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Effect of volcanic gas exposure on urine, blood, and serum chemistry

Michael Durand, Chris Florkowski, Peter George, Trevor Walmsley, Phil Weinstein

Abstract

Aims This pilot study tested the hypothesis that aluminium (Al), rubidium (Rb), arsenic (As), lead (Pb), mercury (Hg), fluorine (F), and chlorine (Cl), which are all known to be present in volcanic emissions, may be useful biological markers for occupational gas exposure in volcanologists.

Methods Ten human subjects were exposed to fumarole gases on White Island, New Zealand, for ~20 minutes. Sulphur dioxide (SO_2) exposure was recorded by personal monitoring tubes. Pre- and post-exposure urine, blood and serum samples (collected using standard protocols) were analysed in the pathology laboratory for trace element and halogen content.

Results Average personal exposure was measured at <75 ppm SO_2 and calculated at ~25ppm HCl, ~8 ppm hydrogen fluoride (HF), ~1 ppm Al, ~0.1 ppb Rb and ~4 ppb Pb. These concentrations almost certainly exceed those usually found in occupational exposure settings. Advanced levels of urinary Al and Rb were found following gas exposure and were statistically significant in the population at $p<0.005$ and $p<0.001$, respectively. The other chemical elements that were analysed (urinary Cl, F, and Hg; blood Pb, and serum Al) did not show such patterns.

Conclusions It is possible that urinary Al and Rb may be useful markers for exposure, a hypothesis which should be followed up in future work.

Volcanic emissions include a suite of toxic gases including sulphur dioxide (SO_2), carbon dioxide (CO_2), hydrogen sulphide (H_2S), hydrogen chloride (HCl), and hydrogen fluoride (HF)—plus aluminium (Al), arsenic (As), mercury (Hg), lead (Pb) and titanium (Ti) in gaseous and molecular aerosol form. The very high gas concentrations which may easily be encountered on active volcanoes mean that researchers may risk the development of reactive airways dysfunctional syndrome or other forms of occupational asthma if they do not habitually use respirators with acid gas cartridges. Despite this, volcanologists in the field rarely monitor occupational exposure to harmful gases.

Biological monitoring following exposure to potentially harmful gases and respirable particles is common in industry, but this is almost unheard of in volcanology. In this report, we present findings from preliminary work on White Island (New Zealand's most active volcano at present), which investigated the value of using blood, serum, and urine markers to study or monitor occupational exposure to volcanic gases.

Methods

White Island volcano—White Island lies in the Bay of Plenty 48 kilometres offshore at the northern end of the Taupo Volcanic Zone in the North Island of New Zealand. The generally mild activity between eruptions and the ease of access by boat made it an ideal natural laboratory for this work. Over

recorded history (1826–present), activity has been characterised by gas emissions from fumaroles (surface vents emitting volcanic gases, fed by a sub-surface hydrothermal system) and mud pools, punctuated by episodes of weak-moderate eruptions, which typically last for several months.[1,2] During the current work (February 2002) we observed moderate-strong gas emissions from fumaroles and the main vent inside the crater complex.

Gas emissions from the volcano are typical of other volcanoes of the same magma type (andesite), at ~430 tonnes per day (t d^{-1}) SO_2 and ~1550 t d^{-1} CO_2.[3] The emission of metals as gas or aerosols is significant and includes ~6000 kg d^{-1} Al, 600 kg d^{-1} Rb and 16 kg d^{-1} As.[4]

Subjects in this experiment were exposed to gases from one of three fumarole complexes in the central subcrater. All three are on the very lower slopes of the crater walls. Presently we cannot determine the elemental concentrations in the gases from each of the fumaroles, but these values are known for the main vent plume at White Island, which is fed by the same hydrothermal system as the fumaroles.[5]

Exposure and sampling procedure—Subjects (average age 27 years; no asthmatics and no smokers included) were on the volcano for 2 hours and each spent 20 minutes close (<10 metres) downwind from one of the three fumaroles, carrying masks around their necks but only using them if deemed necessary. Average exposure during this period was monitored with SO_2 diffusion tubes worn on clothing (Gastec Dositubes no. 5D).

The 'pre-exposure' (control) blood and urine samples were taken the day before the visit to the volcano, using standard protocols (urine sample period: 3.5 hours). The 'post-exposure' sample period began upon arrival at the volcano (and included the period of gas exposure itself), and ended after 4.75 hours when blood samples were taken.

Ethical approval for the work was given by Canterbury Ethics Committee, and participants gave informed consent after the implications of the work were explained.

Analytical methods—Urine chloride was measured using Integrated Chip Technology on an Aeroset Biochemistry Analyser (Abbott Laboratories, Abbott Park, IL 60064, USA). Fluoride was measured by Ion Selective Electrode (Orion Research Inc, Beverly, MA 01915–6199, USA). Urine aluminium, serum aluminium, and blood lead were measured on a Varian AA40 Graphite Furnace Atomic Absorption Spectrometer, and the urine rubidium was measured on a Varian AA100 Flame Atomic Absorption Spectrometer (FAAS) (Varian Techtron Pty Ltd, Mulgrave, Victoria 3170, Australia). Urine mercury was analysed by cold vapour generation using a Perkin Elmer Flow Injection analysis system attached to a Perkin Elmer A100 FAAS (Perkin Elmer Instruments, Norwalk, CT 06859–0010, USA). Creatinine was measured using the Jaffe reaction on an Aeroset Biochemistry Analyser.

Statistics—Wilcoxon's signed-rank test for matched pairs[6]—a non-parametric test for dependant variables—determined the statistical significance of post-exposure changes in blood, serum, and urine chemistry.

Outliers were defined by meeting both of the following standard criteria:

- By lying three standard deviations above the group mean, and
- By lying within the 75th percentile by at least 1.5 × the interquartile range.

Results

Exposure

During the acute exposure period near the fumaroles, SO_2 tubes recorded exposure between 6 parts per million (ppm) (Subject 3) and 75 ppm (Subject 4), with a group mean of 24 ppm. The recorded SO_2 exposure and calculated HF, HCl, Al, Rb, and Pb exposures are shown in Table 1. Mean exposures were calculated at ~2.5 ppm HF, ~8.5 ppm HCl, ~0.5 ppm Al, ~0.05 parts per billion (ppb) Rb, and ~1.5 ppb Pb. Since detailed chemistry of each fumarole on the volcano is not known, these calculations are subject to error perhaps in excess of 50%. Personal exposure away from the fumaroles was not measured but likely to be below 1 ppm SO_2.

Table 1. Average SO_2, HF, HCl, Al, Rb, and Pb exposure concentration recorded by diffusion tubes worn on subjects' clothing

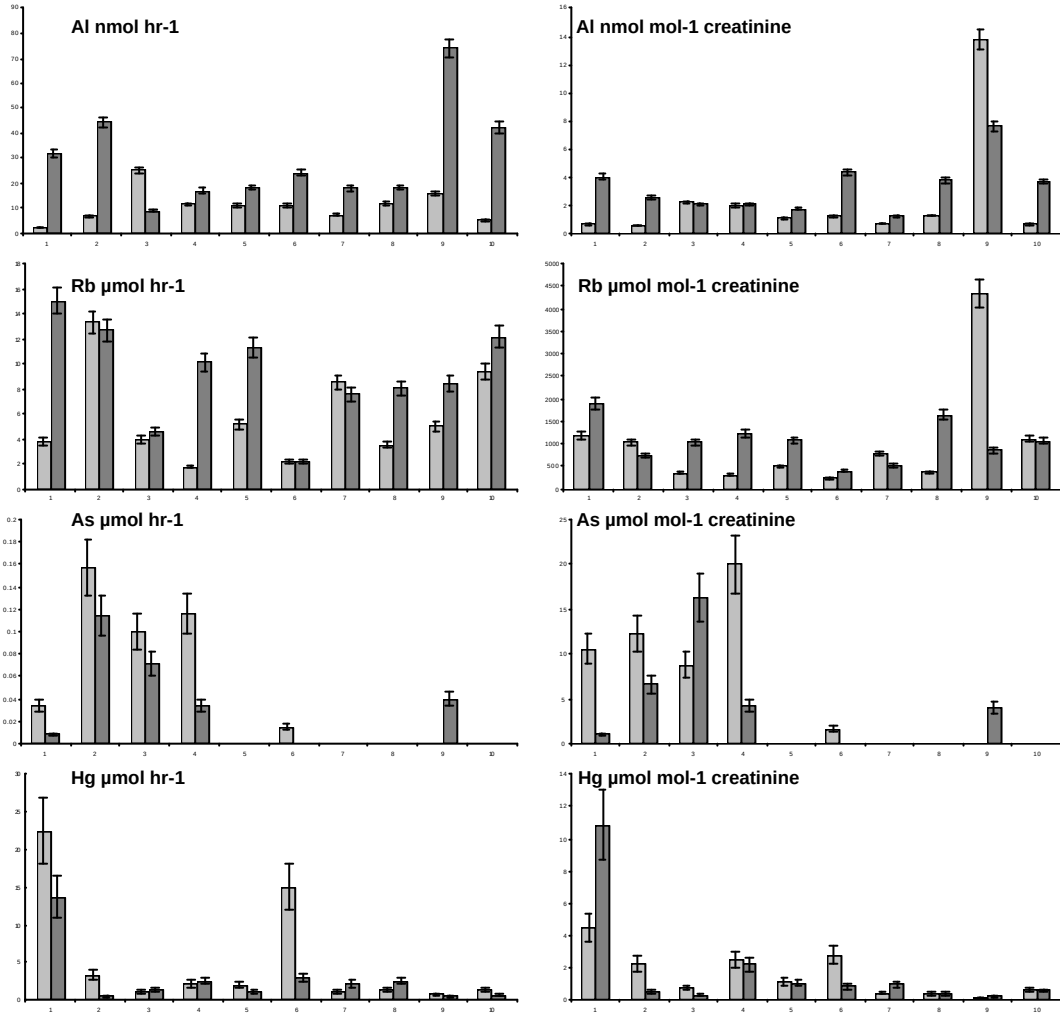
Subject No.	SO_2 ppm*	HF ppm[(a)]	HCl ppm[(a)]	Al ppm[(b)]	Rb ppb[(b)]	Pb ppb[(b)]
1	15	1.6	5.2	0.3	0.03	0.9
2	12	1.3	4.2	0.2	0.02	0.7
3	6	0.6	2.1	0.1	0.01	0.4
4	75	7.8	25.9	1.3	0.13	4.6
5	21	2.2	7.3	0.4	0.04	1.3
6	36	3.7	12.4	0.6	0.06	2.2
7	15	1.6	5.2	0.3	0.03	0.9
8	24	2.5	8.3	0.4	0.04	1.5
9	24	2.5	8.3	0.4	0.04	1.5
10	15	1.6	5.2	0.3	0.03	0.9

* Recorded by diffusion tubes. (a) Calculated from SO_2:HCl and SO_2:HF ratios in plume[1]; (b) Calculated from Al, Rb, Pb:SO_2 ratios in plume[4]

Table 2. Post-exposure changes in elemental outputs in urine, serum, and blood (statistically significant results in bold)

Element and analyte	Mean pre-exposure	Mean post-exposure	Mean change	Standard deviation
In urine				
Chloride mmol L^{-1} urine	128.5	84.4	–44.10	90.91
Chloride mmol hr^{-1}	47.56	46.58	–0.97	8.51
Chloride mmol mol^{-1} creatinine	7.79	5.35	–2.46	7.21
Aluminium µmol L^{-1} urine	0.03	0.063	**+0.03**	0.03
Aluminium µmol hr^{-1}	0.010	0.029	**+0.018**	0.0047
Aluminium µmol mol^{-1} creatinine	2.39	3.30	**+0.9**	2.82
Fluoride µmol L^{-1}	26.29	30.77	+4.48	19.01
Fluoride µmol hr^{-1}	11.25	13.93	+2.67	1.83
Fluoride mmol mol^{-1} creatinine	1.76	1.71	–0.05	1.53
Mercury nmol L^{-1}	5.09	2.82	–2.27	4.52
Mercury nmol hr^{-1}	1.56	1.800	+0.24	0.44
Mercury µmol mol^{-1} creatinine	0.28	0.22	–0.06	0.08
Rubidium µmol L^{-1}	14.54	19.25	+4.7	14.6
Rubidium µmol hr^{-1}	5.66	9.23	**+3.56**	1.17
Rubidium µmol mol^{-1} creatinine	1020.53	1049.53	**+29.1**	13.36
In serum				
Aluminium µmol L^{-1}	0.064	0.062	–0.002	0.018
In blood				
Lead µmol L^{-1}	0.13	0.14	0.01	0.03

Figure. 1. Al, Rb, As, and Hg outputs in urine. Outputs are shown as paired values (pre- and post-exposure) in each subject (1–10) as the excretion rate (µmol or nmol hr^{-1}) and the ratio of each element to creatinine (µmol or nmol mol^{-1} creatinine)



Trace elements and halogens in urine, blood and serum

Positive changes in the population mean outputs were seen in urine Al (all measures), urine Rb (all measures), urine F (micro-moles per litre of urine; µmol L^{-1}), and blood Pb (µmol L^{-1}). In the case of Al µmol L^{-1}, this shift was statistically significant with $p<0.005$; in Al µmol hr^{-1}, the positive change was significant with $p<0.025$; relative to creatinine, Al micro-moles per mol of creatinine (µmol mol^{-1}) increased across the population with $p<0.005$. Rb results were also statistically significant, with Rb the rate of output (micro-moles per hour; µmol hr^{-1}) increasing across the population with $p<0.025$; the population increase in Rb excretion mol^{-1} creatinine was signficant at $p<0.001$. Figure 1 shows individual results for urine Al and Rb.

Urine F and blood Pb showed positive changes in the population as a whole, but these changes were inconsistent across the population and were not statistically significant.

Analyses of other elements and analytes failed to show any clear positive or negative trends and none of the changes were statistically significant.

Discussion

Acute exposure to toxic gases and aerosols on volcanoes has rarely been properly documented, but can be high enough to cause acute illness and deaths from gas exposure have occurred. In the crater of Vulcano, Italy, for example, Baxter et al[7] measured concentrations of H_2S 60–150 ppm, HCl >10 ppm and HF 3–15 ppm close to active fumaroles where scientists were working. These concentrations exceed 10 minute occupational exposure limits by 2–15 times;[8] scientists worked near the active fumaroles for ~4 hours, wearing respirators about half the time. Prior to this study, two geologists had lost consciousness in the crater at Vulcano.[7] In 1997, four hikers died from H_2S poisoning in the Numano-taira crater on Adatara volcano in Japan.

Thus, volcanologists potentially face acute and chronic health risks when visiting actively degassing volcanoes. In this work, we aimed to simulate conditions of acute gas exposure experienced by volcanologists in active craters who were not always using gas masks. The period and intensity of exposure in this experiment could be at the lower end of that encountered by some volcanologists. However, America's National Institute for Occupational Safety and Health (NIOSH) 10-minute exposure limit for SO_2 (5 ppm; ref. 8) was exceeded by at least a factor of 2 in 9 of the subjects; and in the case of subject 4, by ~30 times.

This work is thought to be the first of its kind on an active volcano, but numerous proxy studies of industrially, or accidentally, exposed workers have been completed. Al exposure is common in metal processing industries, and numerous studies have found enhanced Al levels in serum and urine of occupationally exposed workers.[9–14] Pierre et al[15] stressed that the pattern of Al excretion rate varies between Al in different molecular forms, but at present we have no way of determining the form of Al in the White Island gases. Urine does, however, account for >95% of excreted Al in people with a healthy renal system, and is rapidly excreted.[15, 16]

Our results indicate an increased rate of urinary Al output in 9 of the 10 subjects (up to 70 nmol hr^{-1}; Subject 9), which was significant in the population to $p<0.025$. The population's enhanced Al concentrations in urine were significant at $p<0.005$. Subject 9 had the highest Al concentration in urine following exposure, at 0.15 µmol L^{-1}, twice that in the pre-exposure sample. This is, however, well below the mean urine Al reference value for unexposed individuals (m 0.237 µmol L^{-1}; r 0.04–6.2 µmol L^{-1}) determined by White and Sabbioni.[17] Al increased relative to creatinine in seven subjects by a factor of 1.6–64. The mean change in Al was 1700 nmol mol^{-1}, which was significant at $p<0.005$.

Given the rapid and thorough excretion of Al in urine, there is no reason to suspect the post-exposure signal is an artifact, or the result of a confounding factor involved prior to the study period. Although the influence of an unknown confounding source cannot be ruled out, the fact that Al was elevated in 8 of the 10 subjects suggests a common source for this change, rather than the unlikely effect of sources which act upon individuals (e.g. mobile Al in food, drinks, toothpaste or deodorants). Indeed, pulmonary Al absorption is more efficient than gastrointestinal absorption, and little is known of the bioavailabilty of Al in food and drink, except in water.[16]

Little is understood of the bioavailability, kinetics and excretion of Rb. Our results indicate statistically significant shift in the population's rate of Rb output (μmol hr^{-1}), at $p<0.025$. Cornelis et al[18] gathered the most recent reference values of which we are aware, indicating urinary Rb outputs of 0.5–1.95 μmol hr^{-1} are normal in healthy, unexposed individuals. Pre-exposure mean for our group, 1.61 μmol hr^{-1}, lies in the upper part of this range. The post-exposure mean, 2.64 μmol hr^{-1}, exceeds this reference range. Seven of the 10 subjects lay within this reference range prior to exposure; following exposure, only 2 lay within it; 8 subjects exceeding reference value outputs. In the population, this increase in Rb output μmol hr^{-1} was significant at $p<0.025$. The greatest change was in Subject 1, whose post-exposure urine Rb output was 4.3 μmol hr^{-1}, or more than twice the maximum reference values acquired by Cornelis et al.[18] Relative to creatinine, the group's mean positive change in Rb was 417 μmol mol^{-1}, which was significant at $p<0.001$.

If all these data are accurate, then the statistically significant population change in Rb to above reference value limits is potentially very important. Judgment should be made with caution though, especially with regard to the rather old reference values, which are the newest we believe have been published.

Although post-exposure increases in urinary Al and Rb outputs were statistically significant, there was not a clear correlation between excretions of the two elements. Reasons for these discrepancies may be related to a number of factors including differing levels of exposure or perhaps personal differences in renal function. Indeed, in the group as a whole, exposures recorded by the diffusion tubes did not correlate with the observed changes in urinary outputs. A clear correlation should not be expected, however, since respiratory absorption of gases and particles may vary considerably between individuals. It is also possible the tube readings were not an accurate indication of true respiratory exposure. Further uncertainties exist because it was necessary to estimate metal outputs from the fumaroles by using data from other vents.

In contrast to the Al and Rb results, other elements studied here showed little relation between likely exposure and a response in urine or blood. Many studies have now shown the value and reliability of using fluoride concentrations in urine or plasma to evaluate the influence of environmental fluorine compounds.[15,19–22] These studies fall into two categories, in which occupational time-series exposures or laboratory controlled exposures (usually short term; hrs) are analysed. In the former case, Kono et al[20] found that occupational exposure to 2 ppm HF in air caused a statistically significant post-exposure change in urine F outputs.

Ehrnebo and Ekstrand[23] found a positive shift in plasma F concentrations which correlated with exposure measured by personal monitors. Tangible health effects have also been observed in people exposed in the work place or the laboratory. In studies of F exposure and respiratory symptoms, Søyseth et al[21] and Lund et al[22] both found statistically significant associations between exposure, plasma F concentrations and impaired respiratory function. Collectively, these studies suggest the current work might have found a positive response in urinary F outputs following exposure—tube results suggested subjects were exposed to ~1–8 ppm HF during the experiment.

To the contrary, we found no statistically significant post-exposure shift in urinary F by any measure. Only in Subjects 1 and 7 was the elevated total output accompanied

by increased F concentration in urine (μmol L^{-1}); in both these cases, the increased concentration was negligible.

Blood Pb has been observed to correlate with air Pb in occupational and other settings,[24,25] but here there was no uniform nor statistically significant change in concentrations in our results. Mean population concentration was unaffected by the exposure. Subjects 6 and 8 (both males) had blood Pb concentrations slightly above normal for males (0.22 μmol L^{-1}), according to reference values obtained by Apostoli et al[26]; both were already above this value when the experiment began. None of the female subjects exceeded the normal female value of 0.15 μmol L^{-1}. Serum Al was also apparently unaffected and remained well within reference intervals.[27] These intervals have, however, been criticised by Poulson et al,[28] but new levels have not yet been set. No clear pattern was discernible for blood Hg, nor Cl, and both remained within normal limits in all the subjects.[29,30]

Thus, with the exception of Al and Rb, there was little intra-element or intra-subject consistency observed in the results. This makes it difficult to resolve the relationship between exposure and body fluid response at this stage.[4]

Conclusions

This research found enhanced levels of urinary Al and Rb following volcanic gas exposure, which were statistically significant. Other elements analysed here (urinary Hg, Cl, F, blood Pb, and serum Al) did not show such patterns. The result raises questions of why such differences exist, and how future work could test these findings further.

These questions should be resolved in the future, provided improvements in our experimentation are made; notably in:

- Better control of possible confounding factors,
- Improved monitoring of personal exposure in real time with active analysers, and
- An extended sampling strategy with more subjects, elements and several bracketed sample periods, so the kinetics of excretion could be observed.

It is also necessary to have a better understanding of the preferential uptake, retention and excretion of various elements in this multiple element exposure. The molecular form of each element in the volcanic plume is also very important in determining rates of uptake, retention, and excretion—and this can only be resolved with improved techniques in geochemical analysis of the volcanic gas.

Despite these limitations in the present study, our results suggest that when people are acutely exposed to volcanic gases, respiratory absorption and urinary excretion of Al and Rb occurs (perhaps preferentially to other elements).

Given the patterns in urine trace elements we observed following acute exposure, it seems reasonable to extrapolate that chronic exposure might produce more consistent indications of a relationship between exposure and urine and blood chemistry. In that case, quantitative human health risk assessments could follow, but more research would be required before urine and blood chemistry could be used as biomarkers of a potential human disease burden.

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Author information: Michael Durand, Research Fellow, Natural Hazards Research Centre, Department of Geological Sciences, University of Canterbury, Christchurch; Chris Florkowski, Chemical Pathologist; Peter George, Medical Director; Trevor Walmsley, Section Head, Specialist Biochemistry, Canterbury Health Laboratories, Christchurch; Philip Weinstein, Director, Ecology and Health Research Centre, School of Population Health, University of Western Australia, Perth, Australia

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Correspondence: Dr Michael Durand, Department of Geological Sciences, University of Canterbury, Private Bag 4800, Christchurch 8020, New Zealand. Fax: (03) 364 2769; email: michaeldurand2004@yahoo.co.uk

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