

# Why test the smoke reduction – better lung health hypothesis?

- Data clearly link smoke pollution (IAP) in kitchens with ALRI (mostly childhood pneumonia) & COPD
- There are pretty good data that show lower overall respiratory infections with less smoke exposure
- However, there is little data for pneumonia or COPD reductions after interventions to reduce particulates
- Currently, our estimates of benefit are based on extrapolation from outdoor pollution – however, particulate levels in those studies are much lower\*

IAP = Indoor air pollution; ALRI = Acute lower respiratory infection; COPD = Chronic obstructive pulmonary disease

# We're talking orders of magnitude difference

Indoor concentrations of particles usually exceed guideline levels by a large margin:

24-hour mean PM10 levels are typically in the range 300–3000 mg/m<sup>3</sup> and may reach 30 000 mg/m<sup>3</sup> or more during periods of cooking.

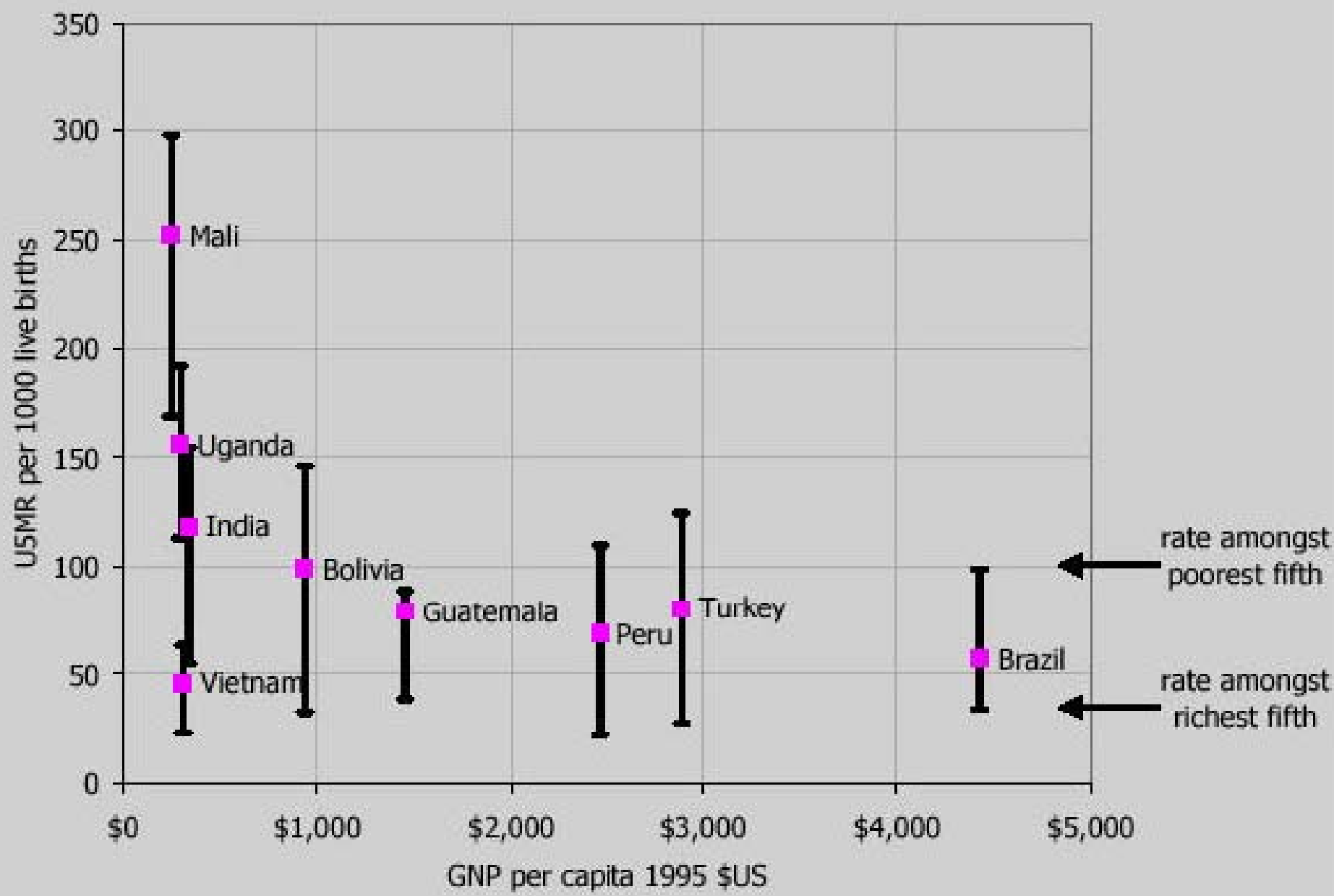
The United States Environmental Protection Agency's standards for 24-hour average PM10 and PM2.5 concentrations are 150 mg/m<sup>3</sup> (which should be exceeded only once per 100 days). The annual average should not exceed 50 mg/m<sup>3</sup> and 65 mg/m<sup>3</sup> respectively.

[Bruce et al. Bulletin of the WHO 2000, 78(9)]

# Why is extrapolation risky?

- If the relationship between particulates and lung risks is linear and continuous, it's a fine guess
- However, if it is less than linear and/or there is a threshold, our estimates may be thrown way off
- It's possible that, while respiratory infections could be lower overall with 80 to 90% reductions of particulate IAP, pneumonia might be no better
- This may even be likely since the nose, throat and sinuses are mostly reacting to large particles and lung disease is caused by extremely fine particles

Figure 1: Under-5 Mortality: Gaps Between and Within Countries



# Childhood pneumonia deaths

- Even though this causes the biggest number of premature deaths from kitchen IAP, it would take a population of hundreds at risk to have more than a few deaths in a year; total pneumonia will be more but still low
- Collecting reliable data for the latter is also problematic without X-ray machines and with imperfect primary health care delivery

# COPD – the other major cause of death (and disability) from IAP

- About 0.5 million premature deaths per year attributed to COPD from biomass smoke
- The damage from exposure to IAP seems directly comparable to cigarette smoking
- COPD is a progressive disease and has been measured and studied as changes in smoke exposure occur through behavior change

# So how can we gauge success?

- We already know that rocket stoves save fuel and therefore reduce labor and injuries
- We believe the decrease in IAP should also lower the rates of death from lung diseases
- However, IAP reduction lacks prominence in global health programs, even those that targeting problems of women and children
- Better data on health impacts could help focus commensurate attention to IAP

# Understanding fine particles and lung damage in ALRI & COPD

- Compounds in both cigarette & biomass smoke cause damage to airway epithelium
- Ultrafine particles (usually  $<2.5$  micron but  $<3.5$  or  $<4$  in some studies) are a cause of a large part of the damage in animal models
- They compromise defenses against lung infection and cause inflammation that leads to airway narrowing as well as permanent damage to pulmonary exhalation force

# Understanding airway obstruction – acute & chronic

- Airflow rate is directly related to this elastic recoil force from lung alveoli (the breathing spaces) and flow resistance in small airways
- Inflammation narrows these small airways by increasing their muscle tone and by the increase in mucus, fluid and cell turnover
- The elastic recoil loss from inflammatory destruction is irreversible but airway size improves rapidly if inflammation decreases

# What happens with long-term tobacco smoke exposure?

- COPD occurs by faster loss of lung elasticity than in non-smokers and from the chronic increase in inflammatory cells, debris, mucus & tissue fluid
- There is large individual variation but a known average rate of loss in pulmonary exhalation force
- The average rate of this loss in smokers is double the age-related loss when measured by spirometer (Forced expiratory volume in 1 second or FEV<sub>1</sub>)
- Over time, the increased difficulty of moving air causes breathing symptoms which we call COPD

# COPD definition & monitoring

- Obstruction that is not reversible by the use of bronchodilator (asthma) medicines
- However, there is often a component of muscular bronchoconstriction in COPD
- Formal definition:  $FEV_1\%$  [FEV/functional capacity]  $< 70\%$  and  $< 80\%$  predicted
- Unfortunately, predictions vary ethnically

# The challenge of measuring change

- The difference in annual decline in  $FEV_1$  between smokers and non-smokers is small\*
- The test itself tolerates 5% intratest variation
- As a result, research only uses averages from large numbers of individuals to find annual difference in decline from smoking cessation
- Fortunately, there is a larger short-term  $FEV_1$  improvement that occurs when smoker's quit

Mean change of 31 ml versus 62 ml or 1.12% versus 2.23% of the mean  $FEV_1$

# What we measured and why

- Rocket stoves should reduce smoke exposure as much as stopping tobacco smoking does
- At one year after cessation of tobacco, there is an average 47 ml improvement in  $FEV_1$
- We planned baseline and 1 yr. follow-up tests in men & women, hypothesizing that with 40 homes we could see the average improve in women but not in men, who are less exposed

# How “real world” issues changed the design of the health testing

- A minority of the 40 households had their stoves in use by 3 mos. after baseline tests so we waited 3 extra months to repeat tests; we couldn't wait more due to rainy season
- Many people with stoves, didn't want to be bothered with doing the tests over for us
- The test is inherently challenging for some so we had a large percent of invalid results
- Men's tests at baseline were as bad as women's

# Saved by studies on the ban on smoking in bars in Scotland

- There is no theory for why smokers who quit would be most improved at 1 year
- Theoretically, improvement should occur in weeks and then decline should set back in
- However, that was the only study available to base this on when I started the design
- But in 2006, Scotland cut non-smoking bar workers a break and researchers did a study

# What really happens with a major reduction in smoke exposure

- Mean improvement in %predicted FEV<sub>1</sub> in non-smoking workers was 8.2, at 1 mo.
- For non-asthmatics only, it was 5.7
- In asthmatics, the improvement was 15.7
- By 2 months, age-related decline had come back and improvement was only 3.38 in non-asthmatics and 10.2 in asthmatics
- Upper 95%ile of non-asthmatics was 6.12



# Unos datos de línea base en esta muestra de 74 personas

	<b>VEF1% (promedio)</b>	(intervalo: bajo a alto)
<b>34 hombres</b>	<b>77,0</b>	(61,3 a 93,5)
<b>40 mujeres</b>	<b>79,1</b>	(61,3 a 87,8)
<b>17 con bronquitis crónica (cuestionario)</b>	<b>77,5</b>	(65,3 a 89,9)
<b>10 hombres que no cocinen regularmente</b>	<b>76,4</b>	(62,7 a 93,5)



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# Diferencia entre personas más o menos viejas (por género)

	<b>VEF1% (promedio)</b>	(intervalo: bajo a alto)
20 mujeres (<35 años)	82,1%	(71,1 a 87,8)
20 mujeres ( $\geq$ 35 años)	76,1	(61,3 a 86,1)
17 hombres( $\leq$ 30 años)	81,4	(66,0 a 93,5)
17 hombres(>30 años)	72,5	(61,3 a 82,3)
8 (>30 años) con la bronquitis crónica	74,4%	(65,3 a 80,7)

# Fourteen people with valid PFTs in both years

PM4 in '05	PM4 in '06		Percent change in FEV1% from 1st to 2nd yr.	Difference from predicted loss if they had had smokers' levels of exposure		
94	617		-6.61	-3.83		
121	30		-2.80	-0.02		
1001	310		5.09	7.87		
2290	288		-1.89	0.89		
650	370		-2.46	0.32		
1430	32		-0.95	1.83		
44	161		-2.09	0.69		
240	307		-6.65	-3.87		
94	122		-4.29	-1.51		
70	83		8.82	11.60		
2546	256		-0.19	2.59		
543	ND		3.05	5.83		
2281	ND		-4.81	-2.03		
565	46		-0.93	1.85		
Mean	218.5	Mean change	-1.19	1.59	2.16	predicted
		Standard deviation		4.35		
		95%C.I. = mean + or - this number		2.28	-0.69	3.87

Split the group in various ways  
(any group closer to predicted 2.16?)

Those with  $< 150 \mu\text{g}/\text{m}^3$  in second year

-1.04 to 6.06

Those with  $>150 \mu\text{g}/\text{m}^3$  in second year

-2.31 to 3.63

Those with  $> 500\mu\text{g}/\text{m}^3$  reduction in second year

0.28 to 5.10

Those with  $< 500\mu\text{g}/\text{m}^3$  reduction in second year

-3.41 to 4.37

# Removing outliers might help (and makes sense if they are asthmatics anyway)

Those with  $> 500\mu\text{g}/\text{m}^3$  reduction in second year

0.28 to 5.10

Those with  $< 500\mu\text{g}/\text{m}^3$  reduction in second year

-3.41 to 4.37

Those with  $< 500\mu\text{g}/\text{m}^3$  reduction minus one outlier

-2.85 to 0.11

(notice this doesn't include 2.16 or the CI for those with higher reductions)

However, removing the one outlier from those with  $> 500\mu\text{g}/\text{m}^3$  reduction

-0.58 to 4.24

(so they now overlap again; the curse of small data sets)