 ppbv SO<sub>2</sub>, researchers found an increase in cough, especially in smokers, and sputum production compared to non-exposed workers (Froom et al., 1998). The current study, with substantially less SO<sub>2</sub>, found no effect for cough with smoking, but identified a positive trend in reported phlegm. The SO<sub>2</sub> cohort showed an increasing effect in reported phlegm relative to controls: 80% in never smokers, 194% with former smokers and 257% in current smokers. The sulfate cohort showed analogous increases relative to smoking status: 17%, 197% and 261%, respectively. Repeated irritation from smoking combined with air pollution perpetuates chronic inflammatory processes that lead to lung disease (Jeffery, 1999).

Smoking often is associated with chronic bronchitis. Although reported chronic bronchitis was not significant [PR = 2.6(CI 95%: 0.96, 7.0)], this clinically diagnosed disease may actually be more prevalent, regardless of smoking status. SO<sub>2</sub> exposed participants had 420% increased prevalence [PR = 5.2 (CI 95%: 1.6, 17.1)] and sulfate participants had 670% [PR = 7.7 (CI 95%: 2.4, 24.6)] of combined cough with phlegm on most days for 3 consecutive months or more during the year.

Most important, this study found similar health effects as historically reported in distant Europe after the 1783 eruption of Laki volcano in Iceland: eye sensitivity, sore throat, bronchitis, headache and asthma-like exacerbations (Durand & Grattan, 2001). Bell and Davis (2001) asserted higher mortality than previously thought from delayed infectious respiratory disease with the London Fog of 1952 (SO<sub>2</sub> and black smoke). No increased prevalence of pneumonia in the exposed cohorts, however, was found in the current study.

### Public health concerns

Further investigations, health screening and public health interventions are indicated in all downwind areas of Kilauea Volcano. Prior to this study, focus was placed on investigating acute responses to "Vog" in asthmatics. A broader general population focus, however, now is indicated using the associations and risks identified in this study.

Pope et al. (2004) clarified that chronic PM exposures are associated with mortality from ischemic heart disease, dysrhythmias, heart failure and cardiac arrest. An 8% to 18% increased mortality from these diseases were found with a 10 $\mu$ g/m<sup>3</sup> elevation in PM<sub>2.5</sub>, with a larger risk to smokers (Pope et al., 2004). In the current study, higher than average rates of lifestyle cardiac risk factors (Table 3.1), diagnosed hypertension, and elevated BP were identified regardless of exposure status. In Ocean View alone, a potential burden from exposure to sulfate aerosol of 102 to 580 residents could have high BP and be in need of medical and nursing intervention. In the control site of Hawi, 74% of participants were overweight or obese ( $\geq 25$  Body Mass Index). The most important risk factor for cardiovascular disease is systolic BP greater than 140 mm Hg in people over 50 years of age (U.S. NIH, 2003), a guideline exceeded by 46% of all  $\geq 50$  year old study participants. Pre-hypertension was measured in 31% of all 335 participants. The

increased prevalence of cardio-pulmonary symptoms and elevated risk factors identified, together with elevated concentrations of air pollution in Kau District, pose a public health intervention opportunity for Big Island residents.

Exposure to SO<sub>2</sub> also has been found to adversely affect another WHO-identified special population- the fetus. A recent finding suggests that SO<sub>2</sub> levels as low as 11.4 ppbv have a 26% increased odds of an infant being below 2500 grams at birth compared to an exposure of <7.1 ppbv (Lin et al., 2004). In another study on infant births from 1985-1998 in Vancouver, Canada, an increase of 5 ppbv of SO<sub>2</sub> was associated with a 9% increased odds for pre-term birth, and a 7% increased odds for intrauterine growth retardation (Liu et al., 2003). The average SO<sub>2</sub> concentration during the study was 4.9 ppbv, with an average maximum of 13.4 ppbv (Liu et al., 2003). These studies suggest that average SO<sub>2</sub> levels near the acute MRL of 10ppbv are associated with adverse birth outcomes. Important to note, however, is that both of these studies were in mixed-pollutant environments, different from the Kau District where air pollution is primarily volcanic SO<sub>2</sub> and fine sulfate aerosols.

## **Conclusions**

After over 22 years of nearly continuous eruption at Kilauea Volcano, this study offers the first population-based prevalence measures of adult health effects associated with volcanogenic air pollution. Increased prevalence of cardio-pulmonary symptoms and diseases in adults were found associated with chronic exposure to SO<sub>2</sub> and fine sulfate aerosols. The threat of further eruption remains in Hawai'i and at other volcanoes near cities worldwide. Therefore, further investigation and intervention in volcanic areas is vital to the health of these affected populations.

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Table 3.1. Study Participant Demographics

The Participants	Control Cohort Hawi	Total Exposed Cohort	SO <sub>2</sub> Exposed Na'alehu	Sulfate Exposed Ocean View
Area population ≥21 yrs * (N)	633	-	603	1,593
Study participants (N)	110	225	115	110
Age in yrs (average, StD.)	52.1 (17.4)	57.2 (16.2)	56.4 (18.9)	57.9 (12.9)
Age Groups:	%	%	%	%
▪ 20-39 yrs.	27.3	17.3	25.2	9.1
▪ 40-59 yrs	34.5	34.2	21.7	47.3
▪ 60 and older	38.2	48.4	53.1	43.6
Gender: Female	58	58	60	56
Male	42	42	40	44
Ethnicity: Hawaiian	27.3	21.3	26.1	16.4
Filipino	23.6	23.1	40.9	4.5
Japanese	16.4	11.1	20.0	1.8
Caucasian	17.3	38.6	6.1	72.7
Other	15.5	5.7	7.0	4.5
Resident for ≥20 yrs Full length of eruption	72	55.1	86.1	22.7
Born in Hawai'i	77.3	46.2	68.7	22.7
Born in the Philippines	8.2	14.6	27.0	1.8
Old plantation-style home	39.1	32.8	64.3	0
Employment: working	60.0	44.0	43.5	44.5
▪ Unemployed	8.2	19.2	22.6	15.5
▪ Retired	29.1	32	32.2	31.8
▪ Disabled	2.7	4.8	1.7	8.2
Smoking history (past/current)	56.4	62.6	54.8	70.9
Current smokers	22.7	22.6	18.3	27.3
Hx. Sugar cane work	12.7	10.6	20.0	0.9
Hx. Dust exposure	46.4	46.6	51.8	41.8
BMI %: underweight <18.5	0.9	4.0	5.2	2.8
▪ normal 18.5- 24.9	25.5	43.5	39.1	47.7
▪ overweight 25- 29.9	46.4	28.3	28.7	27.5
▪ obesity ≥30	27.3	24.2	26.1	22.0
▪ over + obese ≥25	73.7	52.5	54.8	49.5
Health insurance	94.5	81.7	86.1	77.3
Subjective view of health				
▪ Excellent	10.9	16.0	6.1	26.4
▪ Very good	41.8	34.6	33.9	35.5
▪ Average	44.5	42.2	53.0	30.9
▪ Worse	2.7	5.7	5.2	6.4
▪ Very poor	0	1.3	1.7	0.9

Study Participant: 20 years or older, minimum of 7 years residency in cohort areas.

Data given in percentages unless specified.

\*An estimated area adult population, category: 21 yrs. and older (U.S.Census, 2000).

Table 3.2. Prevalence (P) and Prevalence Ratios (PR) of Symptoms

Symptoms	Control		SO <sub>2</sub> cohort			Sulfate cohort		
	P	%	P	%	PR (95% C.I.)	P	%	PR (95% C.I.)
Cough on most days	11/94	11.7	37/103	35.9	<b>*3.1 (1.7-5.7)</b>	47/98	48.0	<b>*4.1 (2.3-7.4)</b>
Dry cough at night	6/94	6.4	28/102	27.5	<b>*4.3 (1.9-9.9)</b>	15/98	15.3	<b>*2.2 (0.8-6.0)</b>
Sinus/nasal congestion often	17/94	18.1	25/100	25.0	<b>*1.4 (0.8-2.4)</b>	44/93	47.3	<b>*2.6 (1.6-4.2)</b>
Wheeze within last 12 months	17/110	15.5	24/115	20.9	1.4 (0.8-2.4)	36/110	32.7	<b>2.1 (1.3-3.5)</b>
▪ most days/nights	8/110	7.3	17/115	14.8	2.0 (0.9-4.5) <sup>B</sup>	17/110	15.5	<b>2.8 (1.2-6.9)<sup>A</sup></b>
▪ with a cold	23/110	20.9	41/115	35.7	<b>1.6 (1.0-2.6)<sup>D</sup></b>	37/110	33.6	<b>1.6 (1.03-2.5)</b>
▪ exercise induced	9/110	8.2	16/115	13.9	1.7 (0.8-3.7)	13/110	11.8	1.4 (0.6-3.2)
Phlegm on most days	12/110	10.9	32/115	27.8	<b>2.6 (1.4-4.7)</b>	32/110	29.1	<b>3.7 (1.9-7.4)<sup>B</sup></b>
Chest colds	56/104	53.8	48/115	41.7	0.8 (0.6-1.0)	52/106	49.1	1.0 (0.7-1.4) <sup>A</sup>
SOB without exertion	11/110	10.0	21/114	18.4	1.8 (0.9-3.6)	18/109	16.5	1.7 (0.8-3.3)
Chest pains (ever have)	22/110	20.0	31/115	27.0	1.4 (0.8-2.2)	28/110	25.5	1.3 (0.8-2.1)
Sore/dry throat (frequent)	3/110	2.7	29/115	25.2	<b>9.3 (2.9-29.5)</b>	21/110	19.1	<b>7.0 (2.2-22.8)</b>
Runny nose (frequent)	6/110	5.5	19/115	16.5	<b>2.8 (1.1-6.8)<sup>B</sup></b>	37/110	33.6	<b>8.4 (3.3-21.7)<sup>B</sup></b>
Head aches (often)	25/110	22.7	43/115	37.4	<b>1.7 (1.1-2.5)</b>	33/109	30.9	<b>1.6 (1.0-2.4)<sup>C</sup></b>
Outdoor cough	5/109	4.6	16/114	14.0	<b>3.1 (1.2-8.1)</b>	26/110	23.6	<b>5.2 (2.1-12.9)</b>
Outdoor eyes burning, irritation	9/110	8.2	37/114	32.5	<b>4.0 (2.0-7.8)</b>	48/110	43.6	<b>5.3 (2.8-10.3)</b>
Outdoor runny nose, congestion	4/110	3.6	17/114	14.9	<b>4.1 (1.4-11.8)</b>	39/110	35.5	<b>9.8 (3.6-26.4)</b>
Outdoor SOB	4/110	3.6	17/114	14.9	<b>4.1 (1.4-11.8)</b>	14/110	12.7	<b>3.5 (1.2-10.3)</b>
Outdoor wheezing	3/110	2.7	13/115	11.3	<b>4.1 (1.2-14.2)</b>	13/110	11.8	<b>4.3 (1.3-14.8)</b>
Cluster of runny nose, sinus congestion & burning eyes	3/110	2.7	9/115	7.8	2.9 (0.8-10.3)	20/110	18.1	<b>6.7 (2.0-21.8)</b>

Bold associations are statistically significant at  $p < 0.05$ .

Prevalence ratios (PR) are un-confounded crude rate ratios unless indicated:

\* PR restricted to participants not taking medications with known side effects

A = adjusted PR for race B = adjusted PR for length of residency

C = adjusted PR for age D = adjusted PR for home type (plantation/modern)

Table 3.3. Prevalence (P) and Prevalence Ratios (PR) of Illness and Disease

Disease/Illnesses (medical Dx)	Control		SO <sub>2</sub> cohort			Sulfate cohort		
	P	%	P	%	PR (95% C.I.)	P	%	PR (95% C.I.)
Asthma	18/110	16.4	25/115	21.7	1.2 (0.7-2.0) <sup>D</sup>	18/110	16.4	1.0 (0.6-1.8)
asthma type: pediatric adult-onset	10/17	58.8	14/25	56.0	1.0 (0.6-1.6)	7/18	38.9	0.7 (0.3-1.3)
	7/17	41.2	11/25	44.0	1.1 (0.5-2.2)	11/18	61.1	1.5 (0.8-2.9)
asthma attacks ≥1 year	7/18	38.8	11/25	44.0	1.1 (0.6-2.3)	6/18	33.3	0.9 (0.4-2.1)
asthma in parents/family	8/18	44.4	9/25	36.0	0.8 (0.4-1.7)	4/18	22.2	0.5 (0.2-1.4)
Bronchitis (within 21 yrs)	27/109	24.5	29/115	25.2	1.0 (0.7-1.6)	49/109	45.0	<b>2.6 (1.6-4.3)<sup>A</sup></b>
chronic bronchitis	5/110	4.5	11/115	9.6	2.1 (0.8-5.9)	13/110	11.8	2.6 (1.0-7.0)
Emphysema	3/110	2.7	3/114	2.6	1.0 (0.2-4.7)	2/110	1.8	0.7 (0.1-3.9)
COPD	3/110	2.7	3/114	2.6	1.0 (0.2-4.7)	6/110	5.5	2.0 (0.5-7.8)
hayfever/allergies	26/110	23.6	32/115	27.8	1.2 (0.8-1.8)	30/110	27.3	1.2 (0.7-1.8)
chronic hayfever	10/110	9.0	22/115	19.1	<b>2.1 (1.0-4.2)</b>	19/110	17.3	1.9 (0.9-3.9)
sinusitis (chronic)	14/110	12.7	14/114	12.3	1.0 (0.5-1.9)	21/110	19.1	1.5 (0.8-2.8)
pneumonia (within 21 yrs)	21/110	19.1	13/115	11.3	0.6 (0.3-1.2) <sup>C</sup>	21/110	19.1	1.0 (0.6-1.7)
coronary artery disease	8/110	7.3	8/115	7.0	1.0 (0.4-2.5)	10/110	9.1	1.6 (0.6-4.5) <sup>A</sup>
myocardial infarction	3/110	2.7	6/114	5.3	1.9 (0.5-7.5)	8/110	7.3	2.7 (0.7-9.8)
Angina	4/110	3.6	9/115	7.8	2.2 (0.7-6.8)	9/110	8.2	2.3 (0.7-7.1)
congestive heart failure	3/110	2.7	4/115	3.5	1.3 (0.3-5.6)	7/110	6.4	2.3 (0.6-8.8)
Hypertension	36/110	32.7	53/115	46.1	<b>1.4 (1.01-2.0)</b>	41/110	37.3	<b>1.6 (1.0-2.5)<sup>A</sup></b>
cerebral vascular accident	1/110	0.9	4/115	3.5	3.8 (0.4-33.7)	7/110	6.4	7.0 (0.9-56.0)
Blood pressure meds	30/110	27.3	39/114	34.2	1.3 (0.8-1.9)	33/110	30.0	1.1 (0.7-1.7)
Respiratory meds	16/110	14.5	16/114	14.0	1.0 (0.5-1.8)	21/110	19.1	1.3 (0.7-2.4)

Bold associations are statistically significant at  $p < 0.05$ .

Prevalence ratios (PR) are un-confounded crude rate ratios unless indicated:

A = adjusted PR for race

C = adjusted PR for age

D = adjusted PR for home type (plantation/modern)

Table 3.4. Prevalence (P) and Prevalence Ratios (PR) of Abnormal Vital Sign Measurements

Vital Signs	Control		SO <sub>2</sub> cohort			Sulfate cohort		
	P	%	P	%	PR (95% C.I.)	P	%	PR (95% C.I.)
<b>Regular pulse <math>\geq 90/\text{min}</math></b>	17/105	16.2	17/106	16.0	1.0 (0.5-1.8)	22/103	21.3	1.3 (0.7-2.3)
▪ Non-smokers	9/81	11.1	12/86	14.0	1.3 (0.6-2.8)	11/73	15.1	1.4 (0.6-3.1)
▪ Smokers	8/24	33.3	5/20	25.0	0.8 (0.3-1.9)	11/30	36.6	1.1 (0.5-2.3)
<b>BP <math>\geq 140</math> or <math>\geq 90</math></b>	43/110	39.1	59/110	53.6	<b>1.5 (1.1-2.0)<sup>D</sup></b>	57/110	51.8	<b>1.6 (1.1-2.3)<sup>A</sup></b>
▪ Undiagnosed	21/110	19.1	25/110	22.7	1.3 (0.6-2.5)	27/110	24.5	1.3 (0.8-2.1)
▪ Non-medicated	27/80	33.7	33/72	45.8	1.4 (0.9-2.0)	35/77	45.5	1.4 (0.9-2.0)
▪ BP meds	16/30	53.3	26/38	68.4	1.3 (0.9-1.9)	22/33	66.6	1.3 (0.8-1.9)
▪ Never smoked	18/47	38.2	27/49	55.1	1.4 (0.9-2.2)	15/32	46.9	1.2 (0.7-2.1)
▪ Former smokers	18/38	47.4	22/41	53.7	1.1 (0.7-1.8)	27/48	56.3	1.2 (0.8-1.8)
▪ Current smokers	7/25	28.0	10/20	50.0	1.8 (0.8-3.8)	15/30	50.0	1.8 (0.9-3.7)
<b>Respirations <math>\geq 20/\text{min}</math></b>	21/110	19.1	19/109	17.4	0.9 (0.5-1.6)	31/110	28.2	1.5 (0.9-2.4)

Bold associations are statistically significant at  $p < 0.05$ .

Prevalence ratios (PR) are un-confounded crude rate ratios unless indicated:

A = adjusted PR for race

D = adjusted PR for home type (plantation/modern)

Table 3.5. Statistical Analysis of Vital Sign Measurements:  
Exposed SO<sub>2</sub> and Sulfate Cohorts verses Control Cohort

Vital Signs	Control			SO <sub>2</sub> cohort				Sulfate cohort			
	Mean	StD.	N	Mean	StD.	N	P <sub>(1)</sub> value	Mean	StD.	N	P <sub>(1)</sub> value
<b>Pulse (normal rhythm)</b>	77.0	11.7	105	79.2	12.7	106	0.10	78.7	12.7	103	0.15
▪ Non-smokers	75.5	11.2	81	78.5	12.8	86	0.06	76.5	11.8	73	0.30
▪ Elderly ≥65 yrs	71.2	9.7	22	78.8	12.6	40	<b>0.009</b>	79.7	13.2	25	<b>0.009</b>
▪ Elderly non-smokers	70.2	8.8	21	78.2	12.2	38	<b>0.005</b>	77.6	11.9	20	<b>0.01</b>
▪ Males	79.2	13.2	43	79.4	12.3	42	0.94	79.0	12.5	44	0.94
▪ Females	75.5	10.3	62	79.1	13.0	64	<b>0.045</b>	78.6	12.9	59	0.08
▪ Female non-smokers	73.9	8.8	52	77.9	13.0	52	<b>0.04</b>	75.0	11.0	43	0.31
<b>Systolic BP</b>	130.2	19.6	110	135.0	20.6	110	<b>0.04</b>	132.0	20.3	110	0.25
▪ Non-medicated	127.3	18.3	80	130.8	19.8	72	0.13	128.5	20.9	77	0.35
▪ Males	137.4	21.3	46	144.1	19.1	45	0.06	135.6	17.8	48	0.33
▪ Females	125.0	16.6	64	128.6	19.2	65	0.13	129.2	21.8	62	0.12
▪ Hawaiians	129.9	16.9	30	128.0	18.5	27	0.35	132.3	20.7	18	0.33
▪ Filipinos	125.9	22.1	26	139.0	22.4	45	<b>0.005</b>	*	*	*	*
▪ Caucasians	124.3	20.2	19	*	*	*	*	131.6	20.7	80	0.09
<b>Diastolic BP</b>	82.6	10.6	110	82.8	13.2	110	0.47	83.8	10.5	110	0.21
▪ Non-medicated	82.4	10.3	80	82.6	12.8	72	0.48	84.0	9.8	77	0.17
▪ Males	84.8	11.5	46	87.0	14.6	45	0.22	84.3	11.2	48	0.16
▪ Females	81.1	9.8	64	79.8	11.4	65	0.26	83.4	10.1	62	0.10
▪ Hawaiians	86.3	9.1	30	86.6	12.4	27	0.46	84.6	10.4	18	0.28
▪ Filipinos	82.6	12.4	26	83.7	13.5	44	0.37	*	*	*	*
▪ Caucasians	78.6	9.9	19	*	*	*	*	82.9	10.5	80	0.06
<b>Respirations</b>	16.1	2.8	110	16.7	2.2	109	<b>0.04</b>	17.2	2.4	110	<b>0.001</b>

Bold p-values: independent *t*-test, directional hypothesis, statistical significance at  $p < 0.05$ .

\*Too few participants in study

Table 3.6. Statistical Analysis of Body Mass Index (BMI) to Systolic Blood Pressure:  
Exposed SO<sub>2</sub> and Sulfate Cohorts verses Control Cohort

Vital Signs	Control			SO <sub>2</sub> cohort				Sulfate cohort			
	Mean	StD.	N	Mean	StD.	N	P <sub>(1)</sub> value	Mean	StD.	N	P <sub>(1)</sub> value
<b>Systolic BP</b>	130.2	19.6	110	135.0	20.6	110	<b>0.04</b>	132.0	20.3	110	0.25
▪ BMI <25	117.2	16.1	29	131.6	20.8	50	<b>0.001</b>	125.31	17.8	55	<b>0.02</b>
▪ BMI ≥25	134.81	18.8	81	137.6	20.2	59	0.20	138.1	20.4	54	0.17
▪ Underweight	116.0	*	1	137.3	25.5	6	*	138.67	23.2	3	*
▪ Normal weight	117.3	16.4	28	130.8	20.3	44	<b>0.002</b>	124.5	17.4	52	<b>0.04</b>
▪ Overweight	131.7	18.3	51	139.1	20.0	31	<b>0.04</b>	131.2	19.5	30	0.46
▪ Obese	140.2	18.6	30	135.9	20.6	28	0.20	146.8	18.2	24	0.10

Bold p-values: independent *t*-test, directional hypothesis, statistical significance at  $p < 0.05$ .

\*Too few participants in study

Table 3.7. Prevalence (P) and Prevalence Ratios (PR) of Symptoms: Low and High Altitude Sulfate sub-cohorts.

Symptoms	Control cohort P %	Sulfate cohort Total		Low 2,000 – 2,950 ft		High 3,000 – 4,800 ft	
		P %	PR (95% C.I.)	P %	PR (95% C.I.)	P %	PR (95% C.I.)
Cough on most days	11.7	48.0	<b>*4.1 (2.3-7.4)</b>	49.1	<b>4.4 (2.3-8.1)*</b>	45.5	<b>3.9 (2.1-7.3)*</b>
Dry cough at night	6.4	15.3	<b>*2.2 (0.8-6.0)</b>	12.7	<b>1.3 (0.4-4.5)*</b>	20.0	<b>3.4 (1.3-8.6)*</b>
Sinus/nasal congestion often	18.1	47.3	<b>*2.6 (1.6-4.2)</b>	45.5	<b>2.6 (1.5-4.4)*</b>	45.5	<b>2.6 (1.5-4.4)*</b>
Wheeze within last 12 months	15.5	32.7	<b>2.1 (1.3-3.5)</b>	23.6	1.5 (0.8-2.9)	41.8	<b>2.7 (1.6-4.6)</b>
▪ most days/nights	7.3	15.5	<b>2.8 (1.2-6.9)<sup>A</sup></b>	12.7	1.8 (0.7-4.6)	18.2	2.9 (0.9-8.9) <sup>A</sup>
▪ with a cold	20.9	33.6	<b>1.6 (1.03-2.5)</b>	30.9	1.5 (0.9-2.5)	36.4	<b>1.7 (1.1-2.9)</b>
▪ exercise induced	8.2	11.8	1.4 (0.6-3.2)	9.1	1.1 (0.4-3.2)	14.5	1.8 (0.7-4.4)
Phlegm on most days	10.9	29.1	<b>3.7 (1.9-7.4)<sup>B</sup></b>	21.8	<b>2.7 (1.1-6.4)<sup>B</sup></b>	36.4	<b>4.8 (2.3-10.1)<sup>B</sup></b>
Chest colds	53.8	49.1	1.0 (0.7-1.4) <sup>A</sup>	43.4	0.9 (0.6-1.4) <sup>A</sup>	54.7	1.0 (0.8-1.3)
SOB without exertion	10.0	16.5	1.7 (0.8-3.3)	12.7	1.3 (0.5-3.1)	20.4	2.0 (0.9-4.4)
Chest pains (ever have)	20.0	25.5	1.3 (0.8-2.1)	16.4	0.8 (0.4-1.7)	34.5	<b>1.7 (1.0-2.9)</b>
Sore/dry throat (frequent)	2.7	19.1	<b>7.0 (2.2-22.8)</b>	16.4	<b>6.0 (1.7-21.3)</b>	21.8	<b>8.0 (2.4-27.2)</b>
Runny nose (frequent)	5.5	33.6	<b>8.4 (3.3-21.7)<sup>B</sup></b>	36.4	<b>8.7 (2.9-26.0)<sup>B</sup></b>	30.9	<b>8.3 (3.0-23.2)<sup>B</sup></b>
Head aches (often)	22.7	30.9	<b>1.6 (1.0-2.4)<sup>C</sup></b>	25.5	1.1 (0.6-2.0)	54.3	1.6 (0.9-2.6)
Outdoor cough	4.6	23.6	<b>5.2 (2.1-12.9)</b>	20.0	<b>4.4 (1.6-11.9)</b>	27.3	<b>6.0 (2.3-15.5)</b>
Outdoor eyes burning, irritation	8.2	43.6	<b>5.3 (2.8-10.3)</b>	47.3	<b>5.8 (2.9-11.5)</b>	40.0	<b>4.9 (2.4-9.9)</b>
Outdoor runny nose, congestion	3.6	35.5	<b>9.8 (3.6-26.4)</b>	36.4	<b>10.0 (3.6-27.8)</b>	34.5	<b>9.5 (3.4-26.6)</b>
Outdoor SOB	3.6	12.7	<b>3.5 (1.2-10.3)</b>	10.9	3.0 (0.9-10.2)	14.5	<b>4.0 (1.3-12.7)</b>
Outdoor wheezing	2.7	11.8	<b>4.3 (1.3-14.8)</b>	10.9	<b>4.0 (1.0-15.4)</b>	12.7	<b>4.7 (1.3-17.4)</b>
Cluster of runny nose, sinus congestion & burning eyes	2.7	18.1	<b>6.7 (2.0-21.8)</b>	20.0	<b>7.3 (2.1-25.2)</b>	16.4	<b>6.0 (1.7-21.3)</b>

Bold associations are statistically significant at  $p < 0.05$ .

Prevalence ratios (PR) are un-confounded crude rate ratios unless indicated:

\* PR restricted to participants not taking medications with known side effects

A = adjusted PR for race    B = adjusted PR for length of residency    C = adjusted PR for age

Table 3.8. Prevalence (P) and Prevalence ratios (PR) of Illness and Disease:  
Comparison of Low Altitude to High Altitude Sulfate sub-cohorts.

Disease/Illnesses (medically diagnosed)	Control cohort P %	Sulfate cohort Total		Low Altitude 2,000 – 2,950 ft		High Altitude 3,000 – 4,800 ft	
		P %	PR (95% C.I.)	P %	PR (95% C.I.)	P %	PR (95% C.I.)
Asthma	16.4	16.4	1.0 (0.6-1.8)	16.4	1.0 (0.5-2.1)	16.4	1.0 (0.5-2.1)
Bronchitis (within 21 yrs)	24.5	45.0	<b>2.6 (1.6-4.3)<sup>A</sup></b>	43.6	<b>3.1 (1.8-5.4)<sup>A</sup></b>	46.3	<b>1.9 (1.2-2.9)</b>
chronic bronchitis	4.5	11.8	2.6 (1.0-7.0)	10.9	2.4 (0.8-7.5)	12.7	2.8 (0.9-8.4)
hayfever/allergies	23.6	27.3	1.2 (0.7-1.8)	27.3	1.2 (0.7-2.0)	27.3	1.2 (0.7-2.0)
chronic hayfever	9.0	17.3	1.9 (0.9-3.9)	18.2	2.0 (0.9-4.5)	16.4	1.8 (0.8-4.2)
sinusitis (chronic)	12.7	19.1	1.5 (0.8-2.8)	14.5	1.1 (0.5-2.6)	23.6	1.9 (0.9-3.7)
pneumonia (within 21 yrs)	19.1	19.1	1.0 (0.6-1.7)	20.0	1.0 (0.5-2.0)	18.2	1.0 (0.5-1.9)
Hypertension	32.7	37.3	<b>1.6 (1.0-2.5)<sup>A</sup></b>	40.0	<b>1.8 (1.1-3.2)</b>	34.5	1.5 (0.8-2.8)
<b>Field measurements:</b>							
BP: $\geq 140$ or $\geq 90$	39.1	51.8	<b>1.6 (1.1-2.3)<sup>A</sup></b>	43.6	1.3 (0.8-2.1) <sup>A</sup>	60.0	<b>2.0 (1.3-2.9)<sup>A</sup></b>
Regular pulse: $\geq 90$ /min	16.2	21.3	1.3 (0.7-2.3)	17.3	1.1 (0.5-2.2)	23.6	1.5 (0.8-2.8)
▪ Non-smokers	11.1	15.1	1.4 (0.6-3.1)	8.1	0.7 (0.2-2.5)	20.0	1.8 (0.8-4.3)
Respirations: $\geq 20$ /min	19.1	28.2	1.5 (0.9-2.4)	27.3	1.4 (0.8-2.6)	29.1	1.5 (0.9-2.7)

Bold associations are statistically significant at  $p < 0.05$ .

Prevalence ratios (PR) are un-confounded crude rate ratios unless indicated:

A = adjusted PR for race



Table 3.9. Estimated Burden of Adult Cases:  
Significant Prevalence Differences (PD) of Symptoms, Illnesses, Diseases

Symptoms/diseases	SO <sub>2</sub> cohort		Sulfate cohort	
	P.D. per 100 (95% C.I.)	Burden of cases	P. D. per 100 (95% C.I.)	Burden of cases
Cough on most days	24.2 (11.9-36.6)*	72 - 221	36.3 (23.4-49.1)*	373 - 782
Dry cough at night	21.1 (10.1-32.1)*	61 - 194	-	-
Sinus/nasal congestion (often)	-	-	29.2 (15.4-43.1)*	245 - 686
Wheeze within last 12 months	-	-	17.3 (5.3-29.3)	84 - 467
▪ with a cold	13.5 (1.5-25.5) <sup>D</sup>	9 - 154	12.7 (0.2-25.3)	3 - 403
Phlegm on most days	16.9 (6.0-27.9)	36 - 168	18.2 (7.0-29.4)	111 - 468
Sore/dry throat (frequent)	22.5 (13.1-31.9)	79 - 192	16.4 (7.5-25.2)	119 - 401
Runny nose (frequent)	11.1 (2.2-20.0)	13 - 121	28.2 (17.5-38.9)	279 - 619
Head aches (often)	14.7 (2.0-27.3)	12 - 165	-	-
Outdoor cough	9.5 (1.1-17.8)	7 - 107	19.0 (9.3-28.8)	148 - 459
Outdoor eye burning, irritation	24.3 (13.4-35.2)	81 - 212	35.5 (24.0-47.0)	382 - 748
Outdoor runny nose & congestion	11.3 (3.0-19.6)	18 - 118	31.8 (21.3-42.3)	339 - 673
Outdoor SOB	11.3 (3.0-19.6)	18 - 118	9.1 (1.0-17.1)	17 - 272
Outdoor wheezing	8.6 (1.2-16.0)	7 - 97	9.1 (1.4-16.8)	22 - 286
Cluster of nose, sinus & eyes	-	-	15.5 (6.7-24.2)	107 - 385
bronchitis (within 20 yrs)	-	-	30.8 (17.3-44.3) <sup>A</sup>	275 - 705
chronic hayfever	10.0 (0.2-19.9)	1 - 120	-	-
hypertension	-	-	14.9 (0.6-29.3) <sup>A</sup>	10 - 467
Field measured BP $\geq 140$ or $\geq 90$	14.6 (0.6-28.5)	4 - 172	21.4 (6.4-36.4) <sup>A</sup>	102 - 580

Burden of cases = estimated minimum and maximum number of additional adult cases in each area due to exposure (95% C.I. of the PD x total adult population of area; given relatively equal exposure).

- PD was not significant, no estimated burden of cases.

\* PD restricted to participants not taking medications with known side effects.

D = adjusted PR for home type (plantation/modern)

**THE HUMAN HEALTH RESPONSE TO VOLCANIC AIR POLLUTION:  
CHARACTERIZATION OF A VOLCANIC SULFUR SYNDROME AND  
RECOMMENDATIONS FOR HEALTH PROMOTION**

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Pending submission 2005

**Abstract**

The human-environmental interaction in a volcanic setting was qualitatively described in an adult population living downwind of Kilauea Volcano, Hawai'i, USA. Previous environmental sampling in Kau District revealed high ambient concentrations of sulfur dioxide and fine sulfate particles, along with epidemiologic data that revealed high prevalence ratios of respiratory and cardiac symptoms. Qualitative descriptions of human responses to the eruption were obtained from informants by open-ended questions and unstructured interviews in the natural setting. These findings suggest that the human-health response to chronic exposure of volcanogenic air pollution may result in a sulfur illness syndrome with primary and secondary characteristics.

## Introduction

Throughout recorded history humans have been challenged to interact with volcanoes. Over the last millennium, the increase in world population spurred migrations into Earth's volcanic areas. In any given year, about 50 volcanoes erupt (Press & Siever, 2002) with two to four eruptions resulting in deaths (Simkin et al., 2001). Today, nearly 500 million people are currently at risk from the world's active volcanoes (Baxter et al., 1999). Knowledge about health effects and human responses associated with volcanic activity, however, is sparse.

In this study, human health responses are qualitatively described from an adult population chronically exposed to volcanogenic air pollution. These findings provide the first characterization of volcanic sulfur syndrome identified from environmental, epidemiological, and qualitative findings.

## Background

Kilauea Volcano, on the Big Island of Hawai'i, USA, is one of the world's most active volcanoes (Figure 4.1). The current eruption began in 1983 and is considered the longest-lived in the last 600 years (Heliker & Brantley, 2003). Lava flows and a gaseous plume are effusively released from a site on the East Rift of the volcano. Kilauea is the largest point source for sulfur dioxide (SO<sub>2</sub>) in the United States with daily emissions averaging 1,600 tons (Sutton & Elias, 2002). The historical record shows that few deaths have been attributed to Kilauea's eruptions. The long-term degassing of SO<sub>2</sub> on downwind populations, however, may have attributed to an insidious, elevated morbidity of cardio-pulmonary disease.

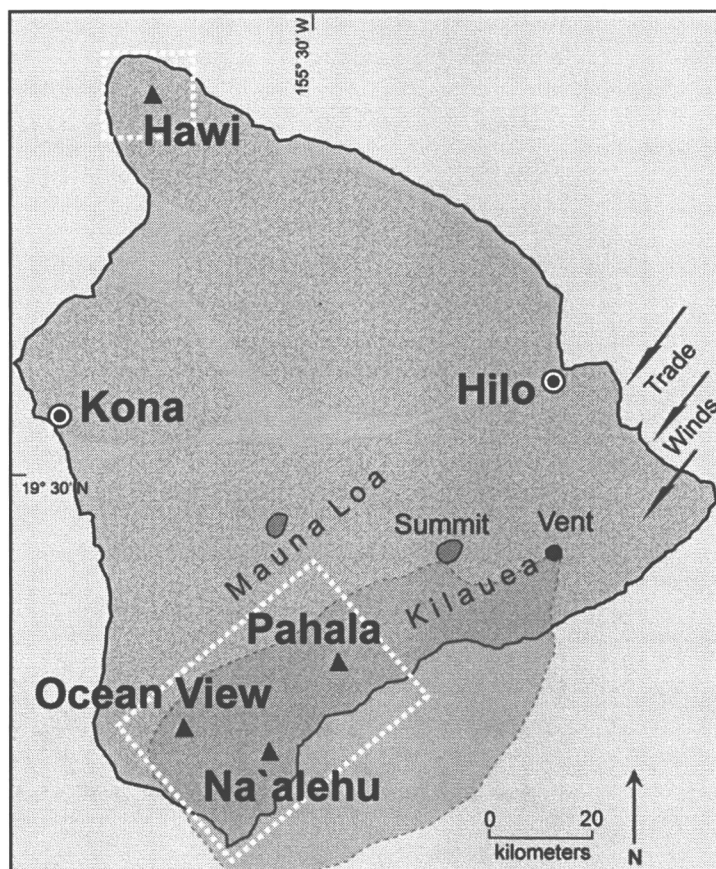


Figure 4.1. Location Map of the Big Island of Hawai'i, U.S.A. The study was conducted in the outlined areas of the exposed cohort of Kau District (towns of Pahala, Na'alehu and Ocean View) and a control cohort area of Hawi. Kilauea Volcano is identified by the summit, vent and plume pattern.

### Environmental exposure

An environmental assessment of  $\text{SO}_2$  and fine sulfate aerosols from Kilauea's volcanic plume indicated high levels of both sulfur species in the Kau District (Figure 4.1), from 37 to 74 km downwind from the eruption (Longo et al., 2005). Ambient  $\text{SO}_2$  concentrations, measured over three-weeks time by diffusion tubes, ranged from 6 to 34 parts per billion volume (ppbv), while  $\text{SO}_2$  penetrated indoors up to 71% of the outdoor concentration (Longo et al., 2005). Comparisons to long-term exposure standards are justified because of the 22-year long eruption and the fact that typical exposure conditions (volcanic emissions and weather) prevailed during sampling.  $\text{SO}_2$  concentrations were above the acute minimal risk level (MRL) of 10 ppbv for an acute

exposure of 14 days (ATSDR, 1998) and above the World Health Organization's (WHO, 1999) guideline of  $50 \mu\text{g}/\text{m}^3$  for an annual exposure. Ambient  $\text{SO}_2$  average concentration in Kau District was 25 times that in control Hawi, and 2.5 to 17 times as great as at other urban locations in the United States during the same time period (Figure 4.2).

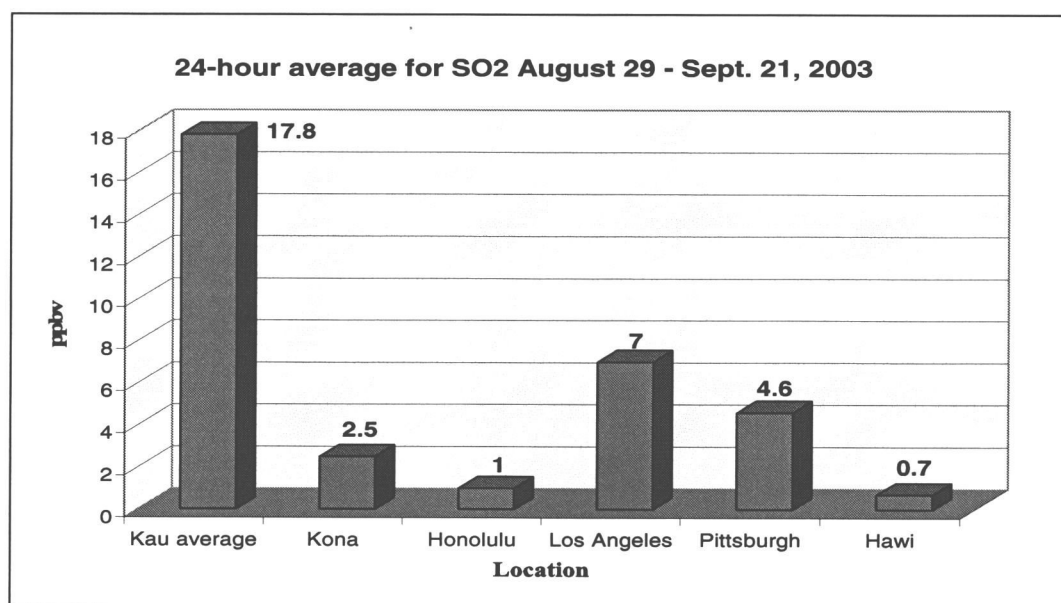


Figure 4.2. Graph of Ambient  $\text{SO}_2$  concentrations measured concurrently in Kau District and other urban areas. Unpublished raw data from: Hawai'i Department of Public Health, Clean Air Branch, 01/22/04, 02/06/04; California Air Resources Board, 10/28/03; Pennsylvania Department of Environmental Protection, 11/05/03.

Moreover, ambient fine particle ( $\leq 0.3 \mu\text{m}$  size) concentrations, measured by grab samples with a cascade impactor, ranged from  $0.61$  to  $11.82 \mu\text{g}/\text{m}^3$  (Longo et al., 2005). These fine particles are likely sulfate aerosols (Chuan, 1995, 1998) with concentrations above the one-year recommended exposure level for sulfuric acid ( $1 \mu\text{g}/\text{m}^3$ ; California OEHHA, 2001) and a proposed sulfate threshold of  $\leq 5 \mu\text{g}/\text{m}^3$  (Thurston et al, 1997).

### Epidemiological findings

Based on the environmental findings, a cohort study of adults ( $N = 335$ ) with a minimum of seven years residency was conducted to assess for chronic health effects from the long-standing eruption. Exposed cohorts of  $\text{SO}_2$  in Na'alehu and fine sulfate aerosol in Ocean View were located in Kau District, and an unexposed control cohort

resided in Hawi on the Big Island (Figure 4.1). Prevalence ratios between control and exposed cohorts revealed substantially increased cough (310%), phlegm (270%), throat irritation (600%), rhinorrhea (740%), sinus congestion (160%), and eye irritation (430%) (Longo et al., 2005). Field-measured blood pressure and respirations were significantly elevated relative to the control. Elderly, non-smoking participants had faster average pulse rates (8 beats per minute) than did their control counterparts. Asthma prevalence in the SO<sub>2</sub> cohort (22%) and sulfate cohort (16%) were not significantly different than control Hawi (16%), but higher than the adult average of 7.9% for the Big Island (Hawaii BRFSS, 2002). These epidemiological findings suggest that long-term residency in degassing volcanic areas may influence adult cardio-pulmonary health.

### Public health

“Vog” is a local term for a visible accumulation of volcanic sulfate pollution. Several studies identified increased hospital visits for respiratory conditions (Bernstein et al. 1984; Mannino et al., 1996; Michaud et al., 2004) and respiratory effects in pediatric asthmatics related to volcanic air pollution (Morrow, 2000). A prospective study into effects on pediatric lung development currently is underway on the Big Island (Sutton et al., 2003).

It is known that asthmatics are sensitive to SO<sub>2</sub> exposure, showing a non-threshold, dose-response relationship of bronchoconstriction or increased pulmonary resistance (WHO, 1999). Other members of the population, however, also are sensitive to SO<sub>2</sub> exposure such as children and adolescents, respiratory and cardiac compromised individuals, and healthy but sensitive to SO<sub>2</sub> individuals (WHO, 1999; Schlesinger, 1999). In addition, sulfate aerosols can negatively affect both sensitive and general populations by inducing respiratory tract irritation that can alter lung defenses, such as airway reactivity, alveolar particle clearance, and mucociliary transport (Holgate et al., 1999; Koenig, 2000).

Public health interventions have been employed. The Hawaiian Volcano Observatory (HVO) of the United States Geological Survey has instituted public health warnings and education for visitors to Hawai'i Volcanoes National Park (USGS, 2001). For Kona residents (Figure 4.1), a Vog level index is used to determine poor air quality

by the Hawai'i Department of Public Health. The American Lung Association of Hawai'i (2002) has provided a list of interventions for Vog-affected residents. Nevertheless, the scarcity of research and lack of environmental and health assessment on downwind populations provided the rationale for this investigation.

## **Methods**

### **Theoretical framework**

The ecocentric perspective was used as a theoretical framework to investigate the human-environmental interaction and human health responses in populations living near a degassing, active volcano. Traditional nursing and medicine use an egocentric perspective that is grounded at the personal level, while public health uses an expanded homocentric perspective grounded at the social-population level (Kleffel, 1996). In contrast, the ecocentric perspective is grounded in the large scale, human-environmental interaction, assuming that all matter is connected – inanimate with animate, alive, whole, and interacting (Kleffel, 1996). In addition, researchers used a nursing framework, that of being concerned with human experiences and partnering with individuals, communities and populations to address the environment and the prevention of disease (ANA, 2003).

Many variables in health assessment cannot or should not be quantified because attempts to separate the unified system of a human being can result in what appears to be a comparable measure of health but lacks true validity. Therefore, this study incorporated a qualitative aspect, which complements the quantitative environmental and epidemiological data, which when used collectively helps too more fully understand the human health condition.

### **The question**

What are the human responses and lived experiences within adults to the actual or potential health problems of residing in a community near an erupting, degassing volcano? Two methods were used to capture descriptions: open-ended questions and unstructured interviews. The epidemiology questionnaire for exposed participants (randomly sampled) also included two open-ended questions: 1) How has Kilauea's eruption affected your life? 2) Do you think or feel that the eruption has affected your



health? These questions were asked by the nurse researcher or read and answered by the participant. The nurse researcher explored or restated the responses to attempt to ensure clarification of meaning from the participant. A signed consent was obtained from all participants (Appendix II). In addition, convenience-selected informants shared descriptions of their lived experience with the eruption during unstructured interviews. Some interviews with informants were audio-recorded with a signed consent (Appendix II).

### **The sample: Participants and informants**

The study consisted of 225 randomly sampled adult residents of the exposed cohort in Kau District, aged 20 years or older. The SO<sub>2</sub> cohort participants were from the Na'alehu area, and the sulfate cohort participants were from the Ocean View area. Also included were descriptions of acute reactions when visiting Vog-affected areas from 10 randomly sampled control participants.

Data was collected in the natural setting, which was the home of participants or in the outdoors with informants such as ranchers and farmers. The nurse researcher lived in the study area, allowing immersion into the local milieu. Contact was made with the hospital, physicians, seniors and rural health association. Study findings were reviewed regularly with a local physician, a member of the research team. These fieldwork techniques supported the internal validity of the study. Random sampling supported the reliability of the study.

### **Analysis of qualitative data**

Descriptions from participants who perceived that their health was affected by the eruption (n = 80) were reviewed for themes and coded for similarities and differences. SO<sub>2</sub> and sulfate cohort descriptions were compared. In addition, these qualitative data were compared to the epidemiological and environmental findings in order to assess the presence of an illness syndrome. The health interventions used by residents affected by the eruption were also cataloged.

## Findings

### Sulfur dioxide descriptions

In the SO<sub>2</sub>-exposed cohort area of Na`alehu, 30% of the participants perceived that the eruption had affected their health. Of those affected, 80% were residents in the area for 20 years or longer. A retired sugar cane worker declared, *"There used to be cane burning here, but the volcano is much worse!"* There were no differences in gender or the type of home (50 year old, plantation-style verses modern), relative to those not affected. Those over 60 years in age made up 44% of the affected group. Considering ethnicity, 43% of Native Hawaiians, 30% of Japanese, 26% of Filipinos and only 14% of Caucasians, felt affected. Of those affected, 74% were born in the Hawaiian Islands. Lastly, 38% of the former smokers, 25% of non-smokers, and 24% of current smokers felt their health had been affected by the eruption.

Sinus congestion was the most frequently described health effect related to the eruption. A lifetime resident that never smoked admitted, *"It makes me feel constantly congested."* Breathing difficulty, eye irritation and sneezing also were frequently described. A long-time elderly resident who smokes stated, *"The Vog surely has affected my respiratory health. I have become immune to it on a daily basis, but on bad days I have breathing problems and watery eyes that burn."* Another elderly long-time resident described, *"I have allergies and sneeze 2-3 times in the morning and evening. I was never like this before the eruption."*

Asthmatics reported an increase in symptoms of wheezing, shortness of breath (SOB), and limited activities of daily living. They shared a common theme of continual battle with their asthma management. Some residents even attributed the eruption to onset of their asthma. A long-time resident with adult-onset asthma explained, *"The plantation was active when I moved here and I had allergies. But when the volcano came, I developed asthma. I definitely believe it has affected my health."* Furthermore, a middle-aged asthmatic asserted, *"The eruption makes my asthma worse, it has affected my life – I can't exercise, walk or move."*

Some participants felt that the eruption had caused an increase in illness. A resident for nearly 60 years claimed, *"I think it has affected the community. It is easy to*

*catch a cold, or have repeated colds and lung illness."* Another middle-aged, lifetime resident was uncertain, *"Maybe, at the start of the eruption there was a lot of sickness. Kids here have asthma and bronchitis. I really don't know."*

### Sulfate descriptions

In the sulfate-exposed area of Ocean View, 40% of the cohort participants perceived that the eruption had affected their health. Ocean View is a relatively new subdivision on the Big Island, located between 2,000-4,500 feet in altitude, with few long-time residents. Of those affected, 70% had lived <20 years in the area. Of participants with  $\geq 20$  year residency, however, half felt affected by the eruption. There were no relative differences in gender or ethnicity between those who felt affected and those who did not. Middle-aged residents (40 to 59 years) described being most affected. Different than SO<sub>2</sub> participants, relatively more sulfate cohort participants with a smoking history felt affected by the eruption. Half of the current smokers and 45% of former smokers reported being affected compared to only 25% of never smokers.

In Ocean View, sinus congestion, eye irritation and breathing difficulties were the primary described health effects from the eruption. A resident of over ten years from the mainland asserted, *"Of course my health is affected! My sinuses are worse everyday, sometimes choking."* An elderly resident in good-health noticed, *"When you sneeze it is about 15 times in a row when the Vog is around, and we don't see that on other days."* Another ten year resident who was a former smoker held that since coming, *"I have sneezes, a tickling throat that I never had before. My nose is free-flowing."* Moreover, a middle-aged resident that never smoked and had lived in Ocean View before the eruption began compares, *"It affects my breathing and my eyes. I know because I was here before the eruption- we get the Vog."*

Descriptions of increased coughing and phlegm or sputum also were common. A high-altitude, smoking, long-time resident complained, *"I cough more with the Vog and the phlegm is worse up here. I'm definitely affected."* Fatigue and headache also were described more often than from the SO<sub>2</sub> affected participants. A resident of only seven years to the area described, *"When it is thick enough to see it, it gives me a headache and makes me feel very sleepy."*

Asthmatics described being affected in the area. A long-time (> 20 years) asthmatic that smokes believed, *"The Vog affects my asthma, I wheeze and loose stamina, and have nausea and a headache."* Not all asthmatics viewed the eruption as affecting their health. A ten-year resident from the mainland pointed out, *"my health is not affected compared to Los Angeles!"* Nevertheless, asthmatics shared the nuisance of the eruption on controlling their asthma, sometimes described helplessness, and even blamed the Vog for adult-onset asthma. A former smoking resident of over ten years compared, *"Before I moved to Hawai'i I didn't have asthma. I'm worse in Kona than here. I have coughing, phlegm and burning eyes."* A report from a resident described a location-related pattern to the symptoms:

*"I worked two weeks on and two weeks off, leaving the islands. When I was gone all the symptoms would disappear, within a week to 10 days. I'd feel great! No eye irritation or nasal problems. I think it would take about a week for the symptoms to reappear when I'd return home."*

### Descriptions of Vog-reactions from control participants

Many residents from the control Hawi area visit Kona or work at resorts along the Kona coast. Thus, their descriptions of acute responses to Vog supplement descriptions from the chronically exposed participants. Eye irritation that burns, waters or itches was the most common response. Second were acute and delayed reactions (up to a two-day lag) of sinus irritation and congestion. Current smokers experienced throat irritation, headache and fatigue whereas non-smokers did not.

### Interventions used by local residents

To reduce health effects attributed to the eruption, some participants stay indoors or leave the area on "bad days." There were no reports of specifically using an air conditioner, air filter system, or masks, to minimize exposure to the volcanic air pollution. A young, lifetime resident suggested, *"I think that the people of Kau and the Big Island have grown to adapt to our native land, whether or not the volcanic activity affects us – we find ways to deal with it."*

Affected participants sought out traditional medical interventions and homeopathic or naturopathic treatments. Traditional treatments consisted of prescribed and self-prescribed over-the-counter medicines focusing on symptom relief. For sinus

symptoms, prescribed nasal steroids were commonly used and reported to be quite effective. Other medications used were systemic anti-histamines, decongestants, and asthma inhalers. Self-prescribed herbal remedies, described as very effective, were nasal washes and eye drops. Homeopathically prescribed treatments were viewed as effective. Some residents felt that Vog depleted magnesium and potassium, and when replaced, was reported effective at reducing fatigue. Outdoor workers reported drinking more water to counteract effects of the Vog.

## Discussion

Most participants did not perceive their health was affected by the volcanic eruption. In fact, some Kau residents did not believe they were exposed to any volcanic air pollution. Regardless, a common theme emerged that the volcanic air pollution or Vog is an unwanted “thing” that affects the community when “it” comes. Kilauea itself was not viewed negatively; instead, the volcano was respected. Informants from the Pahala area of Kau district called the pollution “*the sulfur*”, whereas informants in Ocean View and Na`alehu called it “*the Vog*.” Moreover, there was a common belief that the Kona area was more affected or worse than was the Kau District. The Kona coast visibly accumulates Vog. Kau residents reported that when visiting Kona they have occasionally experienced health effects. Environmental data (Longo et al. 2005) showed the variability of sulfur species and concentrations within Kau District; that is, not all areas downwind of Kilauea experience the same pollution.

In Na`alehu, where SO<sub>2</sub> averaged 25 ppbv in September 2003, visible sulfate pollution (Vog) is not present on most days. When the usual trade winds slow or become variable, Vog forms from the oxidation of SO<sub>2</sub>. Therefore, many residents considered exposure to volcanic air pollution to be an occasional occurrence based on visible observation.

SO<sub>2</sub> is a colorless gas with an odor threshold at a concentration of  $\geq 380$  ppbv (Godish, 1997). Participants reported that they have smelled and even tasted sulfur on occasion in Na`alehu and Ocean View, likely indicating high concentrations of SO<sub>2</sub> or sulfate accumulating in the area. Even in Ocean View, where visible sulfate pollution or

Vog is present on most days, some informants did not think they were being exposed. As one participant described *"I noticed when the eruption stopped for 3-4 months how clear the sky looked and it made you see-realize how hazy it is a lot of the time."*

Most important for health promotion is that there is a daily temporal pattern to volcanic air pollution in the Kau District. Data indicated that Pahala was subject to an afternoon peak in  $\leq 0.3 \mu\text{m}$  particles; the diurnal pattern of volcanic fine aerosol followed the trade-wind pattern for the windward slope of the Big Island (Longo et al., 2005). Ocean View had high concentrations of fine aerosols during dry and wet conditions, mornings and afternoons, as well as during trade and variable wind periods (Longo et al., 2005). Informants in Ocean View confirmed a field-observed pattern of Vog appearing by mid-morning and disappearing at nighttime. Informants throughout higher-altitude areas of Kau confirmed that afternoon upslope winds bring the volcanic plume (Vog) landward. Due to method and instrument limitations, an  $\text{SO}_2$  temporal pattern could not be confirmed (Longo et al., 2005).

### VOLCANIC SULFUR ILLNESS SYNDROME IN ADULTS


<b>Primary:</b> Eye irritation Cough Rhinorrhea Phlegm in smokers Wheeze in asthmatics Dyspnea in respiratory patients	<b><math>\text{SO}_2</math> specific:</b> Dry cough hay fever <b>Sulfate specific:</b> Sinus congestion bronchitis
<b>Secondary:</b> Headaches Sore/dry throat Colds with chest involvement Elevated blood pressure Increased pulse rate in elderly Fatigue	

Figure 4.3. Chart of volcanic sulfur illness syndrome characterizing signs and symptoms.

#### Characterization of a sulfur illness syndrome

Table 4.1 compares epidemiological prevalence findings to qualitative descriptions provided by participants who perceived that their health was affected by the

eruption. Noteworthy are the contrasting prevalences of most respiratory symptoms between those who perceived being affected and those who did not. While these findings do not prove that the eruption caused specific symptoms or illnesses, strong differences in prevalence between the exposed cohorts and control cohort suggest an association with volcanogenic air pollution for symptoms such as rhinorrhea, sore/dry throat, eye irritation, cough, dry cough, phlegm, and elevated blood pressure (Table 4.1). A characterization of a volcanic sulfur illness syndrome is presented in Figure 4.3 based on qualitative and epidemiological findings in the presence of environmental data. Primary, secondary, and species-specific characteristics of the syndrome are offered.

### Health promotion recommendations

There are opportunities in health promotion for the residents living downwind of the volcano. Undoubtedly, more health-related investigations are needed in all communities along the volcanic plume path. Recommendations for Kau District are offered that focus on further air quality assessment for environmental and health effects, and promoting health transformation in the community.

Air monitors for SO<sub>2</sub> in the Na'alehu area and fine particles in Ocean View should be placed to assess concentrations and trends in air quality that are available to the public in real-time. The Hawaiian Volcano Observatory could communicate changes in Kilauea's activity and emissions through multi-disciplinary relationships with Kau institutions such as schools, hospital, clinics, senior centers and the Rural Health Association. For example, fountaining activity has been found to release higher concentrations of SO<sub>2</sub> (Greenland et al., 1985) potentially affecting the health status of sensitive members in the community. Detailed work on indoor penetration of sulfur species and air filtration (i.e., air conditioning, swamp coolers) is needed to determine structures and materials that can best reduce exposure to residents.

Community screening could assess pulmonary and cardiac health, thereby instituting therapeutic interventions as needed. The estimated burden of cough in Ocean View, which is attributed to the eruption, ranges from 373 – 782 individuals (Longo et al., 2005). The Kau community is a rural population, however, that has high unemployment (22.6% in Na'alehu) and an associated lack of health insurance (23% in

Ocean View) (Longo et al., 2005). Therefore, state or federal assistance with health care is needed.

Health promotion activities through the Department of Public Health and the Rural Health Association could include education on air pollution, community smoking cessation programs, cardiac risk factor reduction, and exercise régimes that reduce exposure. Unfortunately, 27% of adult participants in Ocean View smoke; the combined exposure of air pollution and smoking is associated with increased symptoms (Longo et al., 2005) and higher risk of death from cardiac disease (Pope et al., 2004). Therefore, reduction in smoking and secondary smoke exposure, especially with pregnant women, should be a priority. Cardiac health through modifiable risk reduction (weight, diet and eating habits, exercise, and control of blood pressure) should be promoted. In Na`alehu and Ocean View, 46% of participants had un-medicated high blood pressure  $\geq 140/90$ , and 24% were without a prior diagnosis of hypertension (Longo et al., 2005). In addition, 52% of the adult population was overweight or obese (Longo et al., 2005). Exercise programs, under physician care, should be promoted. Morning is likely the best time for outdoor activity to minimize exposure, along with nasal breathing over mouth breathing to improve filtration of air. During typical Pacific trade wind conditions, the volcanic plume moves landward by 11 am. During periods of variable winds or low trades, exercise should be postponed.

Health care clinicians should provide therapeutic régimes for adult clients living in volcanic downwind areas, especially those with known respiratory and cardiac disease. Following a detailed assessment for syndrome symptoms, further assessment may include pulmonary function testing, smoking cessation protocols, blood pressure monitoring, and thorough assessment of self-medicating practices for symptom relief. Important is addressing any potential fluid overload by clients, especially elderly and cardiac clients, who drink water to counteract effects. Clients who are unstable in their management of asthma, cardiac or respiratory disease, may benefit from relocation.



## **Conclusions**

The residents of Kau District, Hawai'i, have lived with Kilauea's eruption for over 20 years, yet less than half of them perceive that their health has been affected. Despite the statistics, to those affected by volcanic air pollution or "Vog" the human health response is real, bothersome, and sometimes debilitating. Descriptions of physical symptoms from adult residents reveal a sulfur illness syndrome that is experienced by both asthmatics and the general population. Current and former smokers report being most affected by volcanogenic air pollution. Research in the world's volcanic areas should explore human health experiences qualitatively, as well as quantitatively. Researchers should partner with local health clinicians, communities and populations to address the environment, health and the prevention of disease.

## **Acknowledgements**

The spirit of Aloha emanated to the researchers from the residents of Kau District and Hawi. The authors wish to thank the following individuals who provided assistance with this study: Dr. Peter Baxter, MD of the International Volcanic Health Hazard Network, Dr. Raymond Chuan, Prof. Chunhuei Chi, Dr. Dennis Elwell, Prof. Anita Grunder, Fr. Manuel Hewe, Mrs. Barbara Marks, Prof. Cathy Neumann, Dr. Ben Ono, MD, Dr. Jackie Paulson, RN, and Prof. J.K. Yun. We thank also the following agencies for providing air quality data: The Clean Air Branch of Hawai'i Department of Public Health, California Air Resources Board, and the Pennsylvania Department of Environmental Protection.

Table 4.1. Comparison of Qualitative Descriptions (D) and Prevalence (P) of Affected vs. Non-Affected Participants

SYMPTOMS AND DISEASES	SO <sub>2</sub> Cohort			Sulfate Cohort			Control
	D	Affected P %	Non-A P %	D	Affected P %	Non-A P %	P %
Cough on most days*	+	55.2	28.4	+	52.5	44.8	12.7
Dry cough at night*		60.7	14.9		20.0	12.1	6.4
Sinus/nasal congestion often*	+	43.3	17.1	+	73.7	29.1	16.4
Wheeze in last 12 months	+	35.3	14.8	+	56.8	16.7	15.5
▪ most days/nights		29.4	8.6		31.8	4.5	7.3
▪ with a cold		47.1	30.9		38.6	30.3	20.9
▪ exercise induced		29.4	7.4		20.5	6.1	8.2
Phlegm on most days		38.2	23.5	+	38.6	22.7	10.9
Chest colds		58.8	34.6		68.2	36.1	53.8
SOB without exertion	+	33.3	12.3	+	32.6	6.1	10.0
Chest pains (ever have)		44.1	19.8	+	47.7	10.6	20.0
Sore/dry throat (frequent)	+	35.3	21.0	+	31.8	10.6	2.7
Runny nose (frequent)	+	23.5	13.6	+	43.2	27.3	5.5
Head aches (often)	+	52.9	30.9	+	52.3	15.4	22.7
Outdoor cough		30.3	7.4	+	47.7	7.6	4.6
Outdoor eye irritation	+	66.7	18.5	+	72.7	24.2	8.2
Outdoor rhinitis, congestion	+	39.4	4.9	+	61.4	18.2	3.6
Outdoor SOB		27.3	9.9		25.0	4.5	3.6
Outdoor wheezing		29.4	3.7		25.0	3.0	2.7
Cluster of nose, sinus & eyes		8.8	7.4	+	29.5	10.6	2.7
Asthma	+	41.2	13.6	+	27.3	9.1	16.4
bronchitis		44.1	17.3		68.2	29.2	24.5
chronic bronchitis		20.6	4.9		22.7	4.5	4.5
COPD		8.8	1.2		6.8	4.5	2.7
Hayfever/allergies		55.9	16.1		40.9	18.2	23.6
chronic hayfever		44.1	8.6		31.8	7.6	9.0
chronic sinusitis	+	27.3	6.2	+	38.6	6.1	12.7
pneumonia (within 21 yrs)		29.4	3.7		18.2	19.7	19.1
coronary artery disease		11.8	4.9		6.8	10.6	7.3
myocardial infarction		5.9	5.0		6.8	6.1	2.7
Angina		14.7	4.9		11.4	6.1	3.6
Dx hypertension		52.9	43.2		27.3	43.9	32.7
Field: BP ≥ 140 or ≥ 90		60.0	51.3		50.0	53.0	39.0

"Affected participants" are those who perceived that the volcanic eruption had affected their health: SO<sub>2</sub> cohort (n = 34, or 30%), Sulfate cohort (n = 44, or 40%).

Non-A = Non-affected participants are those who did not perceive their health was affected by the eruption.

+ = Qualitatively described symptoms associated with volcanic air pollution.

\* Prevalence restricted to participants not taking medications with known side effects.

## CONCLUSIONS

Environmental data indicate that Kau District, Hawai'i, is being exposed to concentrations of volcanic SO<sub>2</sub> and fine particles that warrant public health concern. Concentrations of sulfurous air pollution are strongly correlated with altitude, displaying a pattern of increasing aerosol and decreasing SO<sub>2</sub> with rise in elevation.

Long-term residency in active and degassing volcanic areas may influence cardio-pulmonary health in adults. Relative to an unexposed control cohort, exposed cohorts of SO<sub>2</sub> and sulfates experienced substantially increased prevalence of cough, phlegm, throat irritation, rhinorrhea, sinus congestion, and eye irritation. Blood pressure, pulse rate and respirations also were significantly elevated in certain sub-groups of participants.

Less than half of the exposed participants reported that the volcanic eruption had affected their health. Epidemiological findings and qualitative descriptions from participants suggest that chronic exposure to volcanogenic air pollution may result in a sulfur illness syndrome with primary and secondary characteristics.

### Contributions of the Study

A new model was used for conducting research on adverse health effects from geological events. The contemporary fields of medical geology and environmental epidemiology consider the interactions of the environment on human disease. This model, however, considers environmental, epidemiological and qualitative data to more fully analyze the human-environmental interaction between populations and volcanoes.

This study employed field research techniques unlike those used in other volcanic health studies, including studies in Hawai'i. First, the student researcher contacted local health care clinicians and agencies (hospital, clinics and rural health organizations) within the study area. It is ethically imperative to communicate with local health care clinicians about research because they are the professionals who care for the population's health. In addition, local law enforcement and special groups, such as the senior citizens, were informed of the study. These actions provided collaboration between the researcher and the populations under investigation. Partnering with the community with research efforts

and establishing communication pathways ultimately can disseminate the research findings and facilitate public health interventions. Moreover, knowledge about the cultural aspects of health in the community can be shared with the researcher.

Second, health data were collected by a qualified health care clinician and not by secondary-trained data collectors or researchers from non-health disciplines. Experienced health care clinicians employ refined tools of health assessment, pattern recognition, diagnosis, intervention, and ongoing evaluation that come from professional practice.

Third, the use of a random sampling technique, as opposed to convenience sampling, is the most effective tool to decrease systematic error (sampling bias) and supports the internal validity of the study. In addition, data were collected in-person as opposed to a mailed or telephone survey. Being in the field allows meticulous evaluation of both the environment and participant based on observational assessment. In qualitative and some types of quantitative research, the interaction between the clinical researcher, who is the tool, and the participant, who provides data, can yield fruitful results.

Fourth, these environmental data were collected during typical exposure conditions in meteorology and volcanic emissions. A goal in environmental sampling is to attempt to measure a typical exposure on a population; however, health scientists should consider also the range of peaks and troughs in concentration measurements. These preliminary results, a first-time look, surveyed for elevated concentrations of air pollutants that may warrant concern for health and the need of further investigation.

Fifth, these environmental data were the first collected on a regional scale in Hawai'i. This technique discovered a new plume pattern in the downwind area, an altitude effect on the concentration of SO<sub>2</sub> and fine particles. Data collected by diffusion tubes in this study were adjusted for altitude effects on the mass and volume-mixing concentrations. These correction adjustments had not been made on data from the same type of tubes in other volcanic settings such as Masaya, Poas, or Furnas volcanoes.

Sixth, the environmental sampling prior to the epidemiological data collection allowed the identification of sub-cohorts of volcanogenic SO<sub>2</sub> and sulfates. The residency requirement of seven years or longer was used to capture participants with chronic exposure to each sulfur species in the identified geographic areas. Lastly, the research

findings from this study provided the first identification of an illness syndrome associated with exposure to volcanogenic air pollution, areas of risk, and possible public health interventions.

### **Limitations of the Study**

This dissertation was limited by several factors. The student researcher collected, analyzed (exception was lab work for SO<sub>2</sub> by Harwell Scientifics), and interpreted these data. A single data collector, however, is a risk to reliability because of a potential bias and loss of objectivity throughout the research process. The following are other noted limitations of the study:

- 1) The length of data collection was short-term, especially for environmental data.
- 2) No meteorological data were directly collected at each sample site (temperature and pressure) during sampling.
- 3) Fine particle data were limited to <1.6  $\mu\text{m}$ , not PM<sub>2.5</sub> or PM<sub>10</sub>.
- 4) The sample size for the epidemiological analysis was limited in power to discover a minimal doubling of effect. Investigations involving cardio-pulmonary diseases often have risk ratios less than 2.0 because of the commonality of these pathologies in the general population. These relatively small increases in risk, however, are very important from a public health viewpoint because they may involve large numbers of affected individuals.
- 5) The quantity of spirometry data was insufficient for analysis ( $n = 7$ ). Using spirometry in field research is difficult. Screening of participants for smoking status and cardio-pulmonary health lowered the potential sample size for spirometric assessment. Other impediments that occurred in the field included the requirement of more time from the participant, the need to enter the home to conduct spirometry, lack of portable battery-powered equipment, and the time-effort for calibration at each sample site (altitude effects: temperature, pressure, humidity).
- 6) The health data cannot be generalized to the entire population of Kau District because of the exclusion of residents from Pahala.

## Recommendations

In volcanic settings worldwide, a multidisciplinary research team should utilize the following research method:

- 1) Collaborate with the local population in research efforts, especially health care clinicians and agencies. Respect the local cultural norms.
- 2) Conduct environmental sampling on a regional scale to identify areas of risk.
- 3) Compare pollutant concentrations to minimal risk levels (MRL) that are designed to protect sensitive members of the population, and to WHO long-term air recommendations if degassing activity exceeds one year.
- 4) Determine the prevalence of cardio-pulmonary symptoms and illness in the affected population and a control using a standardized protocol (the study questionnaire and vital sign measurements). Using a standardized protocol would allow future comparisons could be made of the health impacts from different periods of eruption on the population.
- 5) Initiate a prospective cohort study and general health monitoring of the affected population and control at the start of an eruption.
- 6) Determine the additional burden of morbidity and mortality if any on the population.
- 7) Identify the human-health response using ethnographic or other qualitative methods.
- 8) Promote community participation in health by collaborative interventions that are preventative, therapeutic and sustainable.
- 9) Use fixed continual air monitors (location determined by environmental survey) for long-term evaluation of air quality on the affected population.

The following are recommendations for the Big Island of Hawai'i:

- 1) Continue investigation, using the same method as this study, in the communities downwind of Kilauea Volcano, from Ocean View to Captain Cook and the greater Kona area.
- 2) Repeat the environmental survey for SO<sub>2</sub> and fine particles in Kau District during dry trade wind conditions (early summer) and during variable wind periods (winter). Use a full-range QCM particle analyzer that can measure from 0.1 µm up to 2.5 µm.

- 3) Produce a Volcanic Air Pollution Hazard Map of the Big Island for the public. The USGS could produce this map because they have created other hazard maps (lava flow and earthquake) for Hawai'i. USGS also could evaluate the need for an environmental survey of mercury or other volcanogenic pollutants.
- 4) Institute long-term assessment of air quality by placing continual monitors for  $\text{SO}_2$  in Na'alehu (grade school) and  $\text{PM}_{2.5}$  in Ocean View (community center, ~ 2,500 ft. a.s.l.). Use the Vog Index and educate the Kau residents about the meaning of the levels.
- 5) Due to the indoor  $\text{SO}_2$  penetration levels found in this study, indoor air quality testing should be further conducted in the community hospital, schools, and homes of selected residents. Air purification methods (i.e., air conditioning, swamp cooler) and equipment should be tested.
- 6) Cardio-pulmonary health screening (including spirometry) and education should be conducted for the Kau population. The health screenings could be hosted at the community centers located in Pahala, Na'alehu and Ocean View. Involve the local health care clinicians in planning the screening.
- 7) Promote cardiac health through modifiable risk reduction (weight, diet and eating habits, exercise, and control of blood pressure). Promote respiratory health through smoking cessation programs, community smoking deterrents, and asthma awareness education.
- 8) Continue to develop public health recommendations and interventions.
- 9) Create a Volcanic Health Task Force in downwind districts of the Big Island. The county of Hawai'i (Big Island) has an existing Vog Task Force under the mayor, and Kau District has an existing community board of residents and a state representative; therefore interested individuals and groups are in place.
- 10) Involve the International Volcanic Health Hazard Network in the research efforts, thereby encouraging multidisciplinary and international collaboration for the promotion of human health.
- 11) Prepare for future long-lasting eruptions with degassing activity from any of the three active volcanoes on the island.

## Summary

This study contributed to the scientific knowledge of human health effects from chronic exposure to  $\text{SO}_2$  and sulfate aerosols. Millions of individuals are living in areas near degassing volcanoes, and in urban areas polluted with  $\text{SO}_2$  and sulfates from

anthropogenic sources. With Earth's population increasing, more and more people are living in active geological areas, some by choice, and some because of limited socioeconomic resources. Regardless, these findings indicate that disparity in health likely exists in communities with volcanogenic air pollution. In 2001, WHO called for action to conduct quantitative, evidence-based studies to assess regional and local air pollution, and the associated burden of disease. While vast amounts of research have been conducted on urban air pollution consisting of mixtures of air pollutants, the natural laboratories of Earth's volcanoes that produce sulfurous pollution has seen very limited health research. This study begins to answer the basic questions of which geographic areas and populations are at risk, and whether there is an elevated morbidity of cardio-pulmonary disease associated with air pollution from the Kilauea Volcano.



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## **APPENDICES**

## APPENDIX I



OREGON STATE UNIVERSITY  
**INSTITUTIONAL REVIEW BOARD**  
 312 Kerr Administration Building - Corvallis, Oregon - 97331-3140  
 E-MAIL: [IRB@oregonstate.edu](mailto:IRB@oregonstate.edu) - PHONE: (541) 737-3437 - FAX: (541) 737-3093

## REPORT OF REVIEW

TO: Anne Rossignol,  
 Public Health

RE: The Kilnsea Volcano Adult Health Survey (Student Researcher: Bernadette Mae Longo)

Protocol No. 2268

The referenced project was reviewed under the guidelines of Oregon State University's Institutional Review Board (IRB). The IRB has approved the application. This approval will expire on 7/29/2004. This new request was reviewed at the Expedited level. A copy of this information will be provided to the full IRB committee.

Enclosed with this letter please find the original informed consent documents for this project, which have received the IRB stamp. This information has been stamped to ensure that only current, approved informed consent forms are used to enroll participants in this study. All participants must receive the appropriate IRB-stamped informed consent documents. Please make copies of these originals as needed.

- Any proposed change to the approved protocol, informed consent form(s), or testing instrument(s) must be submitted using the MODIFICATION REQUEST FORM. Allow sufficient time for review and approval by the committee before any changes are implemented. Immediate action may be taken where necessary to eliminate apparent hazards to subjects, but this modification to the approved project must be reported immediately to the IRB.
- In the event that a human participant in this study experiences an outcome that is not expected and routine and that results in bodily injury and/or psychological, emotional, or physical harm or stress, it must be reported to the IRB Human Protections Administrator within three days of the occurrence using the ADVERSE EVENT FORM.
- If a complaint from a participant is received, you will be contacted for further information.
- Please go to the IRB web site at: <http://osunet.edu/research/RegulatoryCompliance/HumanSubjects.html> to access the MODIFICATION REQUEST FORM and the ADVERSE EVENT FORM as needed.

Before the expiration date noted above, a Status Report will be sent to either close or renew this project. It is imperative that the Status Report is completed and submitted by the due date indicated or the project must be suspended to be compliant with federal policies.

If you have any questions, please contact the IRB Human Protections Administrator at [IRB@oregonstate.edu](mailto:IRB@oregonstate.edu) or by phone at (541) 737-3437.

Redacted for privacy

*for* Dr. Anthony Wilcox  
 Institutional Review Board Chair

Date: 7/30/03

pc: 2268 file



## APPENDIX II

### INFORMED CONSENT DOCUMENT

#### Project Title: The Kilauea Volcano Adult Health Study

**Principal Investigator:** Anne Rossignol Ph.D., Professor, Public Health

**Student Researcher:** Bernadette Longo, RN, Doctoral Candidate, Public Health

#### PURPOSE

This is a research study. I am Bernadette Longo, the student researcher for this study. I am a Hawaii registered nurse with over 20 years experience, and a student pursuing the doctoral degree in Public Health at Oregon State University. The research team and I want to invite you to participate in this important study.

The purpose of this research study is to gain an understanding of any effects of air pollution from Kilauea volcano on the health and well being of residents in the Kau District. This study will look at how many adults currently have breathing and heart-related illnesses. This is called prevalence of a disease. We will compare this amount of illness to the prevalence in another community on the Big Island (Hawi) that is away from the volcano. A lot can be learned!

First, adults and the elderly have not been studied in Kau. Second, the researcher is also mapping where the air pollution occurs in the area and the amount of it. Maybe there is a problem with air pollution and health, and maybe there isn't. Scientific research may help answer the question. If you participate, you would be part of helping find some answers. Do you think this is important to know? The results of this study are for you and the public, to be shared in your community, and for all scientists and health care workers (doctors, nurses and public health).

The purpose of this consent form is to give you the information you will need to help you decide whether to be in the study or not. Please read the form carefully. You may ask any questions about the research, what you will be asked to do, the possible risks and benefits, your rights as a volunteer, and anything else about the research or this form that is not clear. When all of your questions have been answered, you can decide if you want to be in this study or not. This process is called "informed consent". You will be given a copy of this form for your records.

We are **inviting you** to participate in this research study because you are an adult (20 years or older) who lives in the Big Island communities of Kau, or Hawi. You were selected by a scientific sampling procedure, and your participation is important to the success of this study. There will be about 300 people participating in this study.

**PROCEDURES:** If you agree to participate, your involvement will last for **15-20 minutes**. The following procedures are involved in this study:

1. Please read and complete the questionnaire to the best of your ability.
2. The researcher will re-visit your home in a few days to answer any questions you may have, help you complete the questionnaire if you wish, and review the answers with you.
3. The researcher will measure your **pulse** (beats of your heart) at your wrist, your **blood pressure** on your arm, and count your breaths (respirations).
4. Based on the results of the questionnaire, the researcher may ask you if you would also like to participate in spirometry (a lung breathing test) or be interviewed.

**RISKS:** There are **minimal foreseeable risks** to participating. There may be some discomfort in answering health questions or having your blood pressure taken.

**BENEFITS:** The potential personal benefits that may occur as a result of your participation in this study are having your pulse and blood pressure taken by a registered nurse, and recorded on a sheet that you may take to your doctor if you wish. The researchers anticipate that society may benefit from this study by learning about any health effects in adults that may be associated with the air pollution from the volcano.

Very little research has been conducted on the lives and the health of the **500 million people** who live near active volcanoes around the world. Your participation can help your community, and other communities on the Big Island and around the world.

**COSTS AND COMPENSATION:** You will not have any costs for participating. You will not be compensated with money for participating in this research project.

**CONFIDENTIALITY:** Records of participation in this research project will be kept confidential to the extent permitted by law. However, federal government regulatory agencies and the Oregon State University Institutional Review Board (a committee that reviews and approves research studies involving human subjects) may inspect and copy records pertaining to this research. It is possible that these records could contain information that personally identifies you.

To **protect your confidentiality**, the researcher will not share with anyone that you have participated, or what information about you was obtained. This includes not sharing with your doctor or family, unless you make a request. After signing this consent, it will be removed from the questionnaire and a number assigned that only the researcher will know. These questionnaires and consent forms will be kept in a secure location, only accessible by the research team. After analysis of these questionnaires occurs, they will be destroyed. In the event of any report or publication from this study, your identity will not be disclosed. Results will be reported in a summarized manner in such a way that **you cannot be identified**.

**VOLUNTARY PARTICIPATION:** Taking part in this research study is **voluntary**. You may choose not to take part at all. If you agree to participate in this study, you may stop participating **at any time**. You may skip any question on the questionnaire, or decline having your pulse and blood pressure taken. If you decide not to take part, or if you stop participating at any time, your decision will not result in any penalty or loss of benefits to which you may otherwise be entitled. Your questionnaire would not be used in the study, but it would be kept confidential to the extent permitted by law.

**QUESTIONS:** Questions are encouraged.

If you have any questions about this research project, please contact:

**Bernadette Longo, RN**, local phone # 808-929-7311 email: [blongo1959@aol.com](mailto:blongo1959@aol.com) and/or **Dr. Anne Rossignol (advisor)**, phone # 541- 737-3840, email: [anne.rossignol@oregonstate.edu](mailto:anne.rossignol@oregonstate.edu).

If you have questions about your rights as a participant, please contact the Oregon State University Institutional Review Board (IRB) Human Protections Administrator, at (541) 737-3437 or by e-mail at [IRB@oregonstate.edu](mailto:IRB@oregonstate.edu).

Your signature indicates that this research study has been explained to you, that your questions have been answered, and that you agree to take part in this study. You will receive a copy of this form.

Participant's Name (printed): \_\_\_\_\_

\_\_\_\_\_  
(Signature of Participant)

\_\_\_\_\_  
(Date)

## RESEARCHER STATEMENT

I have discussed the above points with the participant or, where appropriate, with the participant's legally authorized representative, using a translator when necessary. It is my opinion that the participant understands the risks, benefits, and procedures involved with participation in this research study.

\_\_\_\_\_  
(Signature of Researcher)

\_\_\_\_\_  
(Date)

**CONFIDENTIALITY: Audio Recording consent**

(addendum to the general consent form)

By initialing in the space provided, you verify that you have been told that an **audio** cassette tape recording will be generated during the course of the conversation between you and the student researcher (Bernadette Mae Longo, RN) for the **Oregon State University, Kilauea Volcano Adult Health Study**.

This is voluntary and you may decide to stop at any time. Your identity will be kept confidential to the extent permitted by law. You will only be identified on the tape and in the research by your gender (woman or man), ethnicity, age range (young adult, middle aged, or elderly), and length of residency in the area. The time commitment could range from 15 minutes up to 1 hour. You may freely stop at any time.

The purpose of these research conversations is to seek to understand **your** experience of life and health. You can share more about: "How has the volcanic eruption affected your life and your health?" This is an interview that you will lead and you can share what you like. The researcher has no set questions to ask you.

There will be about eight individuals who will have similar conversations with the researcher. There may be no direct benefit to you, but the potential benefit to health care workers (doctors, nurses, and public health) can be to better understand the experience and the effects on health of individuals who have lived near Kilauea volcano. You may feel mild discomfort in sharing about your personal life and your health. Please remember, that you can stop or decline to answer any questions at any time.

Only the student researcher and her advisor, Dr. Anne Rossignol, will have access to the tape. The tape recording will be stored under lock and key until it can be transcribed (typed out while listening to the tape). When the tape has been transcribed by the researcher, it will be destroyed.

\_\_\_\_\_ Participant's initials

## Appendix III



### The Kilauea Volcano Adult Health Study Oregon State University

Thank you for participating! Please complete the following if you are an adult and have lived in the Kau district for at least 7 years:

1. How has Kilauea's eruption affected your life? *Please describe*

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2. Do you think or feel that the eruption has affected your health?

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3. Compared with other people of your same age,

would you consider your health:

- \_\_\_ excellent
- \_\_\_ very good
- \_\_\_ average
- \_\_\_ worse
- \_\_\_ very poor

4.. What is your current weight? \_\_\_ lbs.

and height? \_\_\_ft. \_\_\_ inches

<b>HEALTH SYMPTOMS</b> <i>Do you have?</i>	<b>No</b>	<b>Yes</b>	<b>I don't remember</b>	<b>Refuse/ Does not apply</b>
<b>COUGH:</b> Do you usually cough on most days, for 3 consecutive months or more during the year? *For how many years have you had this cough? ____ years				
<b>PHLEGM:</b> (the stuff you cough up) Do you bring up phlegm on most days, for 3 consecutive months or more during the year? *For how many years have you had trouble with phlegm? ____ years				
<b>WHEEZE:</b> In the past 12 months, have you had wheezing or whistling in your chest? Most days & nights? Only with a cold? During or after physical exercise?				
<b>SORE THROAT:</b> Do you have frequent sore or dry throats?				
<b>DRY COUGH:</b> Have you had a dry cough at night, not counting a cough associated with a cold or chest infection, lasting 14 days or more?				
If you get a cold, does it usually go to your chest?				
When you are not exercising, Do you ever get short of breath?				
Do you have a problem with a runny nose?				
Do you often have sinus or nose congestion? How often? _____				
Do you ever have chest pains?				
Do you have headaches often?				
When you are outside, do you have:				
A cough?				
Burning eyes?				
Runny nose and congestion?				
Shortness of breath?				
Wheezing?				
Other?(describe): _____				

<b>ILLNESS/DISEASE Diagnosed by a doctor Do you have?</b>	<b>No</b>	<b>Yes</b>	<b>I don't remember</b>	<b>Refuse/ does not apply</b>
<b>Asthma?</b> How old were you when you were first told you had asthma? ____ yrs old 1. Do you still have asthma? 2. What age did it stop? ____ yrs old 3. During the Past 12 months, have you had an episode of asthma or an asthma attack? 4. Did/do your parents or relatives have asthma?				
<b>Bronchitis?</b>				
<b>Chronic bronchitis?</b> *How old were you when you were first told you had chronic bronchitis? ____ yrs old				
<b>Chronic obstructive pulmonary disease (COPD)?</b>				
<b>Emphysema?</b>				
<b>Hayfever?</b> <b>Allergies (other than food)?</b> *What age did it start? ____ yrs old				
<b>Chronic sinusitis?</b>				
<b>Heart attack ?</b> *How old were you when you first had a heart attack? ____ yrs old				
<b>Angina? "heart pains"</b> *How old were you when you were first told you had angina? ____ yrs old				
<b>Stroke?</b> *How old were you when you first had a stroke? ____ yrs old				
<b>High Blood Pressure? (hypertension)</b>				
<b>Coronary artery disease?</b> 1. How old were you when you were first told you had coronary artery disease? ____ yrs old 2. Have you had <b>Bypass surgery</b> ?				
<b>Congestive Heart failure?</b> *How old were you when you were first told you had CHF? ____ yrs old				
<b>Pneumonia?</b> How old were you? ____ yrs old				
<b>Any Chest Illness:</b>				

5. Do you have any other **disease or illness** diagnosed by a doctor? Please List:

6. Do you take any **medications**? List: \_\_\_\_\_

**7. SMOKING:** Have you ever smoked? \_\_\_ No, \_\_\_ yes

a. Do you now smoke cigarettes? \_\_\_ No, \_\_\_ yes, how much? \_\_\_ packs/day

b. When did you stop? \_\_\_ years old

c. Do you now smoke cigars, pipe, or any other substance? \_\_\_ No, \_\_\_ yes

**8.** Have you ever *lived or worked* in an area with smoking? \_\_\_ No, \_\_\_ yes

**9.** How old are you? \_\_\_ years old

**10.** Place of Birth: \_\_\_\_\_ (city, country)

**11.** Sex: \_\_\_ Male \_\_\_ Female

**12.** Race: Native Hawaiian \_\_\_  
 Filipino \_\_\_  
 Japanese \_\_\_  
 Chinese \_\_\_  
 White \_\_\_  
 Black/African American \_\_\_  
 Other (specify) \_\_\_\_\_

**13.** What is the highest grade completed in school? \_\_\_\_\_  
 (For example: 12 years is completion of high school)

**14.** How long have you lived in the Pahala – Na’alehu area? \_\_\_ years  
 Where did you live before your current residence? \_\_\_\_\_

**15.** What is the age of your home? \_\_\_ years old.

**16.** Are you working? \_\_\_ Yes, \_\_\_ No, \_\_\_ Laid-off, \_\_\_ retired

a. Did you ever work with sugarcane? \_\_\_ yes, \_\_\_ No

b. Did you ever work in a dusty environment? \_\_\_ yes, \_\_\_ No

**17.** Do you have health insurance? \_\_\_ No, \_\_\_ Yes type: \_\_\_\_\_ (optional)

*Any Comments:* \_\_\_\_\_

*Thank you very much for your time and participation!*

**The Kilauea Volcano Adult Health Study Research Team**

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## APPENDIX IV

The Kilauea Volcano Adult Health Study  
**Aral Tungkol sa Kalusugan ng mga nasa-edad na nakatira  
 malapit sa Kilauea Bulkan**  
 Oregon State University

Thank you for participating! Please complete the following if you are an adult and have lived in the Kau area for at least 7 years:

**Maraming salamat sa inyong pakikibahagi at tulong! Sagutin po ninyo ang mga tanong kung kayo ay nasa edad at nakatira or tumira sa Kau sa pitong o labis sa pitong taon:**

**1. How has Kilauea's eruption affected your life? Anong bisa sa buhay mo ang pagputok ng bulkan na Kilauea? *Please describe Maaring ilarawan:***

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**2. Do you think or feel that the eruption has affected your health? Iniisip mo ba na ang pagputok ng bulkan an na-influensiyahan ang iyong kalusugan?**

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**3. Compared with other people of your same age, would you consider your health: **Kapag itutulad mo ang iyong sarili sa ibang tao na pareho ang edad sa iyo ang kalusugan mo ba ay:****

- ☐ **excellent / mas mabuti**
- ☐ **very good / mabuti**
- ☐ **average / karaniwan**
- ☐ **worse / masama**
- ☐ **very poor / mas masama**

**4. What is your current weight? Ano ang timbang mo sa ngayon?**

**lbs. (sukat)**

and height? **At taas? ft.  inches (sukat)**





<i>Do you have? Mayroon ka bang?</i>	No Hindi	Yes Oo	I don't remember Hindi ko matandaann	Refuse/ Does not apply Hindi ko masasagot
<b>DRY COUGH: (UBO NA WALANG PLEMA)</b> Have you had a dry cough at night, not counting a cough associated with a cold or chest infection, lasting 14 days or more? <b>Nangyari ba sa inyong magka-ubo kayo ng walang plema sa gabi or kung kayo ay walang naming sakit, na tumagal ng 14 araw o mas marami pa sa 14 (labing-apat) na araw?</b>				
If you get a cold, does it usually go to your chest? <b>Kung ikaw ay may sakit, nararamdaman mo ba sa iyong dibdib?</b>				
When you are not exercising, Do you ever get short of breath? <b>Kung hindi nag e-ehersisyo, nararamdaman mo ba na hindi o mahirap maka-hinga?</b>				
Do you have a problem with a runny nose? <b>Nangyayari ba sa iyo na palagi kang may sipon?</b>				
Do you often have sinus or nose congestion? <b>Nangyayari ba sa iyo na palaging barado ay iyong ilong?</b> How often? Ilang beses? _____				
Do you ever have chest pains? <b>Nang-yari na ba sa iyo na ang dibdib mo ay pakiramdam mo ay sumikip?</b>				
Do you have headaches often? <b>Palagi bang ang ulo mo ay sumasakit?</b>				
When you are outside, do you have: <b>Kung ikaw ay nasa labas, nang-yayari ba sa iyo ang:</b> A cough? Umobo?				
Burning eyes? <b>Sumakit ang iyong mga mata?</b>				
Runny nose and congestion? <b>Bumarado ang iyong ilong sa sipon o tumulo ang iyong sipon?</b>				
Shortness of breath? <b>Nahirapan kang huminga?</b>				
Wheezing? <b>Humuni ang iyong dibdib?</b>				
Other?(describe) (Ilarawan ang iba pa): _____				

ILLNESS/DISEASE (SAKIT) Diagnosed by a doctor <b>Nakita o na-obserbahan ng doktor</b> <i>Do you have? Mayroon ka bang?</i>	No Hindi	Yes Oo	I don't Remember Hindi ko na matandaan	Refuse/ does not apply Hindi ko masasagot
<b>Asthma? Hika?</b> How old were you when you were first told you had asthma? <b>Ilang taon ka ng ikaw</b> <b>ay magka-hika? ____ yrs old taon</b> 1. Do you still have asthma? <b>1. Mayroon ka pa bang hika?</b> 2. What age did it stop? ____ yrs old <b>2. Ilang taon ka na tumigil</b> <b>ang iyong hika? ____ taon</b> 3. During the Past 12 months, have you had an episode of asthma or an asthma attack? <b>3. Sa nakaraang taon, ikaw ba ay nagka-hika?</b> 4. Did/do your parents or relatives have asthma? <b>4. Ang iyong bang mga kamag-anak ay may hika?</b>				
<b>Bronchitis?</b> <b>Tuspirina o Bronkaytis?</b>				
<b>Chronic bronchitis? (Tuspirina o Bronkaytis</b> <b>na hindi gumaling- galling?</b> *How old were you when you were first told you had chronic bronchitis? ____ yrs old * <b>Ilang taon ka ba nang malaman mong ikaw ay</b> <b>may tuspirina o bronkaytis na</b> <b>hindi na-galing? ____ taon</b>				
<b>Chronic obstructive pulmonary disease (COPD)?</b> <b>SAKIT SA PULMON na hindi gumaling-galing?</b>				
<b>Emphysema?</b> <b>Sakit sa Baga?</b>				
<b>Hayfever? Alisis at lagnat?</b> <b>Allergies (other than food)?</b> <b>Alerji (hindi sa pag-kain?</b> *What age did it start? <b>Ilang taon ka</b> <b>na ito ay nagsimula? ____ yrs old taon</b>				
<b>Chronic sinusitis?</b> <b>Sakit sa ilong o sinus na hindi gumaling galing ?</b>				
<b>Heart attack ? Atake sa puso?</b> *How old were you when you first had a heart attack? ____ yrs old * <b>Ilang taon ka ba ng nangyari sa iyo ang una</b> <b>mong atake sa puso? ____ taon</b>				

ILLNESS/DISEASE (SAKIT) Diagnosed by a doctor <b>Nakita o na-obsorbahan ng doktor</b> <i>Do you have? Mayroon ka bang?</i>	No Hindi	Yes Oo	I don't Remember Hindi ko na matandaan	Refuse/ does not apply Hindi ko masasagot
Angina? "heart pains" <b>Sakit sa Puso?</b> *How old were you when you were first told you had angina? ____ yrs old <b>Ilang taon ka ba ng nangyari sa iyo ang sakit sa puso? _____ taon</b>				
Stroke? <b>Strok?</b> *How old were you when you first had a stroke? ____ yrs old <b>Ilang taon ka ba na ikaw ay nag-ka strok? _____ taon</b>				
High Blood Pressure? (hypertension) <b>Mataas ang presyon nang dugo?</b>				
Coronary artery disease? <b>Sakit sa ugat nag puso</b> 1. How old were you when you were first told you had coronary artery disease? ____ yrs old <b>1. Ilang taon ka ba ng ikaw ay nag-kasakit sa ugat ng puso? _____ taon</b> 2. Have you had Bypass surgery? <b>2. Na-operahan ka na ba sa iyong puso?</b>				
Congestive Heart failure? <b>Barado na ang puso?</b> *How old were you when you were first told you had CHF? ____ yrs old <b>*Ilang taon ka ba nang ma-barado ang iyong puso? _____ taon</b>				
Pneumonia? <b>Numonya?</b> How old were you? ____ yrs old <b>Ilang taon ka bang nagka-numonya? _____ taon</b>				
Any Chest Illness: <b>Iba pang sakit sa dibdib?</b>				

6. Do you have any other disease or illness diagnosed by a doctor? **Sinabi ba ng doktor na ikaw ay may sakit? Ano ba ang iyong sakit? Please List Maaring I-lista o ipahayag dito:**

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7. Do you take any medications? **Kayo ba sa ngayon ay umi-inom ng gamot List Maaaring ilista o ipahayag:**

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#### 8. SMOKING: NANINIGARILYO:

Have you ever smoked? **Ikaw ba ay nanigarilyo? \_\_\_\_ No Hindi, \_\_\_\_ yes Oo**

Do you now smoke cigarettes? **Ikaw ba ay na-ninigarilyo? \_\_\_\_ No Hindi, \_\_\_\_ yes Oo**

a. How much? **Ilang beses sa isang araw \_\_\_\_ paketes/araw packs/day**

b. When did you stop? **Kailan ka tumigil? \_\_\_\_\_ years old taon**

c. Do you now smoke cigars, pipe, or any other substance?

**Ikaw ba ay na-nabako? \_\_\_\_ No Hindi, \_\_\_\_ yes Oo**



## APPENDIX V

### Pulmonary Function: Spirometry

Spirometry procedure and guidelines as described by the American Thoracic Society (1991, 1995) were followed in this study. The following is a description of the procedure:

1. Selection of subjects: (based on *epidemiology* confounders, or safety measures to *minimize risk* to subjects)
  - Adults, 20 - 70 years of age. *Minimize risk*
  - Never-smokers (Pope, 2000) and former smokers *Epidemiology*
  - No mid-lower respiratory disease diagnosis (asthma, emphysema, chronic bronchitis, T.B. or other) *Minimize risk, Epidemiology*
  - No cardiac disease diagnosis (MI, angina, other cardiac, taking cardiac medications) *Minimize risk*
  - No acute cardiac symptoms (chest pain, shortness of breath, dyspnea) *Minimize risk*
  - No abnormal pulse rate/rhythm or elevated BP for age. *Minimize risk*
  - No acute respiratory illness (congestion, cold, flu, hay fever is acceptable unless experiencing chest tightness) *Minimize risk*
  - No reported acute fatigue prior to or during procedure *Minimize risk*
  - No fever per subject report *Epidemiology*
  - No mouth sores or hand wounds per observation *Minimize risk*
  - No other disease process affecting respiration or ability to do spirometry (multiple sclerosis, scoliosis) *Epidemiology*
2. Consent: a special separate consent form was signed by spirometry subjects.
3. Subject Education: Each subject was given an explanation of the procedure, allowed to ask questions, and demonstrated by role modeling of the procedure. Subjects were encouraged to breath deep and exhale into the machine quickly, forcefully and completely.
4. Testing Position: Standing on feet, with no hyper-flexion of neck.
5. Accuracy: The execution of three acceptable spirometric procedures or blows into the machine.
6. Analysis of test: Comparison of subject data to predicted values in machine for Caucasian or Asian. According to the ATS (1991), differences in race can be observed for the FEV<sub>1</sub>, but similarities exist in the FEV<sub>1</sub>/FVC %.
7. Risks: noninvasive procedure. Subject's oral contact on surface of machine. Requires subject's physical effort and participation to blow into machine.
  - a. Disposable mouth pieces were used to prevent oral transmission of disease. The instrument had a one-way valve. The instruments exterior was cleansed with alcohol swabs between households. The data collector washed hands before the procedure was conducted. Subjects with mouth

- sores or open hand wounds, or acute respiratory illness were excluded to prevent any possible disease transmission.
- b. Repeated maximal efforts can cause acute bronchoconstriction in some subjects, affecting their reading, and health status (ATS, 1991). Therefore, only a maximum of four spirometry maneuvers were allowed (ATS allows up to eight, unless fatigue occurs). In addition, the above criteria for subject selection and the consent were made to exclude high-risk subjects.
  - c. The procedure would have been stopped with any symptoms of physical distress observed or reported from the subject.
8. Personal health: A researcher signed copy of the spirometry data was given to the subject at the time of testing, allowing him/her to share the results with their health care providers.

Data was collected in May-June of 2004, using a Spiromate AS-500 Spirometry instrument from Riko Medical & Scientific Instruments, U.S.A. All participants signed consents and were free of any respiratory symptoms or illnesses. Participants were never smokers and one former smoker. Residency (Exp = exposure) times are indicated. Results indicated that two participants had some restrictive disease as measured by the Forced Expiratory Flow between 25% and 75% of the exhalation (FEF 25-75). Otherwise all pulmonary measurements are within normal range.

Participant Description	Exp yrs	FVC	FVC%	FEV <sub>1</sub>	FEV <sub>1</sub> %	FEV <sub>1</sub> /FVC%	FEF 25-75	PEF%
SO <sub>2</sub> Never	21+	5.82	117.1	4.52	109.4	77.7	78.3*	107.8
SO <sub>2</sub> Never	21+	3.29	119.2	2.8	119.1	85.1	124.2	111.5
Sulfate Never	10	3.87	98.9	3.4	102.1	87.8	98.9	94.5
Sulfate Former	12	3.73	108.7	3.12	111.4	83.6	142.4	120.0
Sulfate Never	9	3.83	101.3	2.92	91.8	76.2	72.9*	139.5
Sulfate Never	21+	6.6	114.5	4.97	111.4	75.3	104.7	115.0
Control Never	21+	3.23	106.2	2.72	99.6	86.9	92.7	97.0

## The Kilauea Volcano Adult Health Study

### CONSENT FOR SPIROMETRY (This is an addendum to the primary consent).

The spirometry test measures the health of your lungs. The spirometer is a small instrument that you blow into (exhale). It measures how air passes out of your lungs. This test can tell if air flows easily out of your lungs or if it is blocked. It also measures how much air your lungs can hold.

The information you provide will be measures from the spirometer. Your results will be compared to average measures for someone of your age and size (normal values). These measures will be grouped into the adults living near Kilauea volcano in Pahala and Na'alehu, or away from the volcano in Hawi. The measures will be compared to see if there are any differences between the two areas.

The purpose of this consent form is to give you the information you will need to help you decide whether to participate in spirometry or not. Please read the form carefully. You may ask any questions about spirometry. When all of your questions have been answered, you can decide if you want to participate in spirometry. You will be given a copy of this form for your records.

We are inviting you to participate in this part of the research study because you are an adult, between **20 – 70 years old**, and you agree with the following statements:

1. I have **never smoked**.
2. I do not have any problem with my heart or lungs.
3. I am in **good health**.
4. A doctor or nurse has never told me that I have a bad heart or breathing problem.
5. I do not have asthma.
6. I have never had a heart attack.
7. I do not take medicine for my heart or lungs.
8. I do not have a disease that I can pass to someone else (like TB).
9. I do not have any pain in my chest or heart, shortness of breath, painful breathing, or tightness in my chest.
10. I am not tired right now.

Please initial here that you agree with the above: \_\_\_\_\_

### PROCEDURES

If you agree to participate, your involvement will last for **5 minutes**.

The following procedures are involved in this study:

1. You will take a deep breath into your lungs; put your mouth to the instrument and blow out fast, forcefully and completely.
2. You will do this procedure three (3) times, resting for a minute or two in between the blows.
3. You will tell the researcher if you feel **any discomfort, pain or weakness** at any time. The spirometry will stop if necessary.
4. The researcher will write on a piece of paper the results of your spirometry. You may take this information to your doctor if you wish.

### RISKS

The possible risks associated with participating in the spirometry test are as follows:

1. Catching a disease by the instrument's mouthpiece. To prevent this from happening, you will use a disposable mouthpiece, not used by any other person. The instrument has a valve that doesn't allow air back at you. The researcher will wash her hands before the procedure and clean the instrument between households.



2. Effort to blow into the machine forcefully could cause a narrowing of your breathing tube or stress on your heart. Narrowing of your breathing tube is called bronchoconstriction. People who have heart problems may have stress on their heart when performing activities that require physical effort, such as spirometry. Bronchoconstriction and stress on the heart can be life threatening. To prevent this from occurring you were asked the screening questions on page 1. Again, **you should decline the spirometry test if you think you have any heart, blood pressure, lung or breathing problems.**

### **BENEFITS**

The potential personal benefits that may occur as a result of your participation in this study are to receive a copy of the spirometry measures that you may take to your doctor. The researchers anticipate that society may benefit from this study by learning about the lung health of non-smoking adults living near an active volcano, and by comparing the spirometry to another community away from Kilauea volcano.

### **RESEARCH RELATED INJURY**

In the event of research related injury, compensation and medical treatment is not provided by Oregon State University.

### **VOLUNTARY PARTICIPATION**

Taking part in the spirometry for the research study is **voluntary**. **You may choose not to take part at all.** If you agree to participate in this study, you may stop participating at any time. If you decide not to take part, or if you stop participating at any time, your decision will not result in any penalty or loss of benefits to which you may otherwise be entitled.

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Your signature indicates that this research study has been explained to you, that your questions have been answered, and that you agree to take part in this study. You will receive a copy of this form.

Participant's Name (printed): \_\_\_\_\_

\_\_\_\_\_  
(Signature of Participant)

\_\_\_\_\_  
(Date)

### **RESEARCHER STATEMENT**

I have discussed the above points with the participant or, where appropriate, with the participant's legally authorized representative, using a translator when necessary. It is my opinion that the participant understands the risks, benefits, and procedures involved with participation in this research study.

\_\_\_\_\_  
(Signature of Researcher)

\_\_\_\_\_  
(Date)

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