Combustion Particles and Global Health: Cooking and Climate

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Presentations at University of Southern California and Stanford University – Feb 2011



Households Using Solid Cooking Fuels



% of HH Exposed to HAP



For 2005, CRA-10 preliminary

Woodsmoke is natural – how can it hurt you?

Or, since wood is mainly just carbon, hydrogen, and oxygen, doesn't it just change to CO_2 and H_2O when it is combined with oxygen (burned)?



Reason: the combustion efficiency is far less than 100%

Energy flows in a well-operating traditional wood-fired Indian cooking stove

A Toxic Waste Factory!!

Typical biomass cookstoves convert 6-20% of the fuel carbon to toxic substances



PIC = products of incomplete combustion = CO, HC, C, etc.

Source: Smith, et al., 2000

Toxic Pollutants in Biomass Fuel Smoke from Simple (poor) Combustion

Organics known to be mutagens, immune system suppressants, severe irritants, inflammation agents, central nervous system depressants, cilia toxins, endocrine disrupters, or neurotoxins.

Several chemicals firmly established as human carcinogens.

Other toxic inorganic chemicals.

- 25+ alcohols and acids such as methanol
- 33+ phenols such as *catechol* & *cresol*
- Many quinones such as *hydroquinone*
- Semi-quinone-type and other radicals
- Chlorinated organics such as *methylene chloride* and *dioxin*

Health-Damaging Air Pollutants From Typical Woodfired Cookstove in India.





"Thank God! A panel of experts!"

Courtesy of Ross Anderson

ALRI/ Pneumonia

Diseases for which we had epidemiological studies available around 2001 Chronic obstructive lung disease

 Lung cancer from coal only

Three outcomes qualified with sufficient evidence to be included in the WHO CRA of 2004

Global Burden of Disease from Top 10 Risk Factors plus selected other risk factors



Percent of All DALYs in 2000

ALRI/ Pneumonia (meningitis)

Low birth weight

Stillbirth Cognitive Impairment

Birth defects? Asthma?

Burns, health and safety impacts of fuel gathering?

Diseases for which we have

epidemiological studies - 2010

Chronic obstructive lung disease

Interstitial lung disease Cancer (lung, NP, cervical, aero-digestive)

Blindness (cataracts, opacity)

Tuberculosis

Heart disease? Blood pressure ST-segment

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Estimated PM2.5 indoors for <u>all</u> households

Estimated PM2.5 for <u>only</u> solid-fuel-using households



				Study	Odds Ratio (random)	Weight	Odds Ratio (random)
Ctudy	NI*			or sub-category	95% CI	70	95% CI
Sludy	IN		90% 01	01 Intervention Studies			
design				Smith(2007)a		5.53	1.18 [0.88, 1.58]
uesign				Smith(2007)b		5.73	1.35 [1.05, 1.73]
				Subtotal (95% CI)	••••••••••••••••••••••••••••••••••••••	11.26	1.28 [1.06, 1.54]
Intervention	2	1.28	1.06. 1.54	Test for heterogeneity: Chi ² = 0.48, df	= 1 (P = 0.49), ² = 0%		
				Test for overall effect: Z = 2.54 (P = 0			
				02 Cohort Studies			
	-	0.40		Armstrong(1991)a		2.80	0.50 [0.20, 1.22]
Conort	1	2.12	1.06, 4.25	Armstrong(1991)b		3.65	1.90 [0.96, 3.75]
				Cambell(1989)		3.25	2.80 [1.29, 6.08]
				Ezzati(2001)		3.86	2.33 [1.23, 4.40]
				Jin(1993)		5.69	0.80 [0.62, 1.03]
				Pandey(1989)a		4.34	2.45 [1.43, 4.19]
				Pandey(1989)b	100 million 100	1.52	40.65 [9.79, 168.75]
				Subtotel (95% C) Test for beteregenetis Chill - 54.07	H - 6 (D - 0 00001) 12 - 99 09	25.11	2.12 [1.05, 4.25]
				Test for overall effect: 7 = 2.11 (P = 0	1 = 0 (P < 0.00001), P = 00.9%		
				Test for overall effect. 2 = 2.11 (F = 0			
^				03 Case-Control Studies			
Case-	15	1.97	1.47, 2.64	Azizi(1995)		3.97	1.20 [0.65, 2.21]
			· ·	Broor(2001)		4.49	2.51 [1.51, 4.17]
control				Collings(1990)		4.85	2.16 [1.40, 3.33]
				De Francisco(1993)		→ 2.15	5.23 [1.72, 15.91]
				Fonsecca(1996)		4.68	1.14 [0.71, 1.82]
				Johnson(1992)a		3.15	0.80 [0.36, 1.78]
				Kossove(1982)		→ 1.96	4.77 [1.44, 15.74]
				Mahalanahas(2002)		2.45	3.07 [1.42, 10.57]
				Morris(1990)		- 2.41	4.85 [1.75 13.40]
				O'Dempsey(1996)		2.59	2.55 [0.98, 6.64]
				Robin(1996)a		2.95	1.40 [0.60, 3.28]
				Victora(1994)a		4.08	1.10 [0.61, 1.98]
				Wayse(2004)		2.90	1.39 [0.58, 3.30]
				Wesley(1996)		1.87	1.35 [0.39, 4.63]
				Subtotal (95% CI)		48.15	1.97 [1.47, 2.64]
				Test for heterogeneity: Chr = 32.72, o	11 = 14 (P = 0.003), P = 57.2%		
0	0	4 40		Test for overall effect. Z = 4.55 (P < 0			
Cross-	3	1.49	1.21, 1.85	04 Cross-sectional Studies			
agetional				Mishra(2003)		3.83	2.20 [1.16, 4.18]
sectional				Mishra(2005)		5.87	1.58 [1.28, 1.95]
				Wichmann(2006)		5.79	1.29 [1.02, 1.63]
				Subtotal (95% CI)	•	15.48	1.49 [1.21, 1.85]
				Test for heterogeneity: Chi ² = 3.19, df	= 2 (P = 0.20), ² = 37.3%		
				Test for overall effect: Z = 3.74 (P = 0	1.0002)		
	26	1 78	45 2 18	Tetel (OSS) ON		100 00	1 80 11 15 1 10 10
				Test for beterogeneity: Chil - 101 74	4t = 26 (B < 0 (0001) 12 = 74 49	100.00	1.78 [1.45, 2.18]
				Test for overall effect: $7 = 5.61 (P \times 0)$	000001) = 74.4%		
Dhorani ot a	Rul		(2008)				
Dherani et a	Dui			0.1 0	0.2 0.5 1 2 5	10	
					Increased risk Decreased risk		

Preliminary CRA Effect Estimates

Health Outcome	Sex	Age	Level of Outcome	Risk Estimate
ALRI	M&F	< 60 mo	la	1.78 (1.45 to 2.18)
ALRI:	M&F	< 60 mo	lb	2.3 (95% Cl ?)
exposure/response				
COPD	F	>15 yr	la	2.7 (1.95 to 3.75)
COPD	М	>15 yo	la	1.9 (1.15 to 3.13)
Lung Cancer (coal)	F	> 15 yr	la	1.98 (1.16 to 3.36)
Lung Cancer (coal)	М	> 15 yr	la*	1.38
Cataract	F	> 30 yr	la	2.45 (1.61 to 3.73)
Cataract	М	> 30 yr	la	?
LBW (OR)	M&F	Perinatal	la	1.52 (1.25 to 1.80)
LBW (mean weight)	M&F	Perinatal	la	93.1g (64.6, 121.6)
Lung Cancer (biomass)	F	> 15 yr	la	1.81 (1.07 to 3.06)
Lung Cancer (biomass)	М	> 15 yr	la	1.26 (1.04 to 1.52)
CVD	F	> 30 yr	lb	1.3 to 1.4 (95% CI)
CVD	Μ	> 30 yr	lb*	1.16

Story of Two Conferences

- Air pollution conference
 - High exposures to large vulnerable population
 - No more health effects work needed
- International health conference
 - Still doubt about causality
 - Need to know exact benefit to be expected
- Where are your randomized controlled trials?

History of an RCT

- ~1980: Early studies of health effects in Nepal and elsewhere
- 1981: First measurements of pollution levels in India
- 1984: International meeting to decide on needed research
 <u>Chose randomized control trial (RCT) of ALRI</u>
- 1986-89: Unfunded proposals to do RCT in Nepal
- 1990: WHO establishes committee to find best sites
- 1990-1992: Criteria established and site visits made
- 1992: Highland Guatemala chosen
- 1991-1999: Pilot studies to establish data needed for proposal
- 1996-1999: Unfunded proposals
- 2001: NIEHS funding secured
- 2002-2005: Fieldwork completed
- 2011: Main results published
- 25+ years from deciding to conduct RCT to results!



RESPIRE: (Randomized Exposure Study of Pollution Indoors and Respiratory Effects)



Traditional 3-stone open fire

Chimney woodstove

Overview of RESPIRE study design



Randomisation: balance of groups at baseline

Variable	Control	Intervention
Socio-demographic factors		
Mother's Age (years)	27.0	26.4
Pregnant at recruitment (%)	48.3	51.3
Own home (%)	92.8	94.1
Migrates part of year (%)	17.7	17.1
House structure		
Separate enclosed cooking area (%)	76.2	74.3
Completely open eaves (%)	42.7	40.6
Walls – adobe (mud) (%)	88.7	90.7
Roof – metal (%)	77.4	74.3
Floor – earth (%)	92.5	88.8
Leaks in roof (water) (%)	24.5	33.3
Electricity (%)	70.8	69.3
Other sources of smoke		
Other fire near house (%)	14.6	14.4
Smoking (tobacco) indoors (%)	26.8	20.4
Use traditional sauna bath (%)	84.5	87.8
Geographic		
Mean altitude (metres)	2613	2601

Overview of child health outcomes assessment



* Respiratory syncitial virus

Overview of weekly visits

		Plancha	Control	
Number of childre	en	265	253	
Weekly visits	Total possible in follow up period	16,446	15,664	
	Completed	14,756	14,369	
% of possible wee	ekly visits completed	89.7%	91.7%*	
Mean (SD, range)	visits per child	55.7 (17.8; 1 to 80)	56.8 (17.3; 2 to 81)	
Number (%) child	lren - no missed visit	17 (6.4%)	19 (7.5%)	
Withdrawals		19 (7.2%)	14 (5.5%)	

* P < 0.001

Unpublished results from RESPIRE have been removed

Watch the website below where they will be posted as soon as they are published.

http://ehs.sph.berkeley.edu/krsmith/





Chimney stove did not protect all children

Guatemala RCT: Kitchen Concentrations



2010

Infant Exposures



Effect of Plancha on PM2.5 Log Scale 1000 Open fire ~90% Reduction, sig. 100 Plancha 48-h ug/m3 10 1 Kitchen

Reasons that child personal exposures did not lower as much as kitchen levels:

- --Time-activity: the kids do not spend their entire day in the kitchen
- --Household (or "neighborhood") pollution: a chimney does not reduce smoke, but just shifts it outside into the household environment, where the difference between intervention and control households was less
 --Other burning around house not different

Child Cognitive Function: First Pilot Study

[results removed until publication – watch website]

Increasing Number of Studies on Disease Signs: Objective Measures

- Lung function, cross-sectional
- Blood pressure, EHP, 2007
- ST-segment, EHP, forthcoming
- Opacity, submitted

Dioxin Daily Dose Estimates

[Unpublished results of measurements in households in Guatemala removed.

Watch website for publication]



Poisonous Coal Use and Household Exposures

Arsenic and Fluorine Exposures in China from Household Coal Use

- More than 95% of the fluorine dose came from food consumption, with the rest mainly from direct inhalation of airborne fluoride -- Ando et al., *EHP* (1998)
- The estimated sources of total arsenic exposure are from arsenic-contaminated food (50-80%), air (10-20%), water (1-5%), and direct skin contact (<1%)
 -- Liu et al., EHP (2002)
- Dioxin might be expected to have a similar pattern.

Kitchen PM_{2.5} in household using open cookfire <u>How many hours should we measure to obtain</u> <u>good estimate of mean?</u>



Inter-instrument Comparison: 30 UCB-PATS UCB Particle and Temperature Monitoring System (custom PM monitor using smoke alarm technology)

> Lopez Kitchen La Cienaga Plancha with chimney

5 PM Sept 24 to 10 AM Sept 25, 2004.

Long-term Household Measurements

hhid=hh04041020



How Close to the True Mean With One Measurement?



Attenuation Bias in Measurement Error



Highland Guatemala Friday, Feb 20, 2004 ~6:15 AM





Sources of Primary PM_{2.5}: India and China



IIASA, 2010

NASA INTEX_B Database Percent PM_{2.5} emissions from households



Global warming in 2005 due to all human emissions since 1750



 CO_2 is important for climate, Several of the non- CO_2 , but so are many other greenhouse gases create pollutants, including the ones a good proportion of both circled that, unlike CO_2 , also their climate forcing and have significant health as health damage through well as climate impacts the secondary pollutant,

twpospheriplozenand organic carbon particles however, have cooling impacts on climate but are still health damaging

IPCC, 2007

Household Fuels and Climate

- Climate impacts come from non-renewable biomass and coal, i.e., from net CO₂ emissions
- Poor combustion also leads to other emissions such as the relatively well-understood GHGs – methane and nitrous oxide – which are "Kyoto" GHGs
- In addition, a wide range of less well-understood shortlived GH-related emissions are emitted including
 - CO and black carbon warming agents
 - Ozone precursors warming But also cooling agents such as sulfates and organic carbon particles
- There are also indirect climate impacts of these pollutants including
 - Reducing carbon capture of forests by ozone damage
 - Darkening of snow/ice by black carbon

Greenhouse warming commitment per meal for typical biomass-fired cookstove in China



Zhang, et al., 2000



India in 2005

Venkataraman et al. 2010

Controllable Global Warming from Black Carbon Emissions

Net of OC, Forcings from IPCC, 2007: 0.25 W/m² Inventory from T Bond Database, V 7.1.1 Feb 2009



Climate Warming in 2020 Under Present Trends



Perfect Storm for Health Impacts

- Highly polluting activity
- Half of world households
- Several times a day
- Just when people are present
- Most vulnerable (women and young children) most likely to be there

In other words, the Intake Fraction is extremely large

IF is the fraction of material emitted that is actually breathed in by someone



IF = 1.0

Intake Fractions : these are rough calculations for typical examples of sources in each class



Smith, 1993

Grams Inhaled per Ton Emitted



Heart Disease and Combustion Particle Doses



Adjusted Relative Risk

Argument from consistency across combustion particle exposure settings

- Assumes fine combustion particles are best measure of risk in each setting and have similar effects per unit mass across the four source types
 - Three are mainly biomass
 - OAP contains significant biomass particles
 - Probably difference by outcome, however e.g., LBW and lung cancer may be related to other components as well
- Remarkable consistency across 3 orders of magnitude of dose measured in mg/day of PM_{2.5}
- Where HAP has no direct epi data, seems reasonable to interpolate for outcomes where there are well established effects at both lower and higher doses.



Dose PM2.5 - mg/day



Dose PM2.5 - mg/day

Relative Risk

Wood is the fuel that

- Heats you twice as Thoreau said?
 - Once when you chop it and
 - Once when you burn it
- Or four times?
 - The fever from respiratory infection and
 - Global warming
- Better combustion will get rid of the second pair

Many thanks

Publications and presentations available at my website: <u>http://ehs.sph.berkeley.edu/krsmith/</u> Or just Google "Kirk R. Smith"