

Prevalence and risk factors for pre-hypertension among adolescents exposed to  
volcanic air pollution on Hawai'i Island

A THESIS SUBMITTED TO THE GRADUATE DIVISION OF THE  
UNIVERSITY OF HAWAI'I AT MĀNOA IN PARTIAL FULFILLMENT  
OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

IN

BIOMEDICAL SCIENCE

DECEMBER 2011

By

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## **Acknowledgments**

Supported by National Institutes of Health R01ES11346, R25 RR01932, CDC R01EH000326, and the American Lung Association of Hawaii and Leahi Fund. . I gratefully acknowledge the technical and community support of the Hawai'i Island Children's Lung Assessment Scientific Study (HICLASS) Research Team. Thanks also to Rei Miike, MPH for assistance with statistical analysis.

## **Abstract**

We tested the hypothesis that exposure to volcanic air pollution (vog) is associated with elevated blood pressure (BP) in a cohort of Hawaii Island school children. Between September, 2009 and May, 2010, we measured BP in 807 participants enrolled in the Hawaii Island Children's Lung Assessment Scientific Study (HICLASS). The initial cohort of 1,986 children had been recruited in 2002-2003 from 29 Hawaii Island schools in areas with different vog exposure. Data were analyzed by uni- and multivariate logistic regression models. The mean age was 17; 51% were boys; 62% were of more than 1 race; mean BMI was 24.3 (125 had BMI>30); 32% reported asthma; 56% reported exposure to environmental tobacco smoke (ETS). Vog exposure (n) was categorized as low (168), intermittent (263), SO<sub>2</sub> (48), or acid (328). The mean BP was 119/70 and the prevalence of BP>120/80 was 8.9%. Univariate analysis showed an association with male gender (OR 3.5, p<0.005) and obesity (OR 4.7, p<0.05), but not with vog or ETS. Multivariate analyses run separately for boys and girls (adjusting for race, BMI, asthma, tobacco smoke exposure) showed no association between BP>120/80 and vog exposure, but a strong association with obesity in both boys and girls. This study was limited by a single measurement of BP in a cross-sectional design. Longitudinal follow up, addressing obesity and other risk factors will be important to understand the relative contribution of vog and other factors in developing hypertension.

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## **Background**

Kilauea volcano on Hawaii Island has emitted more than 350 tons of sulfur dioxide (SO<sub>2</sub>) each day for at least 30 years, recent average output is ~1600 metric tons per day. The SO<sub>2</sub> interacts with water vapor to form highly acidic fine sulfate particles. The volcano's ongoing eruption and the island's unique geography provide a unique opportunity to study chronic adverse health effects of sulfurous air pollution.

Studies have shown an association between fuel-derived air pollution (smog) and cardiovascular morbidity, including hypertension, decreased heart rate variability, and death. One cross-sectional study in Hawai'i reported increased prevalence of hypertension among adults in a community of high vog exposure compared to a relatively vog-free community <sup>1</sup>, but the populations differed in other risk factors. We thus added measures of BP to our study of Hawai'i Island schoolchildren, to determine whether vog and other personal and environmental risk factors are associated with pre-hypertension in children.

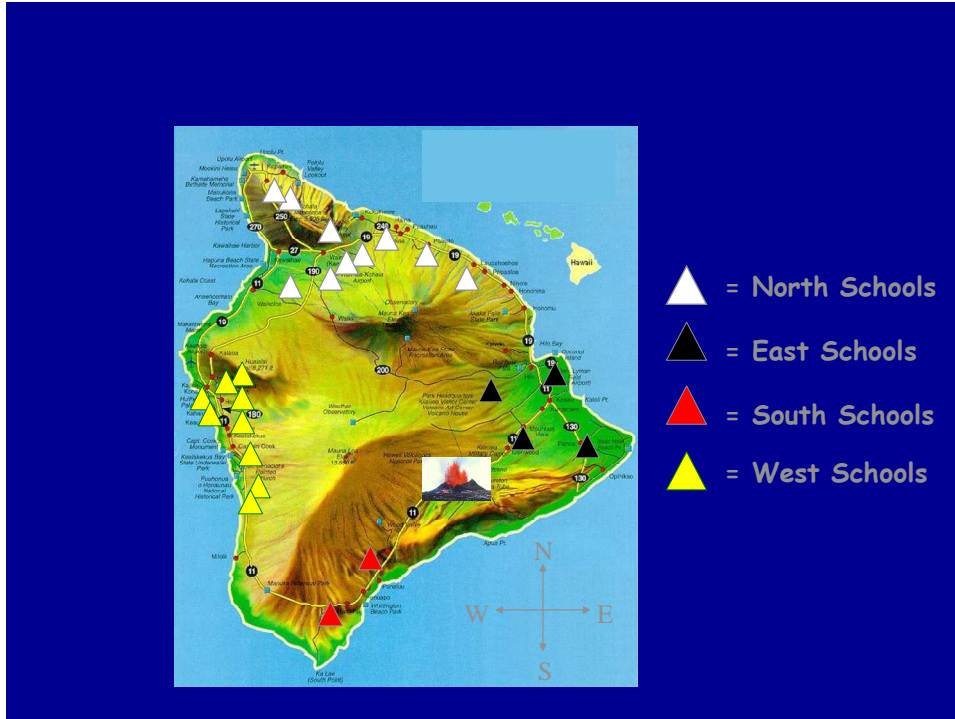
## **Materials and Methods**

### Participants

The Hawaii Island Children's Lung Assessment Scientific Study (HICLASS) cohort was conducted according to a protocol approved by the University of Hawaii Committee on Human Subjects. Participants were recruited in 2002-2003 from 29 schools in four different vog exposure areas on the island of Hawaii. Participants' families responded to a questionnaire of personal and family medical history and environment. In 2009 measurement of systolic and diastolic blood pressure (SBP & DBP) were collected for the first time. The original cohort was comprised of 1986 students; for this paper we analyzed 807 for whom a complete (SBP & DBP) blood pressure reading was available. A participant's vog exposure was categorized as low, intermittent, SO<sub>2</sub> or acid based upon the geographic location of his/her school, Figure 1. North schools are low vog exposure, east are intermittent, south are SO<sub>2</sub> while west are acid.



Figure 1. Geographic Location of Schools



## Measurements

**Blood Pressure and BMI:** Blood pressure was measured in the right arm with an automatic sphygmomanometer and recorded by trained study personnel. In cases where the automatic machine did not read, a manual BP was recorded. If the systolic was  $>135$  OR the diastolic  $>90$ , researchers repeated the measurement in both arms 15-30 min later. BMI was calculated from height and weight measured by the study personnel on the same day as the BP reading.

**Other measures:** Age, sex and race were self-reported. Asthma, smoking, and environmental tobacco smoke exposure were determined by answers to the following questions: Has a doctor ever said you have asthma? How many cigarettes have you smoked in your life? How many cigarettes have you smoked in the last 30 days? Does anyone around you smoke?

## Statistical Analyses

The association of vog exposure and other known risk factors for pre-hypertension, defined as SBP >120 AND DBP >80 were analyzed. After univariate analyses models were developed models for pre-HTN in boys and girls separately and adjusted for race, BMI, asthma, and second hand smoke exposure. All data were analyzed with EpiInfo (CDC Atlanta, GA) and SAS (SAS Institute Cary, NC)

## Results

Among the 807 students for whom BP measurement was available, mean age was 17; 51% were boys; 62% were of more than one race mean BMI was 24.3; 125 (15%) were obese; 32% reported asthma, 56% reported exposure to ETS Table 1. Table 2 shows the distribution of children by Vog Exposure. The overall prevalence of pre-HTN is 8.9%, with a significant gender gap, 4.3% among girls vs. 13.5% among boys. In univariate analysis pre-HTN was associated with male gender (OR 3.5,  $p < 0.005$ ) and obesity (OR 4.7,  $p < 0.05$ ), but not with vog or tobacco smoke exposure. Multivariate analyses, with models run separately for boys and girls (adjusting for race, BMI, asthma, tobacco smoke exposure) showed no association between pre-HTN and vog exposure, but did show a strong association with obesity in both boys  $P < 0.05$  and girls  $p < 0.05$ . In this cross-sectional study, pre-hypertension was not associated with vog exposure nor ETS, but was associated with male gender and, in both boys and girls, with obesity

Table 1 Distribution of demographic and vog exposure variables, n (%)

	Total n=807	Boys n=408	Girls n=399
BMI			
<below avg	21(2.6)	8 (2)	13 (3.2)
average	517(64)	253 (62)	264 (66)
overweight	144(17.9)	77(18.8)	67 (17)
obese	125 (15.5)	70 (17.2)	55(13.8)
Race			
White	110 (13.7)	59 (14.5)	51 (12.8)
Asian	149 (18.5)	80 (19.6)	69 (17.3)
Mixed	498 (61.3)	242 (59.3)	256 (64.3)
Pacific Islander	38 (4.7)	19 (4.7)	19 (4.8)
Other	11 (1.4)	8 (2)	3 (8)
Asthma			
No	550(68)	280 (68.5)	270 (67.4)
Yes	257 (32)	128 (31.5)	129 (32.6)
ETS			
No	358 (44.2)	179 (43.7)	179(44.7)
Yes	449 (55.8)	229 (56.3)	220 (55.3)
Vog Exposure			
Low	168 (20.8)	95 (23.3)	73 (18.3)
Intermittent	263 (32.6)	127 (31.1)	136 (34.1)
SO2	48 (6.0)	27 (6.6)	21 (5.3)
Acid	328 (40.6)	159 (39.0)	169 (42.3)
Pre-HTN			
No	735 (91.1)	353 (86.5)	382 (95.7)
Yes	72 (8.9)	55(13.5)	17 (4.3)

Table 2. Participants Age, height, weight, BMI, blood pressure (mean  $\pm$  s.d.)

	Total	Male	Female	<i>p</i>
	n=807	n=408	n=399	
Age (yr)	17.1 $\pm$ 0.7	17.1 $\pm$ 0.6	17.0 $\pm$ 0.7	NS
Height (cm)	167 $\pm$ 10	173 $\pm$ 8	160 $\pm$ 7	<0.05
Weight (kg)	68.2 $\pm$ 16.8	73.2 $\pm$ 17.3	62.7 $\pm$ 14.5	<0.05
BMI	24.3 $\pm$ 5	24.4 $\pm$ 5	24.2 $\pm$ 5	NS
Systolic BP	119 $\pm$ 14	125 $\pm$ 13	112 $\pm$ 11	<0.05
Diastolic BP	70 $\pm$ 10	71 $\pm$ 10	69 $\pm$ 9	<0.05

Table 3. Associations between risk factors and pre-hypertension stratified by gender

Independent Factor	BOYS					GIRLS				
	n	% with pre-HTN	Adj OR	OR 95% CI	<i>p</i>	n	% with pre-HTN	Adj OR	OR 95% CI	<i>p</i>
	399					393				
BMI*										
below avg	8	0				13	7.7	1.76	0.19-16.53	NS
avg	253	8.7	1.00			264	3.0	1.00		
overweight	77	13.0	1.34	0.59-3.05	NS	67	4.5	1.31	0.19-16.52	NS
obese	70	32.9	5.87	2.91-11.8	<0.05	55	9.1	3.37	1.01-11.29	<.05
Race										
White	59	11.9	1.00			51	3.9	1.00		
Asian	80	17.5	1.16	0.40-3.32	NS	69	7.3	0.89	0.14-5.67	NS
Mixed	242	13.2	0.65	0.25-1.68	NS	256	3.5	0.42	0.07-2.38	NS
P.I.	19	5.3	0.17	0.02-1.68	NS	19	5.3	0.91	0.07-11.61	NS
Other	8	12.5	0.88	0.09-8.84	NS	3	0			
Asthma										
No	280	12.5	1.00			267	3.0	1.00		
Yes	128	15.6	1.13	0.60-2.14	NS	129	7.0	2.63	.90-7.71	<.10
ETS										
No	178	10.7	1.00			178	4.5	1.00		
Yes	229	15.7	1.55	0.83-3.01	NS	220	4.1	0.84	0.30-2.37	NS
Vog										
Low	95	16.8	1.00		NS	73	4.1	1.00		NS
Intermittent	127	15.8	.87	.41-1.85	NS	136	6.6	1.42*	.36-5.67	NS
SO2	21	11.1	.48	.12-1.96	NS	21	0.0	*		NS
Acid	169	10.1	.55	.25-1.93	NS	169	3.0	0.71	.15-3.17	NS

\*bmi groups based on CDC cutoffs for Caucasian children

\*\*includes SO2 group

## Discussion

Unlike adults for whom HTN is a clinical diagnosis, in children HTN is based upon the normative distribution of blood pressure in healthy children. In the United States normative BP percentiles are based upon measurements from the National Health and Nutrition Examination Survey (NHANES)<sup>2</sup>. Since body size is an important determinant of BP in children, HTN is best defined when values are adjusted for height, weight, age and gender<sup>3</sup>. The current guidelines for the definition of HTN and pre-HTN were published in 2004<sup>3</sup>.

For children  $\leq 18$  years old, normal BP is defined as both SBP and DBP  $<90^{\text{th}}$  percentile. According to the most recent guidelines of the National High Blood Pressure Education Program Working Group (NHBPEP), pre-HTN is defined as BP either SBP or DBP  $>90^{\text{th}}$  percentile but less than the  $95^{\text{th}}$  percentile or  $>120/80$  regardless of percentile. HTN is defined as either SBP or DBP  $\geq 95^{\text{th}}$  percentile on at least three separate measurements. We defined pre-HTN as a BP  $>120/80$ .

The overall prevalence of pre-HTN (8.9%) is in line with studies using data from NHANES which have been reported prevalence of pre-HTN from 7.7-10%<sup>4,5</sup>. According to the same population based surveys the prevalence of HTN has been increasing since the 1980's<sup>4,5</sup>. This increase is thought to be due at least in part to the increasing prevalence of obesity in school age children.<sup>4-6</sup>. Also similar to previous published reports we found pre-HTN to be associated with the male gender and increased BMI<sup>6</sup>. Not all studies of children have had dissimilar prevalence for pre-HTN in boys and girls, in our univariate analyses there was a strong association with male gender OR=3.1  $P<0.005$ . For this reason we performed the multivariate analysis models separately for

boys and girls. Because of differences in size and BMI, results for boys and girls are presented separately. An analysis of pre-school children in Germany male gender was not associated with elevated BP<sup>7</sup>.

Regardless of other findings, nearly all studies report that elevated BMI is strongly associated with pre-HTN or HTN<sup>3,4,8-10</sup>, even when other factors contribute to the findings<sup>11</sup>.

In adults there is a strong association between HTN and African American race which has not been found in children. Other racial disparities, specifically an increased risk of HTN and pre-HTN for Hispanic boys have been reported<sup>8</sup>. Our study has a unique ethnic distribution which differs both from other studies done in the U.S. as well as those reported from Europe and China. More than half of our participants are of more one ethnicity, but few are African American or Hispanic. We did not detect any statistically significant racial differences in BP; though because of the ethnic make up of our population it may not generalize to the U.S. population as a whole. It also makes comparison with other findings of racial differences difficult.

We examined our data and found no association between asthma and pre-HTN. While this association may exist in adults, we did not see it, nor did studies of adolescents in Brazil<sup>12</sup> and Detroit<sup>13</sup>.

We did not see an increased risk for pre-HTN with ETS exposure in the adolescent children in our study, although it has been seen in younger children<sup>7</sup>. Our questionnaire only asked about current ETS exposure, so it is unknown if previous ETS exposure is a factor in this study.

Blood pressure measurements were added to the HICLASS data collection instrument to see what effect if any vog exposure has on blood pressure, we did not find any association with Vog and pre-HTN in this one time point. Further longitudinal data are needed to see if the association seen in adults is replicated in children<sup>1</sup>.

Since evidence shows that adult HTN begins during childhood<sup>9, 10</sup> resulting in the early development of cardiovascular disease (CVD) in adults it is important to identify modifiable risk factors in children, specifically ETS and BMI Identifying children with pre-HTN and intervening to prevent the development of HTN could have a significant future impact on the incidence of CVD in adults.

This study was limited by availability of only a single measurement of BP and relatively small sample size. . Longitudinal follow up, and addressing obesity and other risk factors, including parental factors will be important to understand the relative contribution of vog and other factors in developing hypertension. Future analyses with additional BP readings possibly with the addition of waist circumference will be needed to fully assess weather the association between elevated BP in adults is also manifest in children.



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