Global Burden due to Household Air Pollution from Cooking Fuel
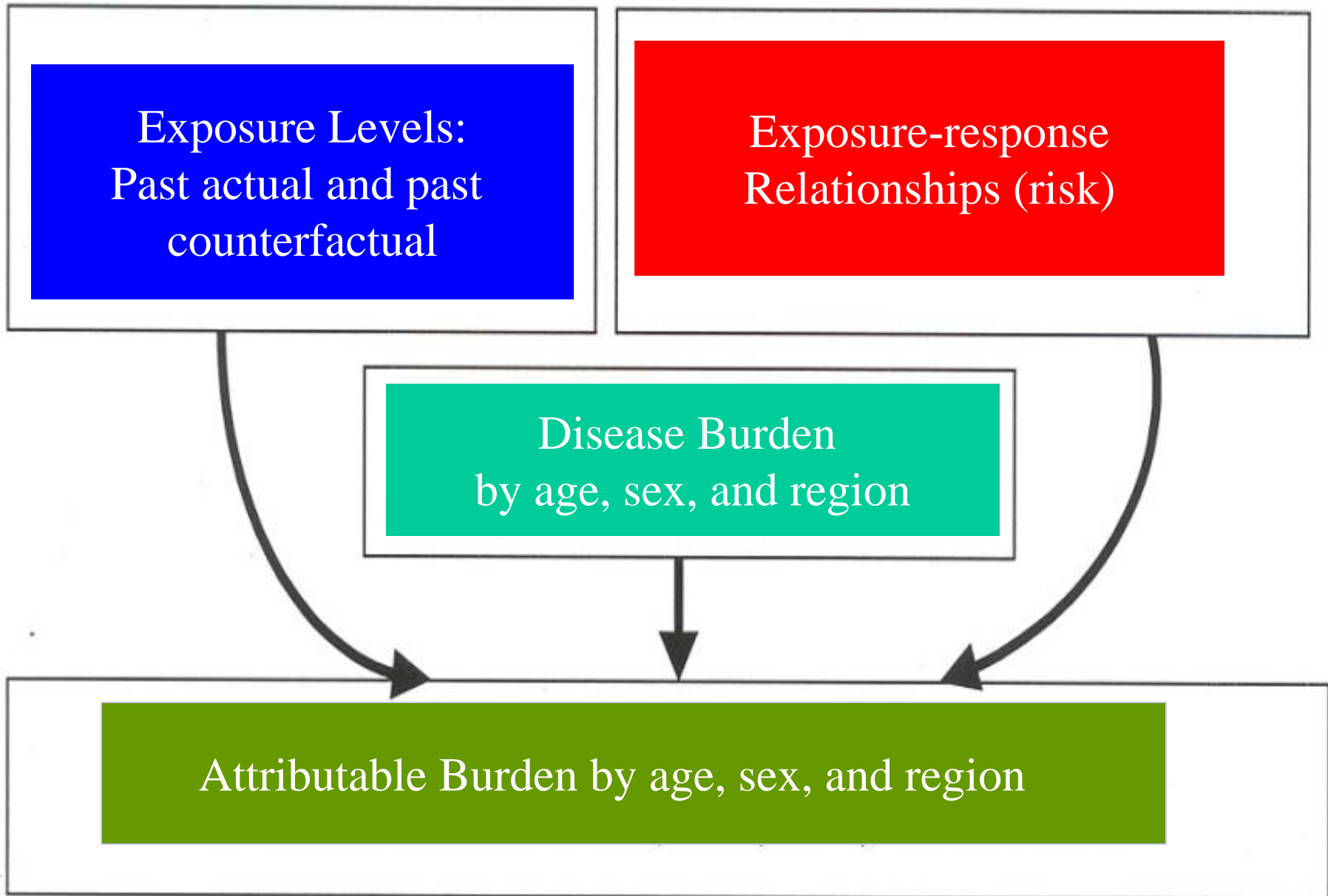
Kirk R. Smith, UC Berkeley
For the HAP Expert Group

International Society for Environmental Epidemiology
Columbia, South Carolina
August 29, 2012

Expert Group

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- Peking University: Jinliang Zhang, Xiaoli Duan
- Universidad Peruana de Ciencias Aplicadas: Claudio Lanata
- With much help from Majid Ezzati, Imperial/GBD; and Aaron Cohen, HEI

Comparative Risk Assessment Method

Exposure Levels: Past actual and past counterfactual

Exposure-response Relationships (risk)

Disease Burden by age, sex, and region

Attributable Burden by age, sex, and region
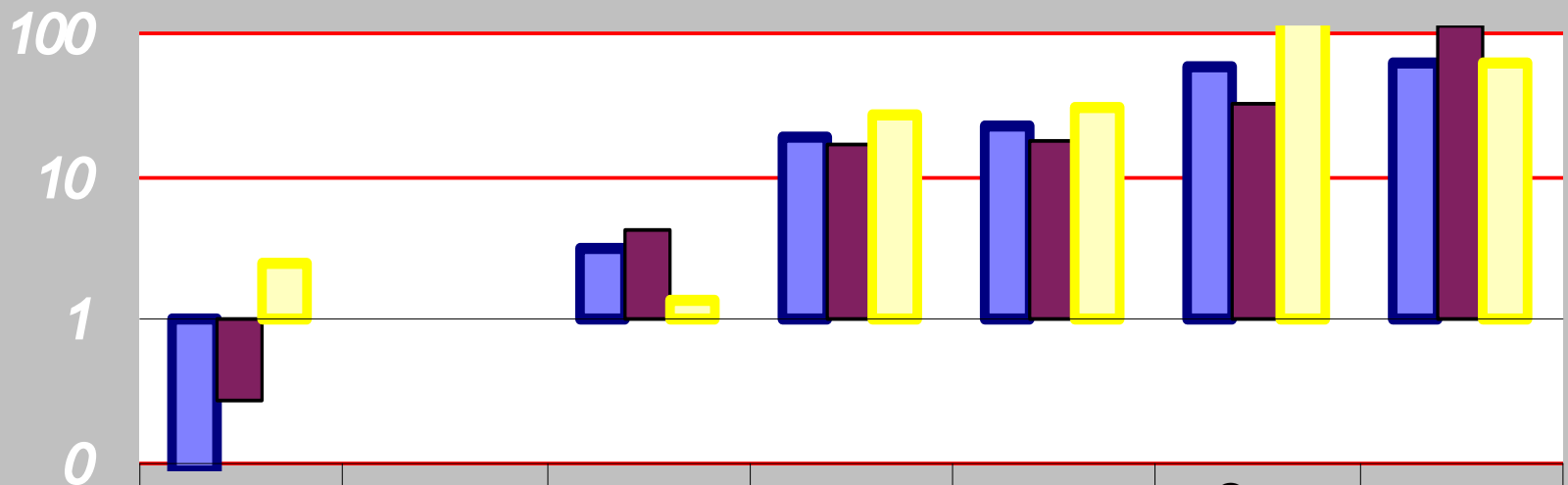
Road Map

- Framing, counterfactuals, arguments for consistency

- Updated exposure assessment

- Updated outcome assessments

- Final burden estimates: in progress

National Household Solid Fuel Use, 2000

<20%	20% - 40%	40% - 60%	60% - 80%	>80%

Household solid fuel use known

No data
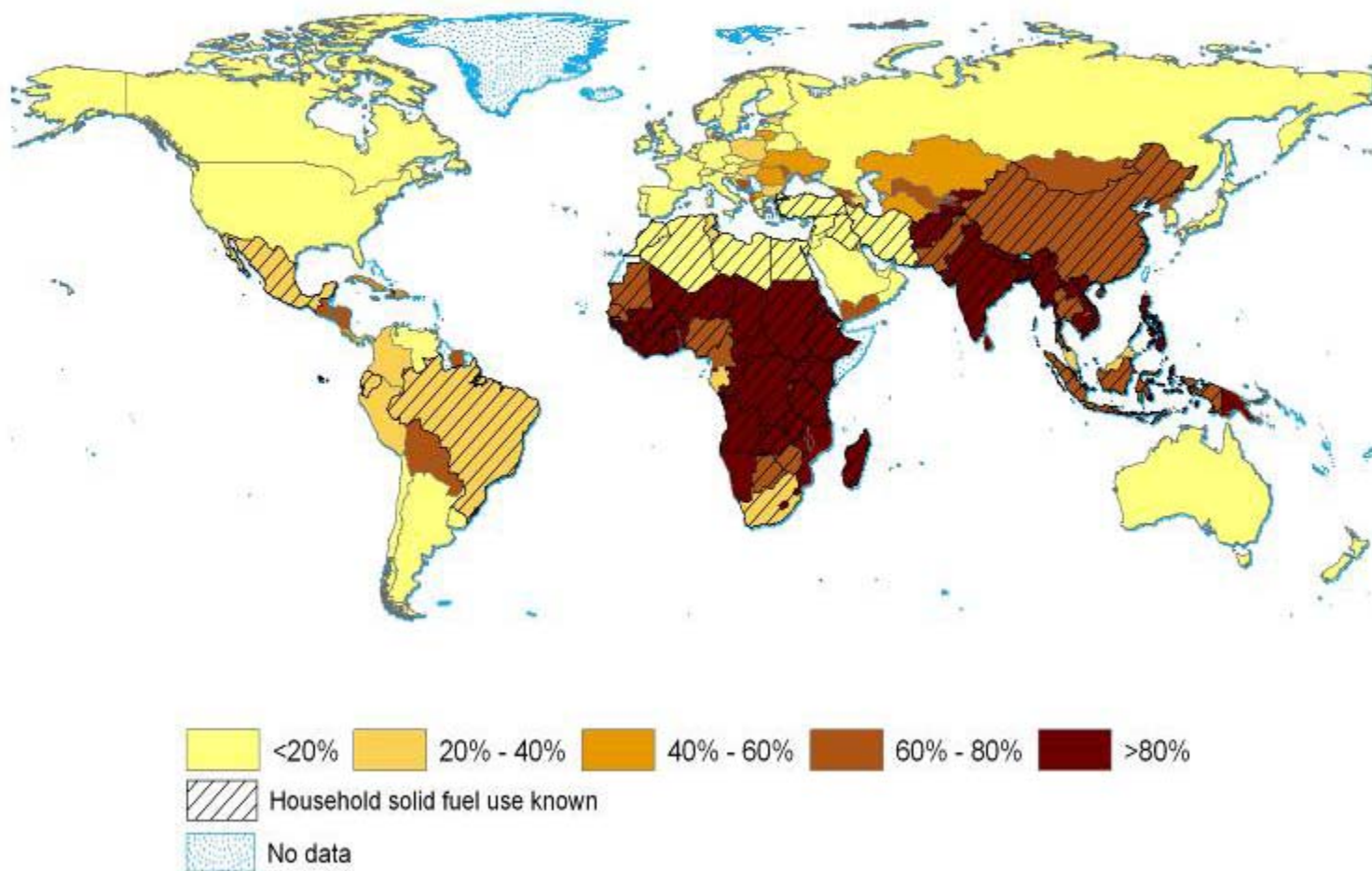
Health-Damaging Pollutants per Unit Energy Delivered
Ratio of Emissions to LPG

	Biogas	LPG	Kerosene	Wood	Roots	Crop Residues	Dung
☐ CO	0.1	1.0	3	19	22	60	64
■ Hydrocarbons	0.3	1.0	4.2	17	18	32	115
☐ PM	2.5	1.0	1.3	26	30	124	63

Legend: ☐ CO ■ Hydrocarbons ☐ PM

Indian measurements, Smith, et al., 2005

CRA-2000

- Indoor air pollution from household solid fuel use
- Counterfactual: non-solid fuel use
- Exposure: Solid fuel use from household surveys, separating biomass and coal, modeled globally
- Outcomes
  - ALRI in children
  - COPD
  - Lung cancer from coal use

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

Risk Factor	Percent of All DALYs in 2000
Underweight	~9.5%
Unsafe sex	~6.3%
Blood pressure	~4.4%
Tobacco	~4.1% → 4.9 million premature deaths/y
Alcohol	~4.0%
Unsafe water/sanitation-E*	~3.7%
Child cluster vaccination	~3.4%
Road traffic accidents	~2.8%
Cholesterol	~2.8%
Indoor smoke-E	~2.7% → 1.6 million deaths/y
Overweight	~2.3%
Low fruit and veg	~1.9%
Occupational (5 kinds)-E	~1.6%
Lead (Pb)-E	~0.9%
Urban outdoor air-E	~0.5% → 800,000 deaths/y
Climate change-E	~0.4%

Percent of All DALYs in 2000

Framing for CRA-2010: HAP

- Household air pollution (HAP) from cooking fuel – incomplete combustion
- Cooking fuels only, although sometimes difficult to separate from space heating
- Uses long-term fine particle ($PM_{2.5}$) exposures as metric, where possible,
- Otherwise solid fuel use
- Includes household contribution to outdoor air pollution

Potential HAP Counterfactuals

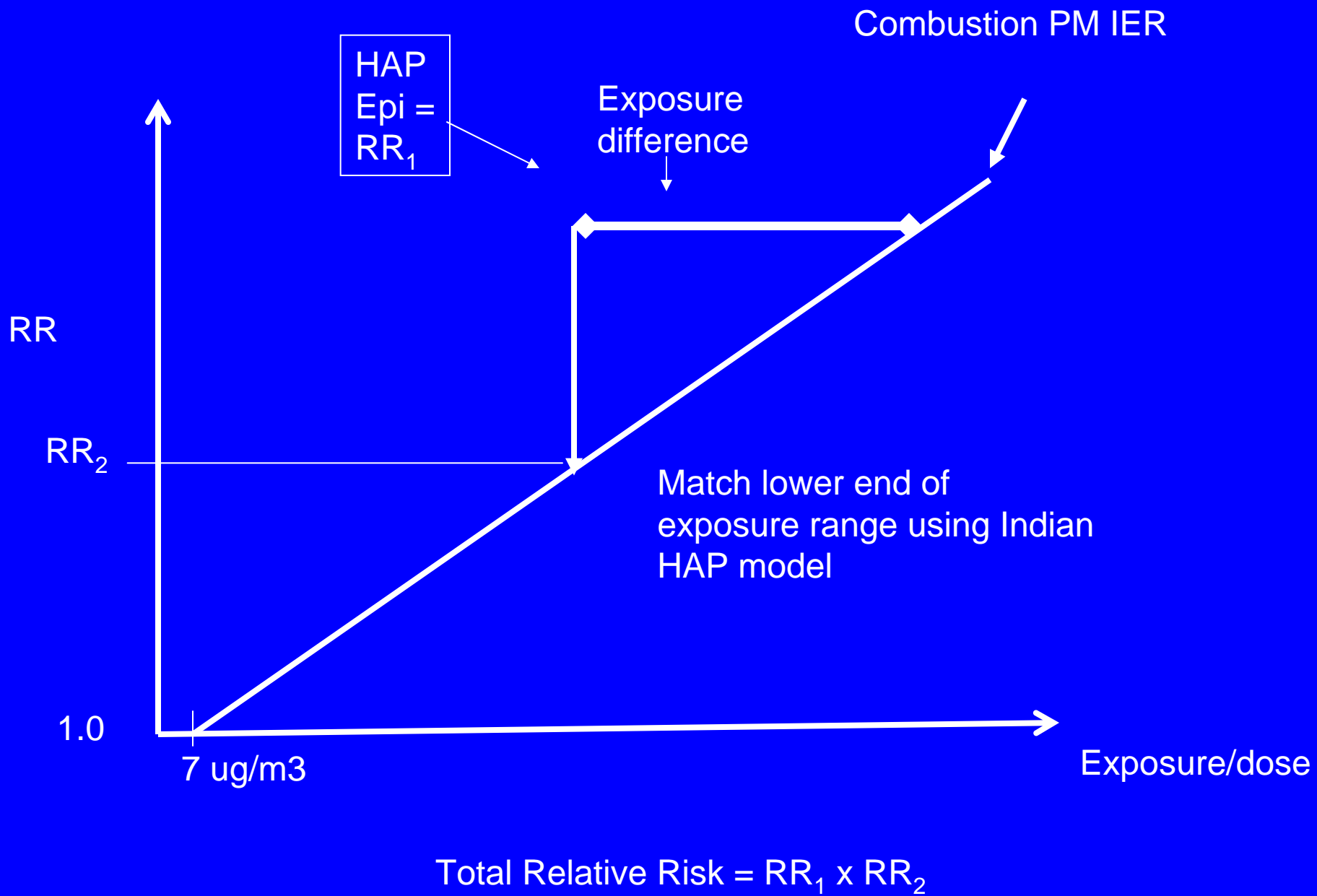| Potential CF	PM2.5 equivalent (ug/3)	How relates to epidemiology	Consistency with proposed outdoor air CF?	Currently achievable?	Useful target for medium term?
1.Dichotomous solid vs. 'low exposure' comparison	Unknown in almost all studies; perhaps 40-100, maybe more	Consistent, but poorly defined	Higher, uncertain, not consistent	Yes, although actual levels uncertain, and very mixed	Too poorly defined
2. WHO Air Quality Guideline	10 (annual)	Probably lower than 'low exposure group' in most or all studies	Higher, not consistent	Not globally, in short term	Yes, especially with AQG 'IT' phased approach concept

HAP Counterfactuals: cont.

Potential CF	PM2.5 equivalent (ug/3)	How relates to HAP epidemiology	Consistency with outdoor air CF?	Currently achievable?	Useful target for medium term?
3. Proposed OAP CF	7 (annual)	Lower than 'low exposure group' in all studies	Same	Not in short to medium term	Probably unrealistic and not useful for policy
4. Gas cooking	5-20 ug/m3, lower with venting	May be similar to some clean fuel' comparisons	Reasonably consistent	~50% of world at this level	Similar to WHO AQG and matches Indian policy
5. Electric cooking	Zero emissions – no combustion	There is developed country epidemiology	Lower, but reasonably consistent	Potentially over time, but raises question of how to treat current gas use	Less so than with gas but still easily understood

Counterfactual Chosen

- 7 ug/m3 annual average PM$_{2.5}$ – same as OAP
- This can be achieved with electric cooking or gas cooking with ventilation
- Clearly feasible – gas and electricity now used by three-fifths of world population

How to estimate risk down to 7 ug/m3 with no direct epi?

- Extrapolating from high to low doses – inverse of usual problem

- Link HAP epi with integrated exposure-response (IER) models

- Use Indian exposure modeling to determine ug/m3 equivalent of epi studies

Combustion PM IER

HAP Epi = RR_1

Exposure difference

RR

RR_2

Match lower end of exposure range using Indian HAP model

1.0

7 ug/m3

Exposure/dose

Total Relative Risk = $RR_1 \times RR_2$

Advances in CRA-2010

- Much more robust global modeling of fuel use
- Proportion of outdoor air pollution from HAP
- Modeling of $PM_{2.5}$ exposures for 25% of world solid fuel households, those in India
- New SR/MAs for the previous 3 outcomes (ALRI, COPD, LC from coal)
- RCT and exposure-response also available for ALRI
- New SR/MAs for 3 additional outcomes (LBW, cataracts, LC from biomass)
- Better discrimination of male/female outcomes/ exposures
- Consistency from IERs derived from outdoor air, passive smoking, HAP, and active smoking
- Interpolation of CVD outcomes from IERs

Exposure to Household Air Pollution from Cooking Fuel

Exposure Overview

1. Estimating household cookfuel use – 1990, 2005, 2010

   - Update of global model to include the much larger set of nationally representative household surveys available, nearly 600 in total – 155 countries

2. Estimating the HAP contribution to ambient $PM_{2.5}$ concentrations – 1990, 2005, 2010

   - A portion of the exposure to outdoor air pollution and burden will thus be attributable also to HAP

3. Estimating household $PM_{2.5}$ exposures based on a model linking actual measurements to household parameters found in DHS surveys – India

   - Can link HAP with IER models
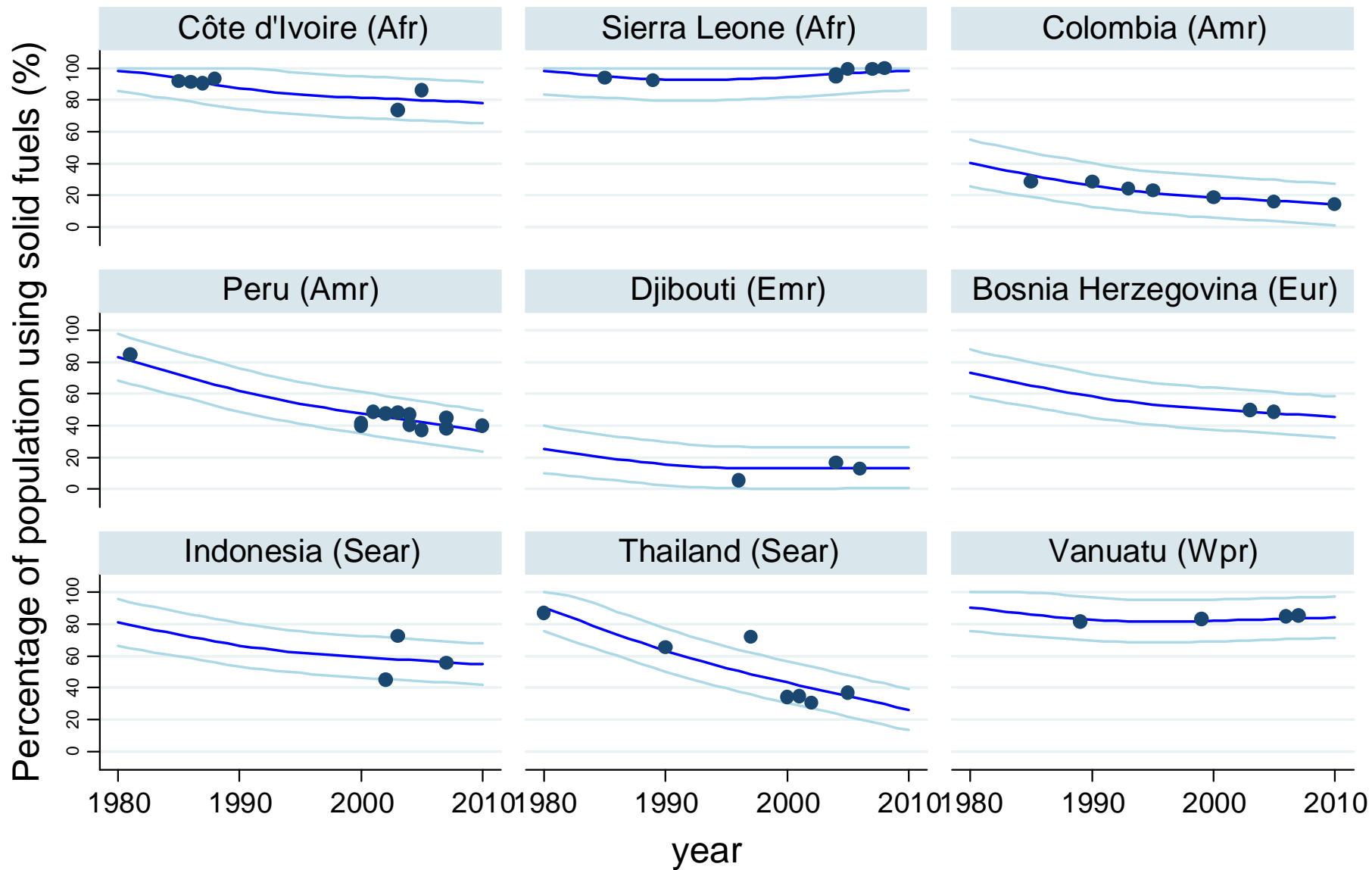
Bonjour et al., CRA-2010
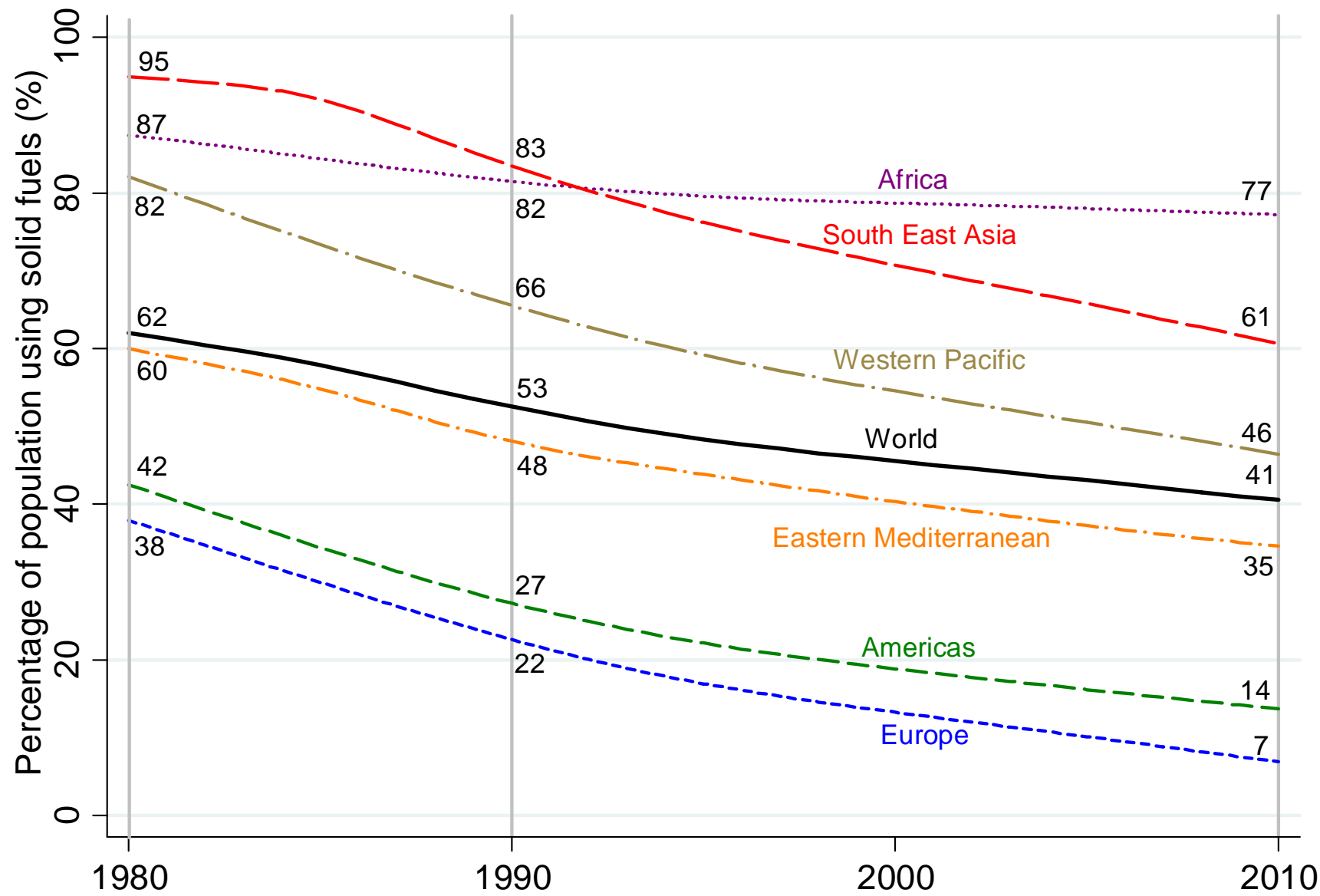
Percent of households cooking with solid fuels by region

Bonjour et al., CRA-2010
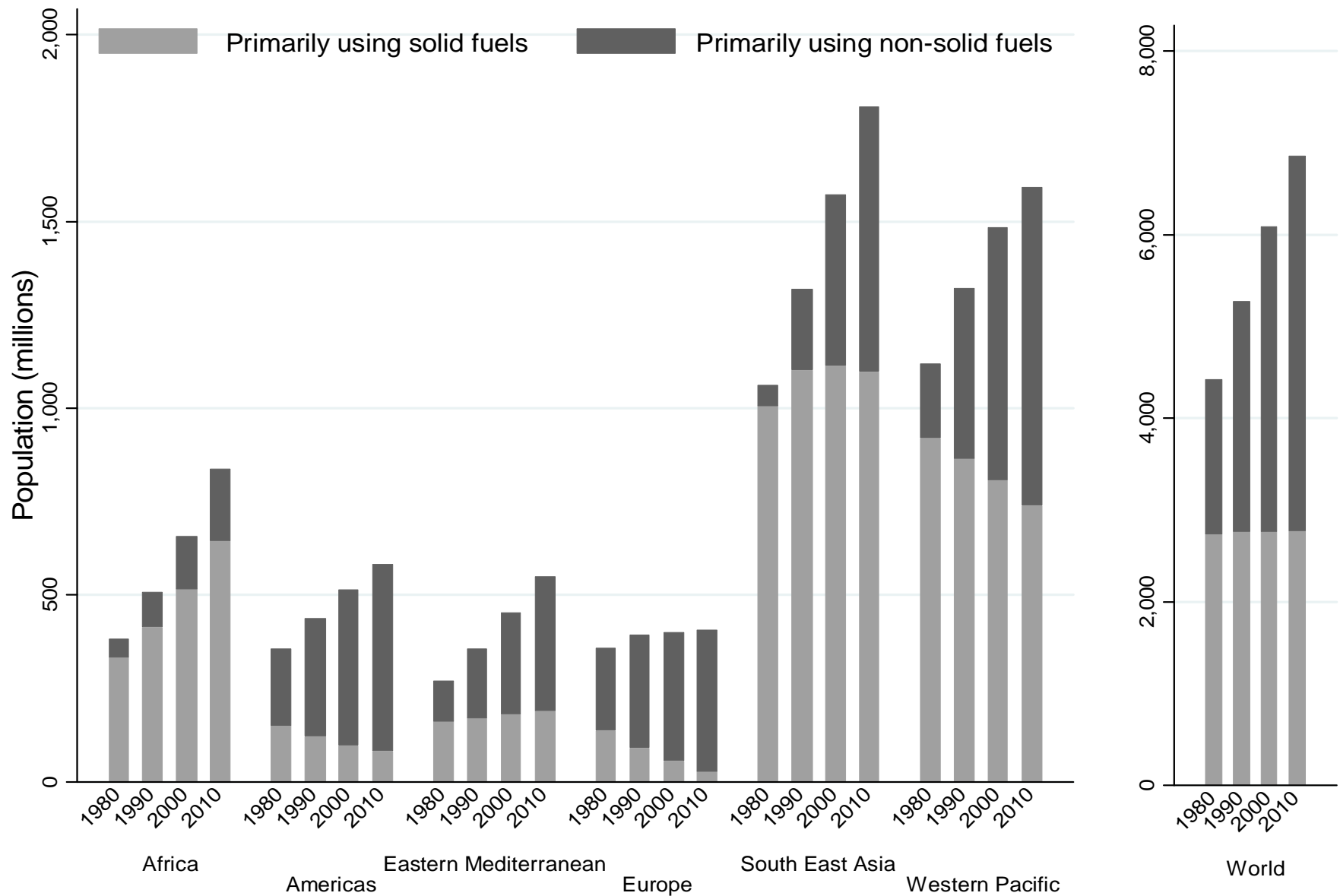
Total Population Cooking with Solid Fuels

Bonjour et al., CRA-2010

2. Estimating the HAP contribution to ambient PM$_{2.5}$ concentrations

- IIASA Greenhouse Gas and Air Pollution Interactions and Synergies (GAINS) model gives the fraction of of total household PM2.5 emissions attributable to the combustion of solid fuels for household cooking, by country.

- TM5-FASST database of the European Joint Research Centre gives the fraction that total household emissions make of total PM2.5 emissions.

- Together they give the proportion of total PM2.5 attributable to household cooking.

- Do not deal directly with secondary PM formation, but estimated fractions reasonable given HH sources also include PM precursors.

%PM$_{2.5}$ from "Residential" Emissions from INTEX_B

% of Anthropogenic Primary PM2.5 from Residential Sources (INTEX_B 2006)
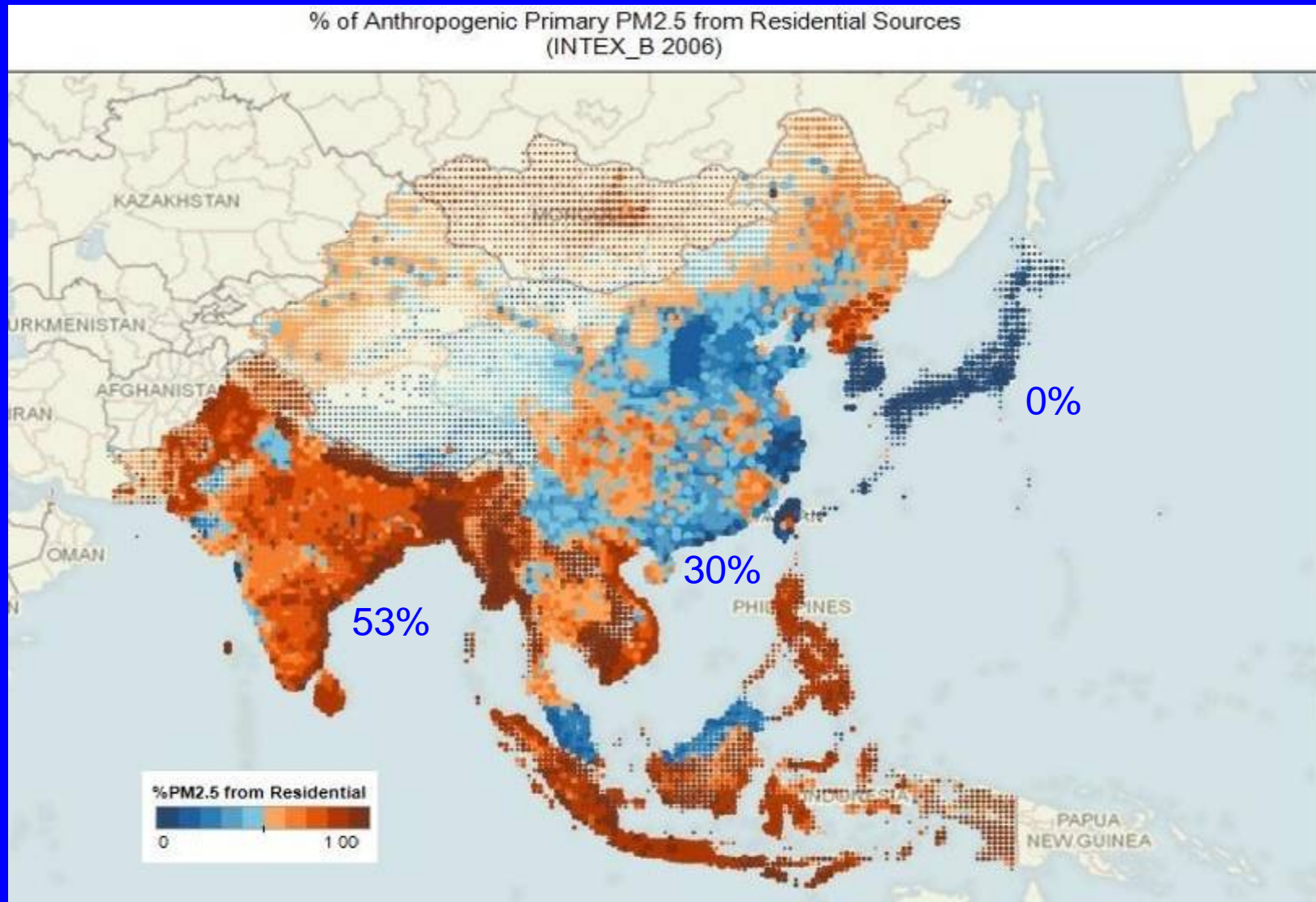
Sectoral contributions to total PM2.5 (population-weighted), 2010

Legend:
- Household Cooking
- Household (other)
- Non-household

Y-axis: Total PM2.5 (ug/m3)

X-axis categories: INDIA, CHINA, UNITED STATES, INDONESIA, BRAZIL, PAKISTAN, BANGLADESH, NIGERIA

Chafe et al., CRA-2010
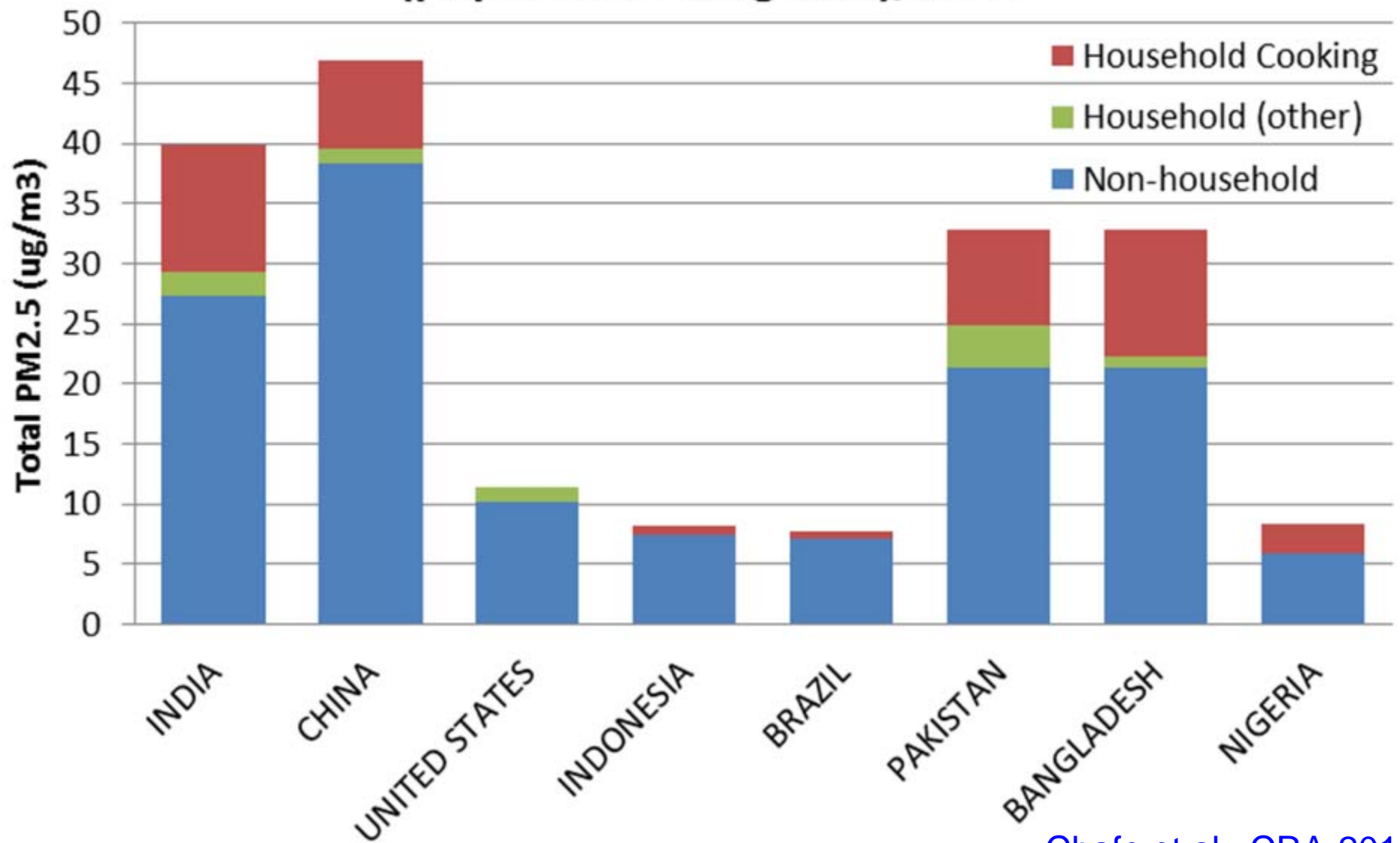
Total PM2.5 from household cooking, and average annual total PM2.5 (population-weighted) in 2010
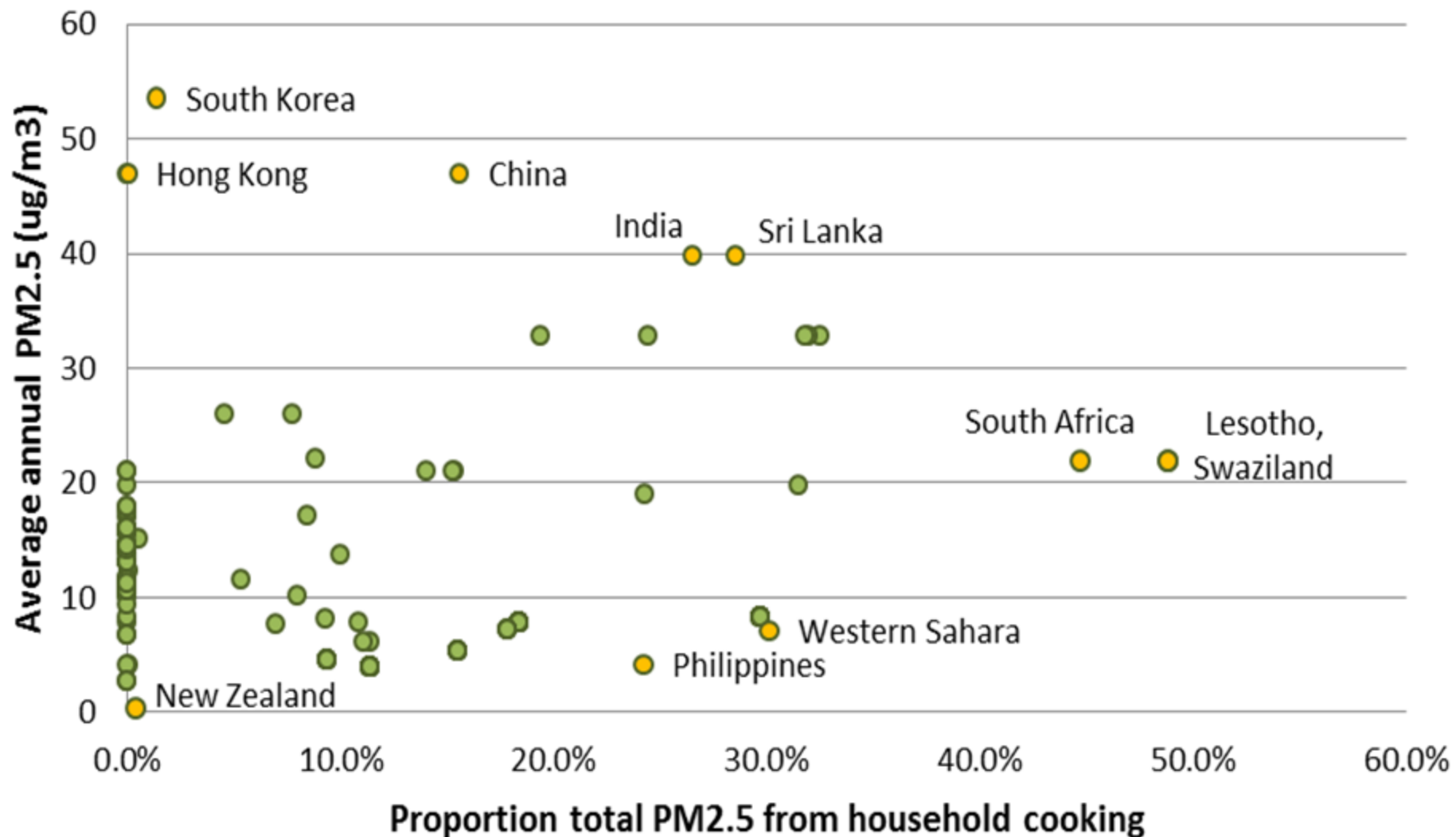
3. Estimating household PM$_{2.5}$ exposures in India

- Large-scale monitoring studies in six states modeled against household parameters commonly assessed in household surveys – fuel type, kitchen location, stove type, agro-climatic region

- National exposure estimates can thus be estimated without measurements in each state

- Distribution and trends can be assessed

- Exposure distributions will be derived for major combinations of fuel use and kitchen type in India

- Distribution of modeled exposures will enable use of continuous exposure-response functions

Ranges of pollutants (PM2.5) across the six states with systematic measurements

Indoor PM Area concentration (μ g/m3)

- ■ >1000
- ■ 500-1000
- ■ 300-500
- □ Unknown
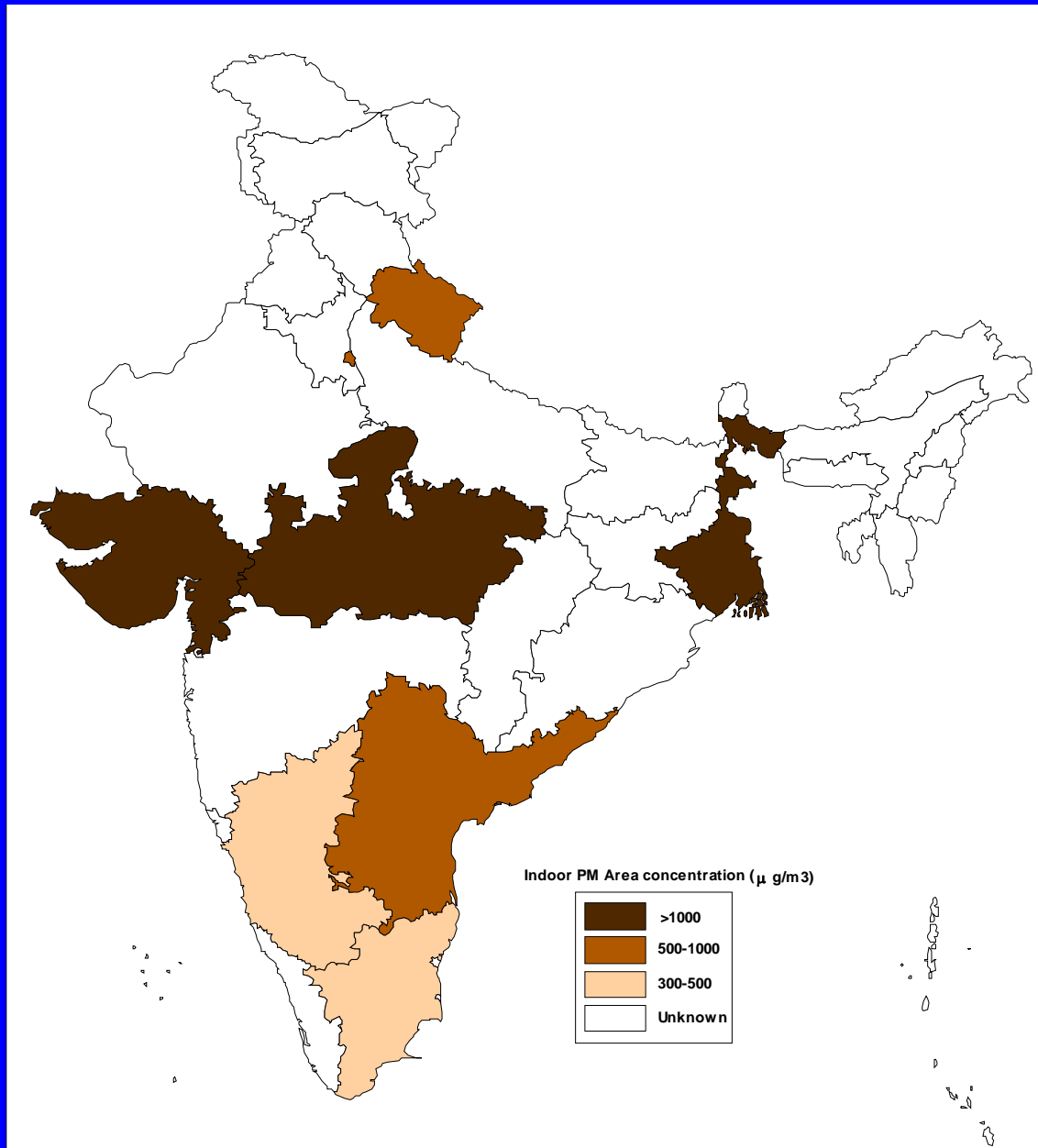
Balakrishnan, et al, -- CRA-2010

Exposure Model for India based on measurements in ~1000 households

~330 ug/m3 mean WHO guidelines = 10-35 ug/m3

Estimated PM2.5 exposure
For women in solid-fuel-using households

Balakrishnan, et al. Household Air Pollution Comparative Risk Assessment-2010

24 HRS Kitchen Concentration (mic.g/m3)

800   700   600   500   400   300   200   100
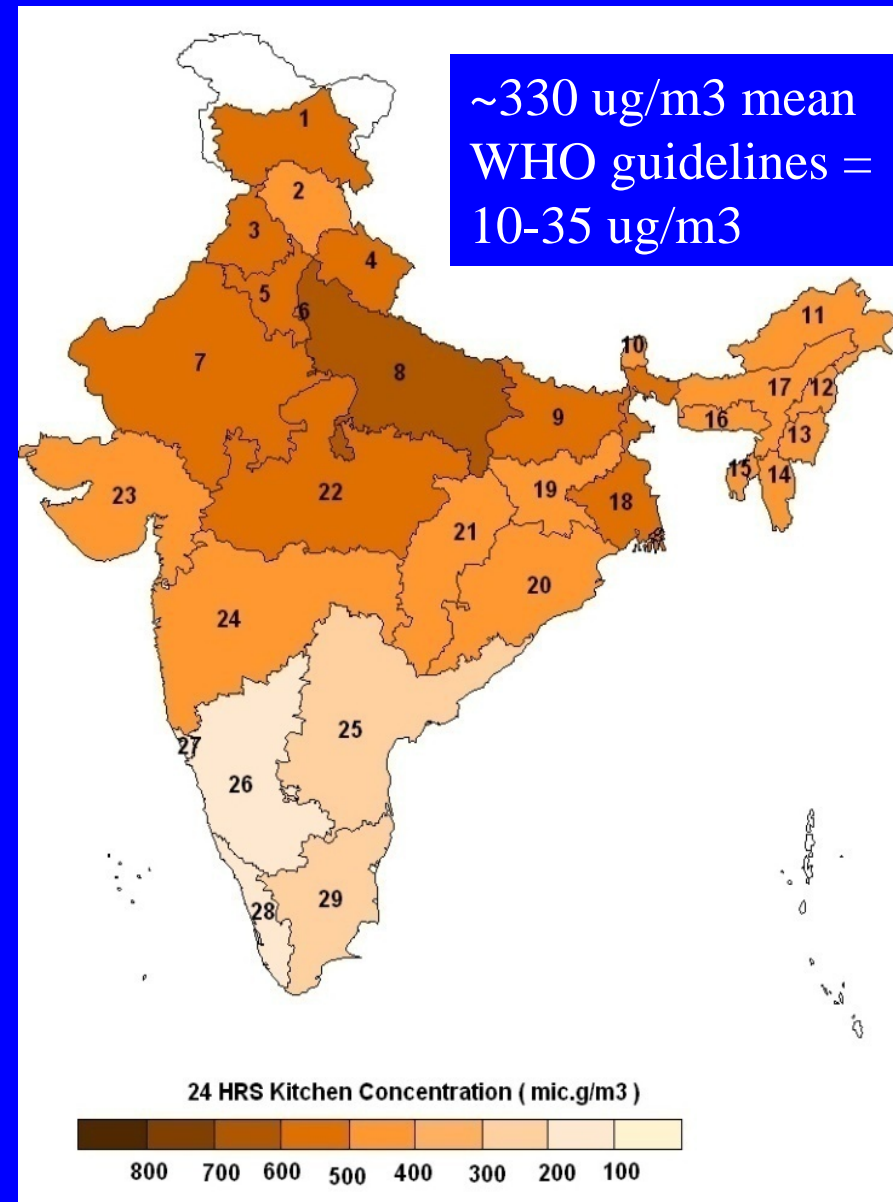
Household air pollution (HAP)

Health outcomes

Evidence classes

- All evidence classes have plausible physiological mechanisms based on toxicology
- Class Ia: Quantified primary outcome
  - Multiple epi of good quality in LDC household settings sufficient for meta-analysis
  - Consistent results as well as significant and positive summary estimate
  - Supporting epi from other particle exposures
  - Fits RR trends for other particle exposure categories

Evidence classes

- Class Ib: Quantified primary outcome, cont.
  - Very strong epi from other particle exposure settings both at higher and lower exposure allowing interpolation for HAP

Evidence classes, cont.

- Class II: Quantified secondary outcome
  - Multiple epi LDC household settings sufficient for MA
  - Unconvincing adjustment for confounding and/or exposure assessment
  - Inconsistent results and/or non-significant positive result
  - Supporting epi from other particle exposures
  - Generally fits RR trends for other particle exposure categories

Evidence classes, cont.

- Class III: Unquantified secondary outcome
  - Still thought likely to be causal
  - Weak or insufficient epi from LDC households for meta-analysis
  - Less strong support from other particle exposure categories

Overview

Level	Outcomes	Planned reporting
I (a)	Child ALRI, Lung cancer, COPD, LBW, Cataract	Full description of review Effect estimate
I (b)	CVD (including stroke)	Full description of methods Effect estimate

Overview

Level	Outcomes	Planned reporting
I (a)	Child ALRI, Lung cancer, COPD, LBW, Cataract	Full description of review Effect estimate
I (b)	CVD (IHD and stroke)	Full description of methods Effect estimate
II	TB, Cancer of UADT	Briefer description of methods; forest plot
III	Ca cervix, adult ALRI, asthma, O/Media, cognitive effects	Briefer description of methods; forest plot

Only Level I included in GBD

Level	Outcomes	Planned reporting
I (a)	Child ALRI, Lung cancer, COPD, LBW, Cataract	Full description of review Effect estimate
I (b)	CVD (including stroke)	Full description of methods Effect estimate
II	TB, Cancer of UADT	Briefer description of methods; forest plot
III	Ca cervix, adult ALRI, asthma, O/Media, cognitive effects	Briefer description of methods;
Outside system	Burns, hygiene, time saving, climate and forests	Descriptive summary, with indication of evidence available
Benefit	Malaria	Summary of recent review

Child ALRI

Child ALRI: Evidence and approach

- Evidence available:
  - Published SR, mainly observational studies [Dherani (2008)]
  - RESPIRE (RCT) (a) Intention to treat (b) exposure response association [Smith (2011)]
- Approach taken:
  - Assess consistency of observational and RCT effects (ITT and exposure-response)
  - IER links RESPIRE exposure-response with OAP and SHS epidemiology for PM2.5
  - Derive OR for 'typical' PM2.5 child HAP exposure seen for homes using solid fuels vs. counterfactual

Study design	N*	OR	95% CI
Intervention	2	1.28	1.06, 1.54
Cohort	7	2.12	1.06, 4.25
Case-control	15	1.97	1.47, 2.64
Cross-sectional	3	1.49	1.21, 1.85
All	26	1.78	1.45, 2.18

Dherani et al Bull WHO (2008)

Study or sub-category	Odds Ratio (random) 95% CI	Weight %	Odds Ratio (random) 95% CI
01 Intervention Studies			
Smith(2007)a		5.53	1.18 [0.88, 1.58]
Smith(2007)b		5.73	1.35 [1.05, 1.73]
Subtotal (95% CI)		11.26	1.28 [1.06, 1.54]
Test for heterogeneity: Chi² = 0.48, df = 1 (P = 0.49), I² = 0%			
Test for overall effect: Z = 2.54 (P = 0.01)			
02 Cohort Studies			
Armstrong(1991)a		2.80	0.50 [0.20, 1.22]
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		1.52	40.65 [9.79, 168.75]
Subtotal (95% CI)		25.11	2.12 [1.05, 4.25]
Test for heterogeneity: Chi² = 54.07, df = 6 (P < 0.00001), I² = 88.9%			
Test for overall effect: Z = 2.11 (P = 0.03)			
03 Case-Control Studies			
Azizi(1995)		3.97	1.20 [0.65, 2.21]
Broor(2001)		4.49	2.51 [1.51, 4.17]
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]
Kumar(2004)		2.45	3.87 [1.42, 10.57]
Mahalanabas(2002)		3.63	3.97 [2.00, 7.88]
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: Chi² = 32.72, df = 14 (P = 0.003), I² = 57.2%			
Test for overall effect: Z = 4.53 (P < 0.00001)			
04 Cross-sectional Studies			
Mishra(2003)		3.83	2.20 [1.16, 4.18]
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: Chi² = 3.19, df = 2 (P = 0.20), I² = 37.3%			
Test for overall effect: Z = 3.74 (P = 0.0002)			
Total (95% CI)		100.00	1.78 [1.45, 2.18]
Test for heterogeneity: Chi² = 101.74, df = 26 (P < 0.00001), I² = 74.4%			
Test for overall effect: Z = 5.61 (P < 0.00001)			

0.1 0.2 0.5 1 2 5 10

Increased risk   Decreased risk

Funnel plot - all studies:
Assessment for evidence of publication bias

Funnel plot with pseudo 95% confidence limits

All: Begg's p=0.027; Eggar's p=0.005

Excl Pandey: Begg's p=0.098; Eggar's p=0.016

Pandey 1989 (Group II)
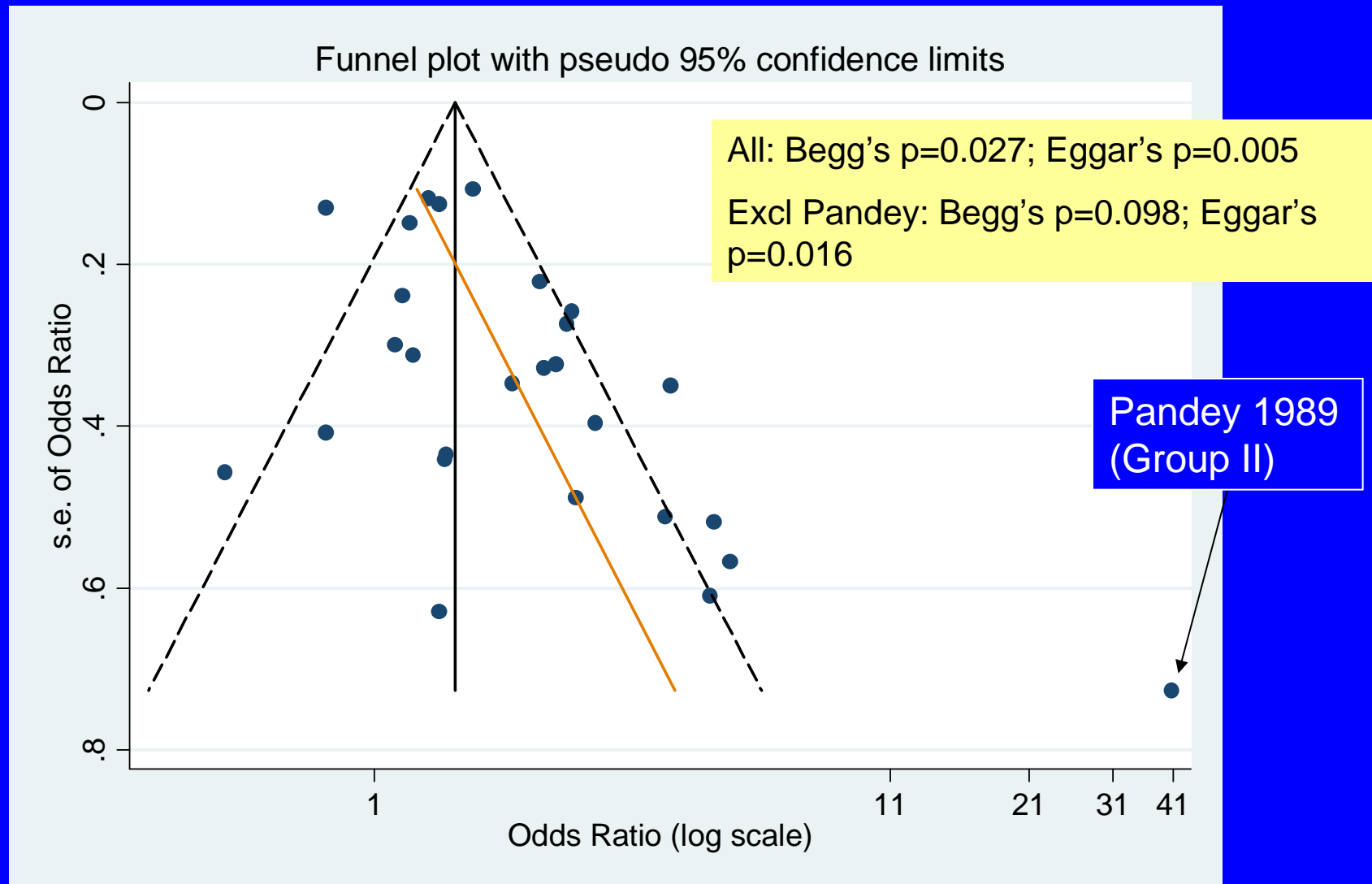
Sensitivity analysis

Group	Detail	Random effects estimates		
		N	OR	95% CI
All studies	All	27	1.78	1.45, 2.18
	Excl. Pandey (outlier)	26	1.67	1.39, 2.01
	+ excl. Low prevalence	22	1.79	1.46, 2.21
Controls	Unbiased selection	9	1.50	1.05, 2.14
Confounding	Good adjustment	16	1.77	1.43, 2.18
Exposure	Good categorisation	16	1.67	1.33, 2.09
	Solid vs. clean fuel	14	1.69	1.29, 2.20
Outcome measure	Excluding DHS	23	1.72	1.37, 2.17
	MD diagnosis/CXR	20	1.65	1.26, 2.15
Age group	< 60 months	11	1.62	1.21, 2.15
	< 36 months	4	2.05	1.38, 3.07
	< 24 months	12	1.96	1.36, 2.82
Adjustment for publication bias		[-3 studies]* 1.64 (1.34, 2.01) [-5 studies]** 1.54 (1.25, 1.89)		

* Manual trim   ** Metatrim (Stata)

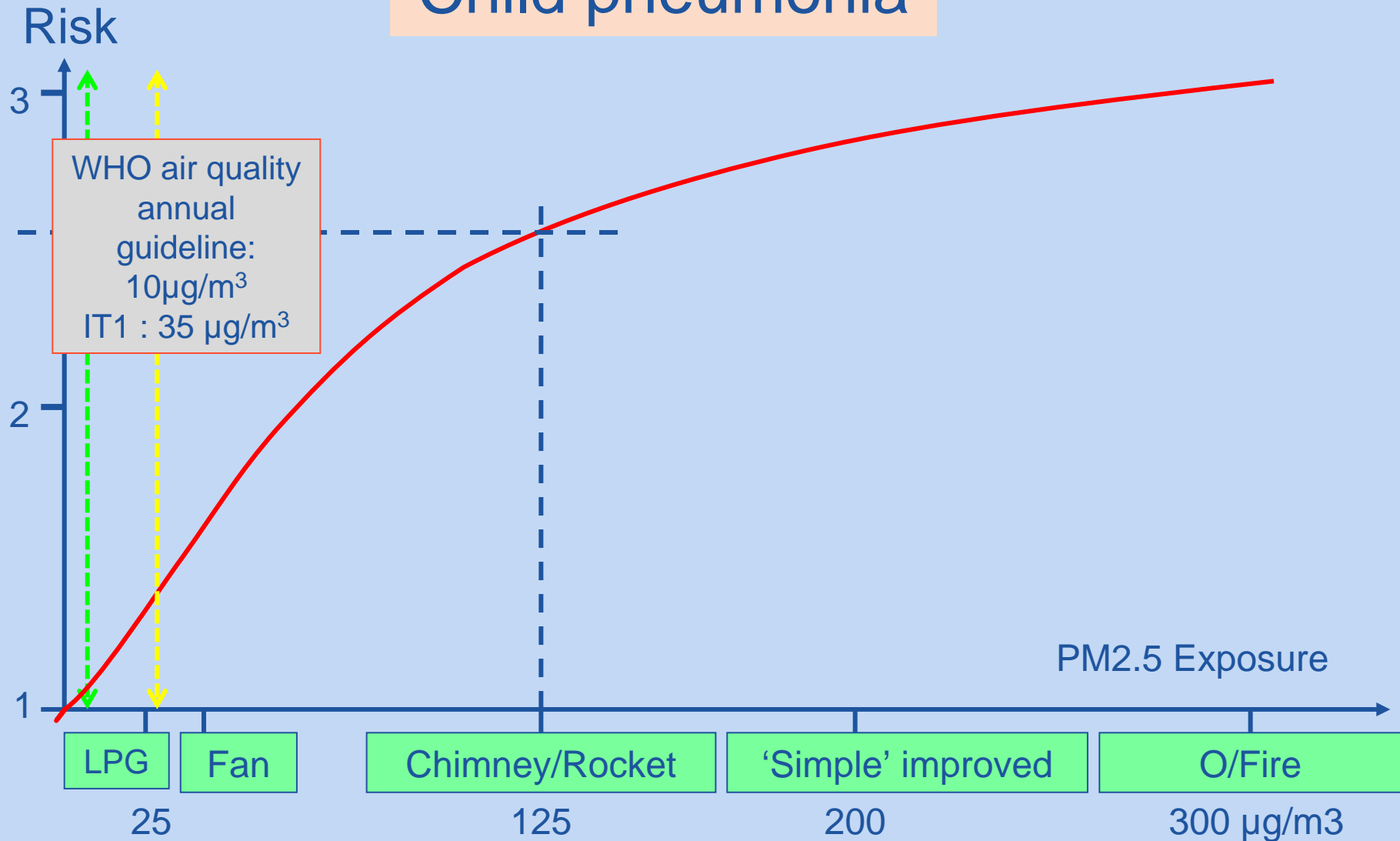# Exposure – response relationship
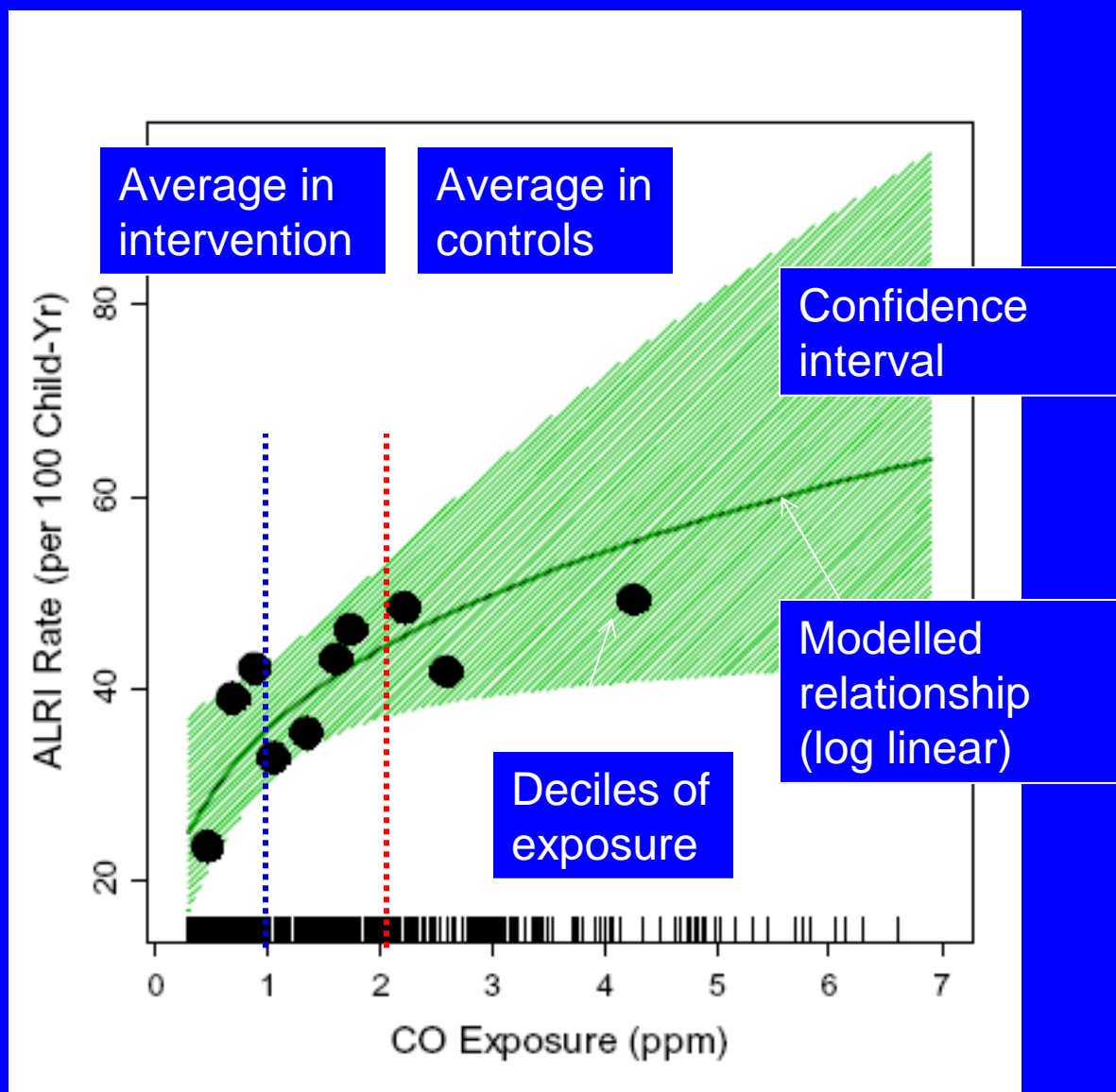
Physician-diagnosed pneumonia

From the

RESPIRE Studies in Guatemala

Pneumonia from combustion particles
Annual average PM2.5 in ug/m3

CRA, 2010
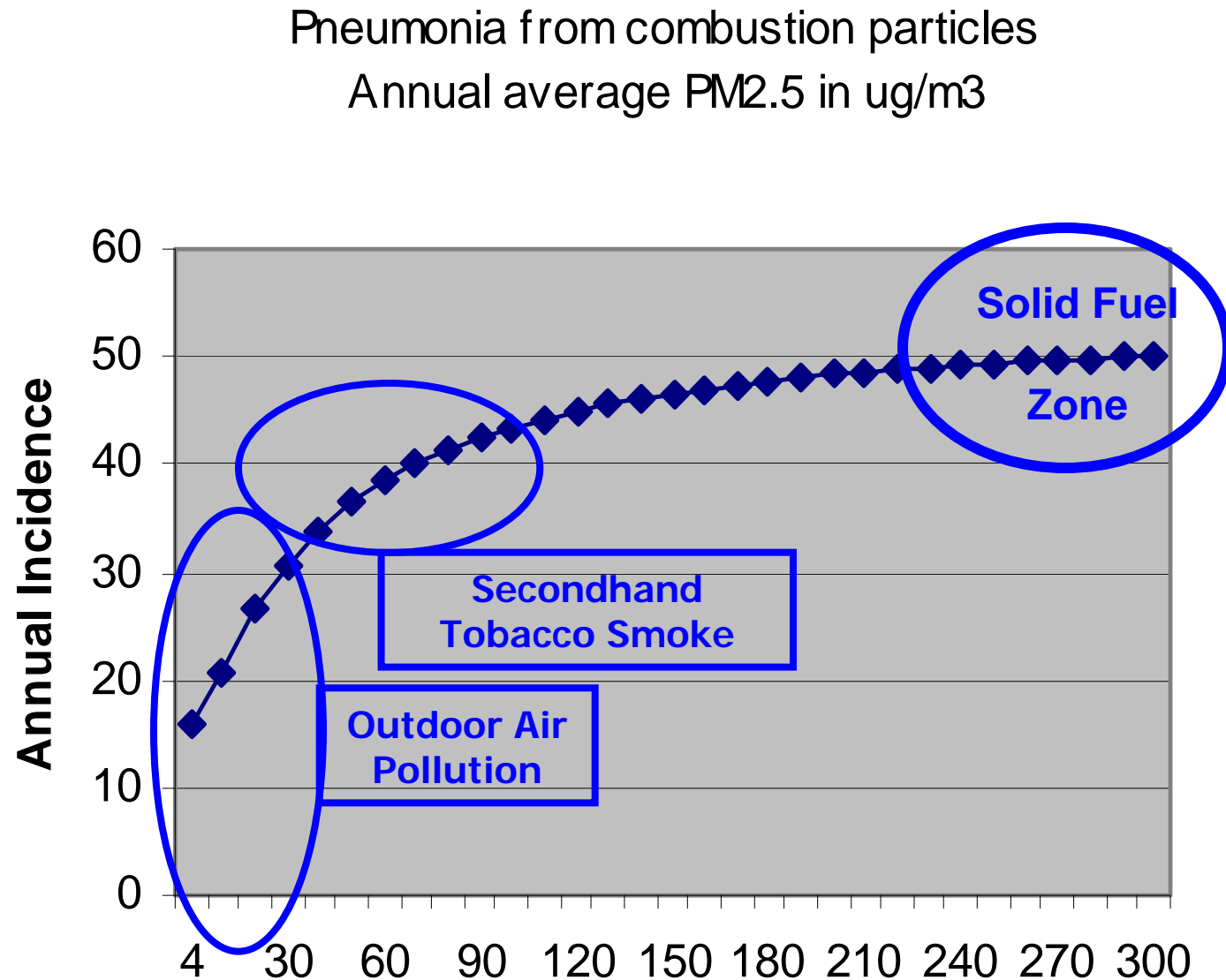
# Exposure-response relationship

Child pneumonia

Cardiovascular Disease

Evidence and approach

- No studies of CVD and HAP
  - But studies showing effects on blood pressure and ST-segment, important disease signs
- Epidemiologic evidence shows clear, consistent evidence of increasing risk
  - at higher exposures – ATS
  - and lower exposures – OAP and SHS
- Interpolation indicates that HAP would also increase risk

Heart Disease and Combustion Particle Doses

From "Mind the Gap," Smith/Peel, 2010 and Pope et al., 2009
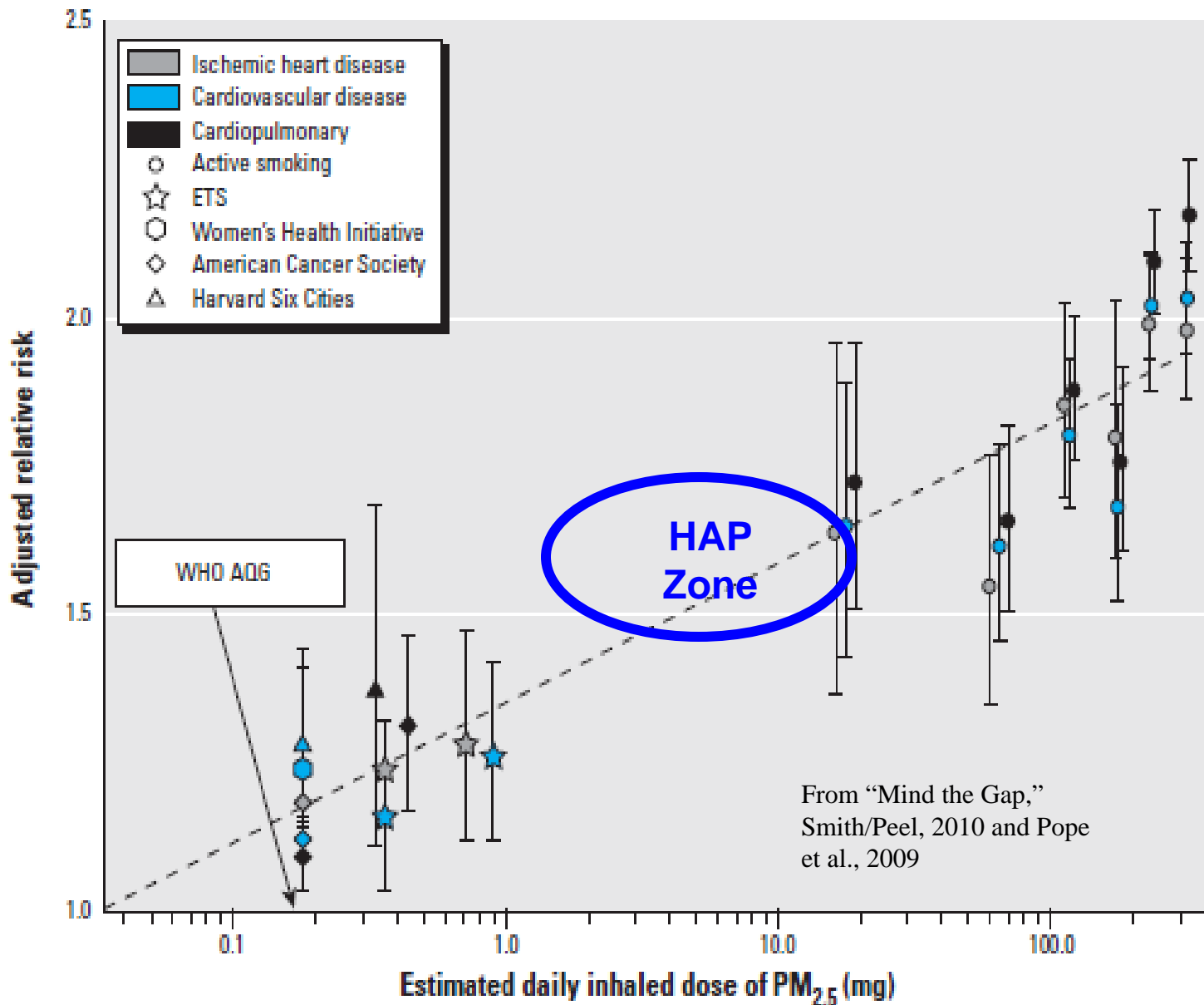
Chimney Stove Intervention to Reduce Long-term Wood Smoke Exposure Lowers Blood Pressure among Guatemalan Women

John P. McCracken,[1,2] Kirk R. Smith,[3] Anaité Díaz,[4] Murray A. Mittleman,[1,5] and Joel Schwartz[1,2]

EHP, 2007

Between group analysis

Table 3. Crude and adjusted between-group differences in SBP and DBP (mm Hg) associated with *plancha* compared with open fire use during the trial period.

| | No. of subjects (measures) | | Crude mean difference | | | Adjusted mean difference[a] | | |
	Control group	Intervention group	Estimate	95% CI	p-Value	Estimate	95% CI	p-Value
SBP	71 (111)	49 (115)	−2.3	−6.6 to 2.0	0.30	−3.7	−8.1 to 0.6	0.10
DBP	71 (111)	49 (115)	−2.2	−4.7 to 0.3	0.09	−3.0	−5.7 to −0.4	0.02

Before and after analysis

Table 4. Crude and adjusted within-subject differences in SBP and DBP (mm Hg) after the *plancha* echo-intervention compared with before.

| | No. of subjects (measures) | | Crude mean difference | | | Adjusted mean difference[a] | | |
	Trial period	Echo-intervention	Estimate	95% CI	p-Value	Estimate	95% CI	p-Value
SBP	55 (88)	55 (65)	−3.7	−6.0 to −1.4	0.002	−3.1	−5.3 to −0.8	0.01
DBP	55 (88)	55 (65)	−2.3	−3.8 to 0.9	0.003	−1.9	−3.5 to −0.4	0.01

Household
Air
Pollution
and
Blood Pressure

In Yunnan, China

Baumgartner et al.
EHP 2011
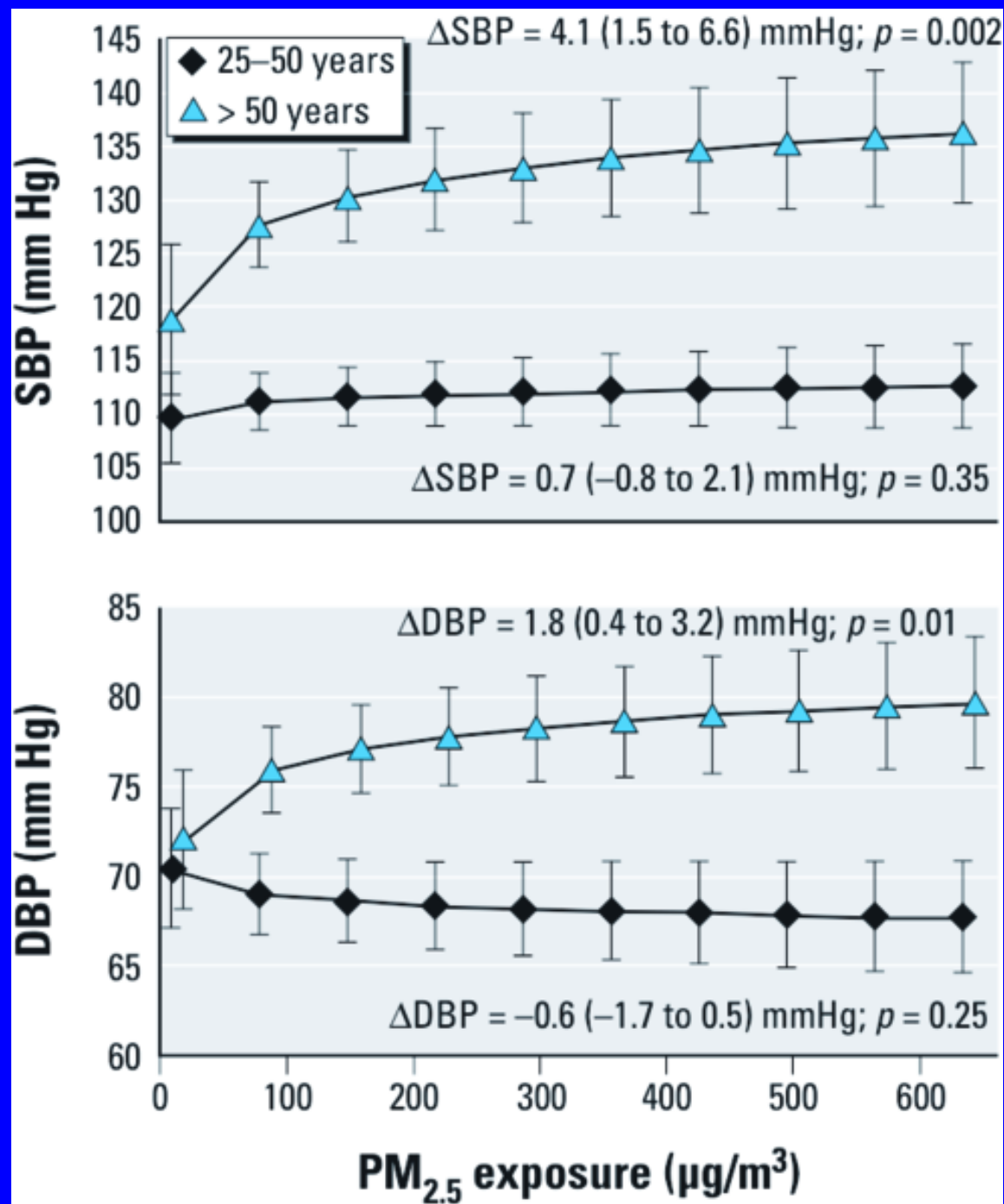
Intervention to Lower Household Wood Smoke Exposure in Guatemala Reduces ST-Segment Depression on Electrocardiograms

John McCracken,[1,2] Kirk R. Smith,[2] Peter Stone,[3] Anaité Díaz,[4] Byron Arana,[4] and Joel Schwartz[1]

[1]Department of Environmental Health, Harvard School of Public Health, Boston, Massachusetts, USA; [2]Environmental Sciences Division, University of California, Berkeley, California, USA; [3]Brigham and Women's Hospital, Boston, Massachusetts, USA; [4]Center for Health Studies, Universidad del Valle, Guatemala City, Guatemala

Table 3. Odds ratios (ORs) for nonspecific ST-segment depression (30-min average ≤ –1 mm, regardless of slope) associated with chimney-stove intervention compared with open fire from two study designs: between-groups and before-and-after analyses.
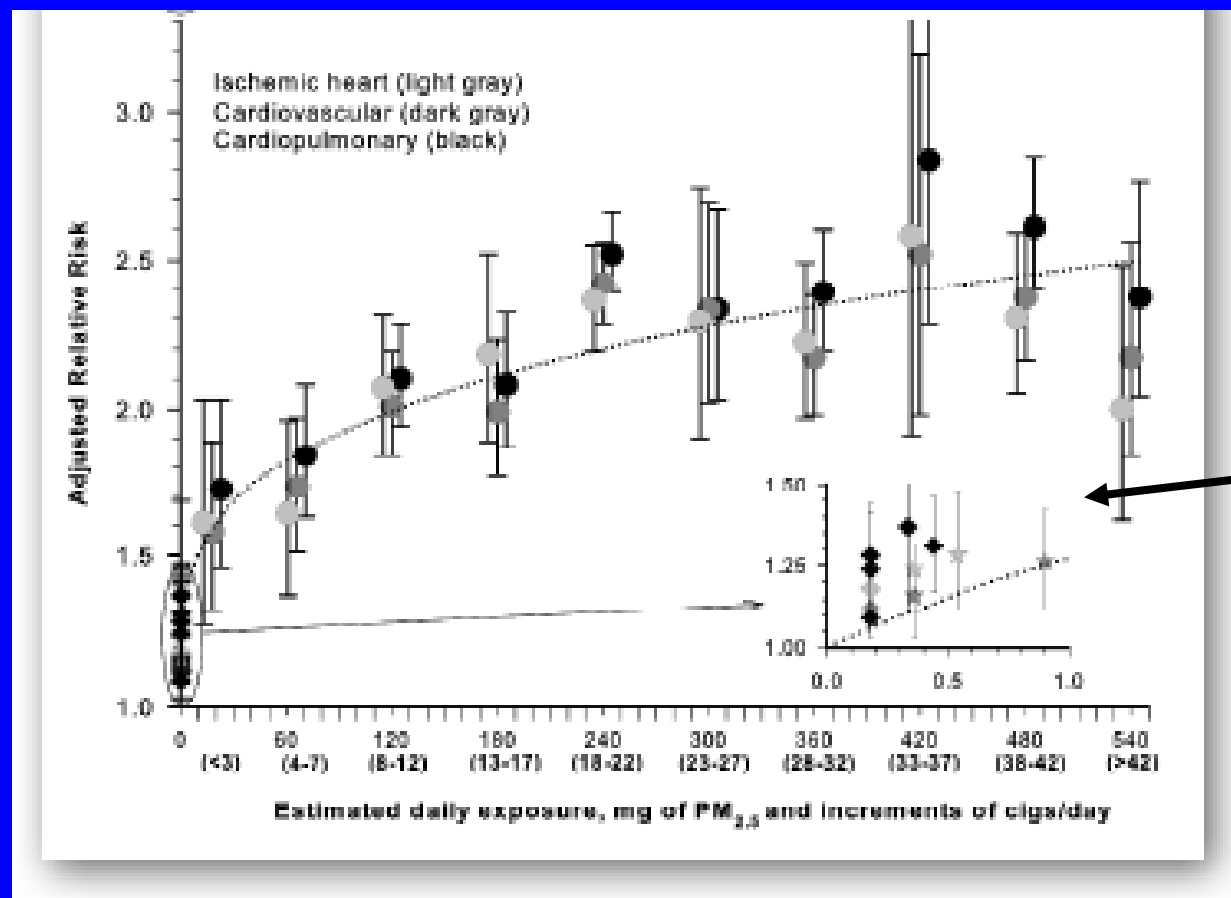
Comparison	Crude		Adjusted	
	OR (95% CI)	p-Value	OR (95% CI)	p-Value
Between-groups	0.34 (0.15, 0.81)	0.015	0.26 (0.08, 0.90)[a]	0.033
Before-and-after (only control group)	0.41 (0.24, 0.70)	0.001	0.28 (0.12, 0.63)[b]	0.002

[a]Adjusted for age (quadratic), BMI (quadratic), asset index category, ever smoking, SHS, owning a wood-fired sauna, recent use of wood-fired sauna, and time of day (natural spline with 5 degrees of freedom). [b]Adjusted for age (quadratic), day of week, season (wet/dry), daily average temperature and relative humidity, daily rainfall, interactions of weather variables with season, recent use of wood-fired sauna, and time of day (natural spline with 5 degrees of freedom).

Table 2. Adjusted relative risk estimates[a] for various increments of exposure from cigarette smoking (versus never smokers), second hand cigarette smoke, and ambient air pollution from the present analysis and selected comparison studies.

Source of risk estimate	Increments of Exposure	Adjusted RR (95% CI)				Estimated Daily Dose PM$_{2.5}$ (mg)[b]
		Lung Cancer	IHD	CVD	CPD	
ACS- present analysis	≤3 (1.5) cigs/day	10.44 (7.30-14.94)	1.61 (1.27-2.03)	1.58 (1.32-1.89)	1.72 (1.46-2.03)	18
ACS- present analysis	4-7 (5.5) cigs/day	8.03 (5.89-10.96)	1.64 (1.37-1.96)	1.73 (1.51-1.97)	1.84 (1.63-2.08)	66
ACS- present analysis	8-12 (10) cigs/day	11.63 (9.51-14.24)	2.07 (1.84-2.31)	2.01 (1.84-2.19)	2.10 (1.94-2.28)	120
ACS- present analysis	13-17 (15) cigs/day	13.93 (11.04-17.58)	2.18 (1.89-2.52)	1.99 (1.77-2.23)	2.08 (1.87-2.32)	180
ACS- present analysis	18-22 (20) cigs/day	19.88 (17.14-23.06)	2.36 (2.19-2.55)	2.42 (2.28-2.56)	2.52 (2.39-2.66)	240
ACS- present analysis	23-27 (25) cigs/day	23.82 (18.80-30.18)	2.29 (1.91-2.75)	2.33 (2.02-2.69)	2.33 (2.03-2.67)	300
ACS- present analysis	28-32 (30) cigs/day	26.82 (22.54-31.91)	2.22 (1.97-2.49)	2.17 (1.98-2.38)	2.39 (2.19-2.60)	360
ACS- present analysis	33-37 (35) cigs/day	26.72 (18.58-38.44)	2.58 (1.91-3.47)	2.52 (1.98-3.19)	2.83 (2.28-3.52)	420
ACS- present analysis	38-42 (40) cigs/day	30.63 (25.79-36.38)	2.30 (2.05-2.59)	2.37 (2.16-2.59)	2.61 (2.40-2.84)	480
ACS- present analysis	43+ (45) cigs/day	39.16 (31.13-49.26)	2.00 (1.62-2.48)	2.17 (1.84-2.56)	2.37 (2.04-2.76)	540
ACS-air pol. original	24.5 µg/m^3 ambient PM$_{2.5}$	-----	-----	-----	1.31(1.17-1.46)	0.44
ACS-air pol. extend.	10 µg/m^3 ambient PM$_{2.5}$	1.14(1.04-1.23)	1.18(1.14-1.23)	1.12(1.08-1.15)	1.09(1.03-1.16)	0.18
HSC-air pol. original	18.6 µg/m^3 ambient PM$_{2.5}$	-----	-----	-----	1.37(1.11-1.68)	0.33
HSC-air pol. extend.	10 µg/m^3 ambient PM$_{2.5}$	1.21(0.92-1.69)	-----	1.28(1.13-1.44)	-----	0.18
WHI-air pol.	10 µg/m^3 ambient PM$_{2.5}$	-----	-----	1.24(1.09-1.41)[c]	-----	0.18
SGR-SHS	Low- moderate SHS exp.	-----	-----	1.16(1.03-1.32)	-----	0.36
SGR-SHS	Moderate-high SHS exp	-----	-----	1.26(1.12-1.42)	-----	0.90
SGR-SHS	Live with smoking spouse	1.21(1.13-1.30)	-----	-----	-----	0.54
SGR-SHS	Work with SHS exposure	1.22(1.13-1.33)	-----	-----	-----	0.72
INTERHEART	1-7 hrs/wk SHS exp.	-----	1.24(1.17-1.32)[d]	-----	-----	0.36
INTERHEART	Live with smoking spouse	-----	1.28(1.12-1.47)[d]	-----	-----	0.54

CVD

Generalized Exposure-Response: Outdoor Air, SHS, and Smoking and Heart Disease

IHD risks from combustion particles
Annual average PM2.5 in ug/m3

Smokers →

Relative Risk axis: 1.00, 1.20, 1.40, 1.60, 1.80, 2.00, 2.20

X-axis: 4, 30, 60, 90, 120, 150, 180, 210, 240, 270, 300

Solid Fuel Zone

Secondhand Tobacco Smoke

Outdoor Air Pollution
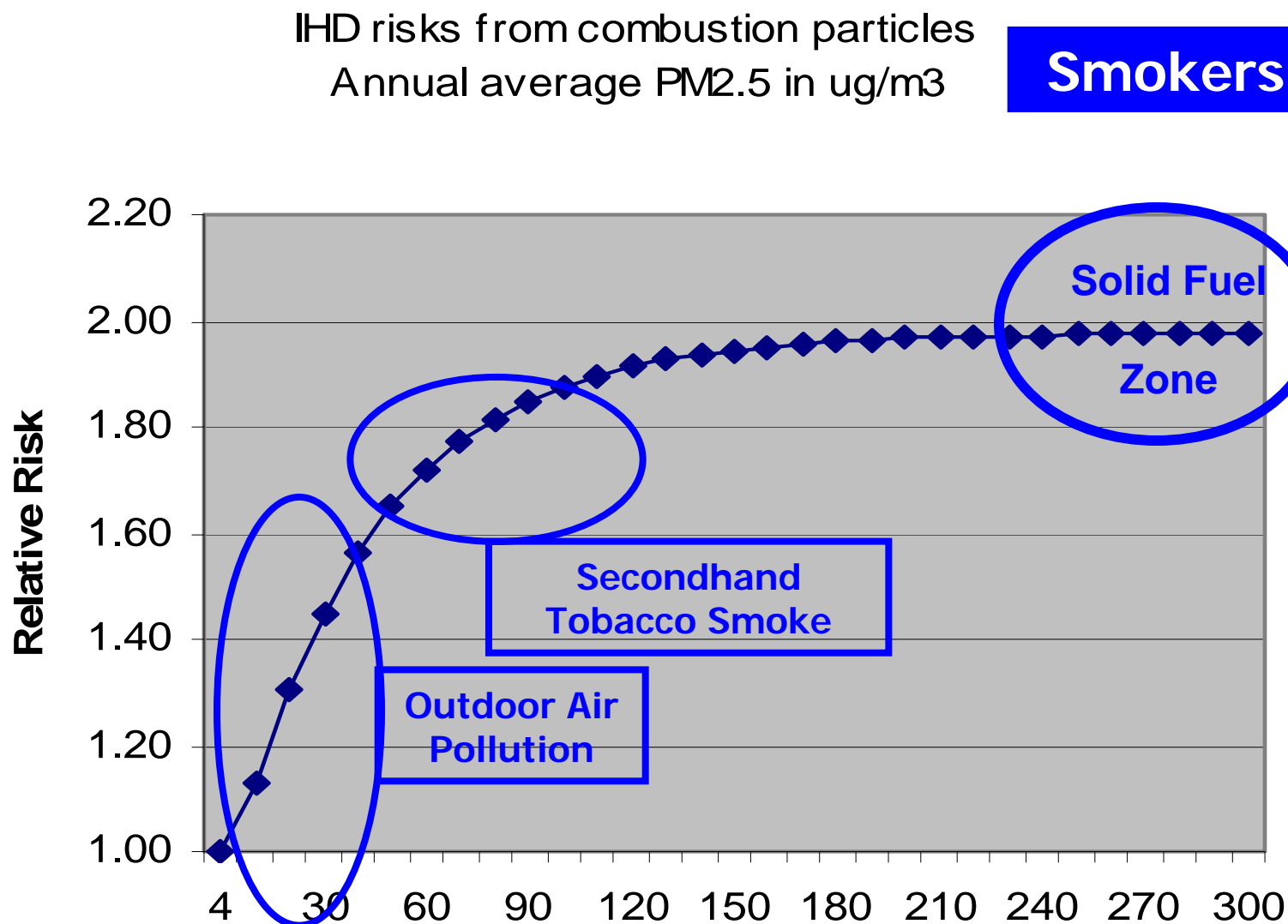
CRA, 2011

Disease	Sex	Age	RR	Evidence
ALRI	M/F	<60 mo	2.71	SR/MA, RCT, IER
Cataracts	F	>25 y	2.47	SR/MA
COPD	F	>25 y	??	??
COPD	M	>25 y	??	??
Lung Cancer	F	>25 y	1.99	SR/MA, IER
Lung Cancer	M	>25 y	1.60	SR/MA, IER
IHD	F	25-80+ y	2.83-1.31	IER
IHD	M	25-80+ y	2.53-1.27	IER
Cerebro	F	25-80+ y	4.50-1.57	IER
Cerebro	M	25-80+ y	3.71-1.54	IER

Preliminary estimates suggest the HAP burden is considerably higher than in the CRA-2000

- Because evidence now supports
  - 1) impacts on men as well as women,
  - 2) inclusion of additional diseases (CVD, LC from biomass, and cataract);
  - 3) a portion of OAP burden is now included
  - 4) a lower counterfactual level is applied, equivalent to cooking with gas
- In spite of smaller COPD RRs and a smaller global background rate of ALRI

Thanks to Funders

HAP CRA Website: http://ehs.sph.berkeley.edu/krsmith/page.asp?id=25