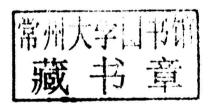
BREATHIG

Smoking, Pollution and the Haze

ANTHONY REBUCK

BREATHING POISON

Smoking, Pollution and the Haze



ANTHONY REBUCK



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וַיָּפָּח בְּאַפָּיו נִשְׁמֵת חַיִּים וַיְהִי הָאָדָם לְנֶפֶשׁ חַיָּה

And He breathed into his nostrils the soul of life, and man became a living soul.¹

Genesis: chapter 2, verse 7

With many thanks for their help, guidance and input to:

Mark E. Cooper and Michael S. Rabieh

INTRODUCTION

On May 18, 1980, south-western Washington State and north-western Oregon State in the USA were showered with volcanic ash as Mount St. Helens violently erupted. By world standards, there had been many worse eruptions: Santa Maria in Guatemala, Krakatoa in Indonesia and Mount Katmai in Alaska, for example. Nevertheless, Mount St. Helens was a big one, estimated to be comparable with the serial detonation of 27,000 atom bombs at a rate of one per second for nine hours. An article in the prestigious journal *Scientific American* described Mount St. Helens as having released energy equivalent to 100 times the generating capacity of all U.S. power stations.

The 42 people who died in the vicinity of the terrible eruption asphyxiated from inhalation of volcanic ash. The ash mixed with mucus in their lungs and completely plugged their upper airways. Two people staggered away from the mountain and subsequently died in hospital from purulent tracheabronchitis attributable to inhalation of volcanic ash, hot volcanic gases and particulate matter. Naturally, considerable concern was expressed as to the long-term health and respiratory effects of the disaster.

From the scientific and medical perspective, the response was immediate and thorough. An epidemiological team from the U.S. Centres for Disease Control (CDC) arrived immediately and teamed up with the Washington State Department of Social and Health Services to evaluate the potential health effects. Twenty-seven hospitals in the area were surveyed for their statistics on emergency room visits and admissions for respiratory illnesses. Professor Sonia Buist, one of the most respected pulmonary physicians in the world, happened to be practicing in the region, and she lent her energy and expertise to arranging a conference among all the investigators in 1982 entitled: "Are Volcanoes Hazardous to Your Health? What Have We Learned from Mount St. Helens?"

We have learned a great deal, but in terms of long-term effects, the somewhat surprising finding was that the likelihood of permanent lung damage from silicosis was found to be extremely remote. Exposures were neither high nor long enough to cause long-term harm in the general population; in particular, there was no increased risk of Chronic Obstructive Lung Disease (COPD).

The Mount St. Helens episode indicated that it is not sudden environmental disasters that most endanger human breathing but, rather, more mundane, omnipresent threats that rarely grab the headlines. These omnipresent threats, that is to say smoking and pollution, are responsible for the fact that COPD has risen from seventh leading cause of death and disability in the world in 1996 to fifth at the present time, and predicted by the World Health Organization to be in third place by the year 2020. Tens of millions of people are suffering from moderate to severe COPD with at least three million people dying of it every year. Literally, hundreds of millions of people in the world are suffering from at least the early form of a disease caused solely by the air they breathe.

The disease is treatable and preventable, but remains incurable. For too long the impact of COPD has been underestimated, poorly understood and misdiagnosed.

The growing health care concern regarding death and disability from COPD will affect Asia more than anywhere else in the world. The air has become poisoned in many places by

cigarette smoking, automobile and industrial pollution and the haze from illegal wildfires on the Indonesian island of Sumatra. Smoking is the main factor responsible for the development of COPD. The number of smokers is the highest in the world in the Western Pacific Region and China, and continues to rise there, the increase of COPD within Asia will undoubtedly be dramatic.

It is hoped that this book will be a small contribution to public awareness and educational initiatives about the risk of COPD.

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CHAPTER 1

Breathing Poison

The advice given by public health officials at the time of the Mount St. Helens eruption was reasoned and appropriate, though not based on scientific evidence because little or none was available at the time. There was no published literature on the effect of volcanic ash on health in May 1980, although such evidence quickly accumulated after the event. People were advised to minimize exposure to ash by staying indoors whenever possible, and to use masks approved by the National Institute of Occupational Safety and Health when outside in the ash. In addition to this sage advice—which is still appropriate in today's battle against smoke haze—the public was told that deposition of ash in the lungs could be minimized by nose breathing.

There is a lot to recommend nose breathing. Indeed, much has been written on the topic, all the way from the second chapter in the first book of the Bible: "He breathed the breath of life into the man's nostrils." The nose warms, moistens and filters air before it reaches the lungs and, by being our only sensor of smell, can warn of noxious odorous gases that we

might otherwise be in danger of inhaling. The olfactory nerve, or cranial nerve number one, is derived from an embryonic structure known as nasal neural placode. The nerve is so important that it is capable of regeneration if damaged and is the only nerve in the body whose receptor neurons are exposed to the environment, lying bare in the nasal mucosa. Among its other unique features are that it is the shortest of all the twelve cranial nerves and, other than the optic nerve, is the only one that does not join the brain stem but travels directly into the brain. The pseudo-stratified columnar epithelium, otherwise known as the respiratory epithelium of the nose, together with cilia and mucus along the inside wall of the nasal cavity, were ideally "designed" to trap and remove dust from the Mount St. Helens air, which, by any standard, was laden with particles. The particles would have moved down the nasal cavity to the pharynx, where they would have been swallowed or spat out.

The earliest reports of the content of the ash suggested that it contained 60 per cent free silica, enough to lead respiratory disease specialists, present company included, to predict publicly a resulting epidemic of silicosis and tuberculosis. We were wrong, as it turned out, as were the initial reports of the content of the ash. In the initial period of limited information, fears were also expressed about the carcinogenic (cancer-causing) properties of the ash, and there was speculation as to whether it contained radon gas. Eventually, accurate information emerged. The ash particles were on the whole less than 10 micrometres (µm) in size, small enough to penetrate the length of the airways, right down to the alveoli-or air sacks-of the lungs. The good news was that the free silica content of the ash contained approximately 6 per cent free silica, not 60 per cent as originally reported in the media, and of the 6 per cent, 2 per cent was quartz and 4 per cent, cristobalite.

Any long-term damage from the volcanic dust would depend on the composition of the ash, how much was inhaled, the size of the particulates that were inhaled, and the presence or absence of pre-existing lung disease. The exposure to crystalline silica at 6 per cent content, with concentrations ranging from 0.8 to 1.25 mg/m³, as it turned out, was unlikely to cause silicosis. For the most part, except for the rescue workers on the mountain itself, the general population received only limited exposure, due mainly to the air-cleansing effects of rain, weathering, and the ash becoming worked into the topsoil. Those who were heavily exposed in the first few days experience irritation and inflammation of the upper and lower respiratory tracts, and exacerbation of asthma in those with hyperactive airways. Aside from irritated eyes and a variety of psychological symptoms related to stress, it appears likely that to the extent anyone suffered long-term respiratory consequences, those consequences were quite limited, primarily because the ash deposits were rather modest.

In Asia, the combination of toxic haze from fires, unacceptably high levels of pollution, and pervasive smoking gravely threatens the pulmonary health of tens of millions of people: in sharp contrast to Mount St. Helens, the disabilities resulting from these threats are likely to be permanent and in many cases fatal. As Emerita Professor Sonia Buist and colleagues from Oregon stated (Am J Public Health. 1986), after studying more than 9,000 people over the age of 40 from twelve different countries, "Chronic Obstructive Pulmonary Disease (COPD) is the cumulative response of the lungs to the burden of all that is breathed over a lifetime." Let us consider what it is that people in Asia are breathing today, and the devastating effect it is going to have on their health and well-being.

HAZE

By contrast with the paucity of knowledge that was available about the health effects of volcanic ash before the Mount St. Helens eruption, a great deal of information about haze has been published and was available long before the shocking South-east Asian haze of 2013.

Haze is biomass smoke that arises as a consequence of forest fires and the burning of other vegetation (whether living or dead) and of particles previously deposited on leaves during land-clearing activities. The immediate effect of burning is the release into the atmosphere of gases and particulates, dominated by submicron-size (0.05-0.9 µm) particles that scatter incoming solar radiation and thereby reduce atmospheric visibility and alter regional climate. Since a major outbreak of haze in 1997, Singaporeans have known that acidic aerosols and gases in haze are associated with increased hospital admissions for asthma and other respiratory ailments. Compared with the 2013 haze, the hazes of the 1990s were relatively mild. In 1997, the Pollutant Standards Index (PSI) peaked at a record level of 226. At midday on Friday June 21, 2013, however, Singapore experienced a PSI of 401; a level described in Singapore government guidelines as "life-threatening to ill and elderly persons." Across the Strait of Malacca, just up-wind of Singapore in Sumatra, the PSI reached a shocking 900.

The PSI tells only part of the story, however. Haze contains a complex mixture of particles as well as acidic gases, and Southeast Asia has been experiencing haze from man-made activities with increasing frequency since 1960, with particularly severe episodes occurring in 1990, 1991, 1994, 1997 and 1998. In 1990, haze caused peninsular Malaysia to suffer reduced visibility in the middle of August for at least two weeks. The haze of 1991 was even worse; the region experienced both a volcanic eruption from Mount Pinatubo and a biomass burning that persisted through most of September and October. In 1994, smoke affected 3 million square kilometres in Malaysia, Singapore and Indonesia. Concentrations of sulphur, potassium, titanium, vanadium, manganese, nickel, arsenic and lead in the air were six times higher than average during the haze period in October.

The rest of the world sat up and took notice of the haze of 1997. First, it lasted from May until November. Second, it impacted Malaysia, Singapore, Thailand and the Philippines, not