

EVIDENCE BASED PUBLIC HEALTH POLICY AND PRACTICE

Air pollution from biomass burning and asthma hospital admissions in a sugar cane plantation area in Brazil

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Objective: To evaluate the association between the total suspended particles (TSPs) generated from preharvest sugar cane burning and hospital admission due to asthma (asthma hospital admissions) in the city of Araraquara.

Design: An ecological time-series study. Total daily records of asthma hospital admissions (ICD 10th J15) were obtained from one of the main hospitals in Araraquara, São Paulo State, Brazil, from 23 March 2003 to 27 July 2004. The daily concentration of TSP ($\mu\text{g}/\text{m}^3$) was obtained using Handi-vol equipment (Energética, Brazil) placed in downtown Araraquara. The local airport provided the daily mean figures of temperature and humidity. The daily number of asthma hospital admissions was considered as the dependent variable in Poisson's regression models and the daily concentration of TSP was considered the independent variable. The generalised linear model with natural cubic spline was adopted to control for long-time trend. Linear terms were used for weather variables.

Results: TSP had an acute effect on asthma admissions, starting 1 day after TSP concentrations increased and remaining almost unchanged for the next four days. A $10 \mu\text{g}/\text{m}^3$ increase in the 5-day moving average (lag1–5) of TSP concentrations was associated with an increase of 11.6% (95% CI 5.4 to 17.7) in asthma hospital admissions.

Conclusion: Increases in TSP concentrations were definitely associated with asthma hospital admissions in Araraquara and, despite using sugar cane alcohol to reduce air pollution from automotive sources in large Brazilian urban centres, the cities where sugar cane is harvested pay a high toll in terms of public health.

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Epidemiological studies on asthma suggest that its prevalence and severity increase constantly in many countries.^{1,2} In the US, from 1980 to 1994, the prevalence of asthma increased by 75%.³ The tendency to develop asthma can be inherited, but genetic factors alone are unlikely to explain the increases seen over the past 20 years.⁴ Environmental exposure is one of the many proposed reasons for these increases.⁵

In this respect, the most extensively studied environmental factor so far is air pollution, as it can increase the risk of asthma attacks through different mechanisms such as (1) a direct effect on sensitive airways, (2) a toxic effect on the respiratory epithelium, (3) the triggering of bronchial hyper-reactivity, both allergen-specific and non-specific or (4) a change in immune response by increasing susceptibility to an immunological trigger.^{6,7}

An association between increased pulmonary morbidity, including asthma exacerbations, and particulate matter (PM) pollution has been noticed through observational and experimental studies in various countries.^{8,9} A report from the Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society¹⁰ suggests that each $10 \mu\text{g}/\text{m}^3$ increase in the ambient concentration of inhalable particulates is associated with a 3% increase in asthma exacerbations.

Biomass burning is a major contributor to toxic and greenhouse gases, as well as the build-up of suspended particles throughout the world, thus resulting, in most cases, in exposure to high levels of air pollution.¹¹

The world oil crisis of the 1970s led Brazil to look for an alternative, locally produced, renewable fuel source as a

replacement for gasoline. Being the world's biggest producer of sugar cane, ethanol derived from that crop was the obvious choice. Today, 70% of the country's sugar cane is burnt before being harvested manually for ethanol production.

Biomass fuels are seen as cleaner alternatives to oil. The air quality in major Brazilian urban centres has improved since the decision to use ethanol as the automotive fuel was taken.¹² Notwithstanding this, the burning of the sugar cane crop produces large amounts of particles and toxic gases that affect the entire population of the nearby settlements for at least 6 months of every year. A study has shown that burning is the major source of fine and ultrafine particles in sugar cane producing areas during the crop-burning periods.¹³ The World Health Organization¹¹ suggests that vegetation fires produce adverse respiratory health effects, including an increase in the number of emergency room visits and hospitalisation events. Sadly, in the city of Araraquara, with 192 000 inhabitants, sugar cane burning is already associated with an increase in emergency room visits for inhalation therapy.¹⁴

This being so, this study was designed to evaluate the association between total suspended particles (TSPs) generated from the burning of the sugar cane crop and the increase in hospital admissions due to asthma (asthma hospital admissions) in the city of Araraquara.

METHODS

This is an ecological time-series study using secondary health data. Total daily records of asthma hospital admissions (ICD 10th J45) were obtained from one of the main hospitals in the

Abbreviation: TSP, total suspended particles

Table 1 Descriptive analysis of the main variables used in the study

Variables	Mean (SD)	Minimum	Maximum
Asthma (HA)*			
Total	1.3 (1.3)	0	7
Burning*	1.5 (1.4)	0	7
Non-burning	0.96 (1.2)	0	4
TSP ($\mu\text{g}/\text{m}^3$)			
Total	46.8 (26.4)	6.7	137.8
Burning*	56.86 (25.07)	8.1	137.8
Non-burning	28.45 (17.53)	6.7	99.9
Temperature ($^{\circ}\text{C}$)†	16 (3.5)	2	26
Humidity (%)‡	62 (14.5)	20.5	93.3

*Hospital admission (HA); different from non-burning period ($p < 0.001$, Mann-Whitney U test).

†Minimum temperature.

‡Mean relative humidity.

city of Araraquara, São Paulo State, Brazil, between 23 March 2003 and 27 July 2004. Asthma hospitalisation is herein defined as an admission in which asthma was the primary diagnosis.

Of the 493 days considered for the study, sugar cane was burnt on 318 days. Daily TSP concentration ($\mu\text{g}/\text{m}^3$) data were obtained through the use of a Handi-vol sampler (Energética, Brazil), with fibreglass filtering elements, and a certified flow rate of 3 litres/min. The equipment was placed in downtown Araraquara under a canopy to protect it from the rain. Filters were weighed before and after each 24 h period of particle collection. The daily concentration of TSP in micrograms per cubic metre of air was estimated using the following equation:

$$PC = \frac{\Delta M}{TV_{24h}}$$

where PC is the TSP concentration ($\mu\text{g}/\text{m}^3$) under standard conditions of temperature and barometric pressure, ΔM is the mass difference in filter weight (g) before and after use and TV_{24h} is the total volume (m^3) of sampled air during each 24 h interval. The local airport authority provided the daily mean figures for temperature and humidity.

The Mann-Whitney U test was used to compare the mean TSP concentrations and the daily asthma hospital admissions during the burning and non-burning periods. The daily number of asthma hospital admissions was considered as the dependent variable in generalised linear Poisson regression models,¹⁵ and the TSP concentration was considered the independent variable. Natural cubic spline¹⁶ was used to control for long-time trend. We used 5 degrees of freedom to smooth the time trend. The number of degrees of freedom for the natural spline of the time trend was selected to minimise the autocorrelation between the residuals and the Akaike Information Criterion.¹⁷ After adjusting for the time trend, no remaining serial correlation was found in the residuals and therefore the use of autoregressive terms was not necessary.

Araraquara is a warm city, and previous analysis has shown that local temperatures present a linear effect on respiratory diseases.¹⁴ Hence, we decided to use linear terms (lags 0 and 1) to control for the effect of temperature and humidity on asthma hospital admissions. Moreover, an indicator for days of the week was included as an additional means of control for short-term seasonality.

The elapse of time between an increase in air pollution and its effect on health has been analysed using different approaches. This study used the lag structure from 0 to 9 days before hospital admission using a fourth degree polynomially distributed lag

model,¹⁸ which imposes some restraint but allows enough flexibility in the estimation of a biologically plausible lag structure, thereby better controlling multicollinearity than an unconstrained lag model would do. The standard errors of the estimates for each day were adjusted for overdispersion.

Having defined the lag structure for TSP, our models included lag 0 or moving averages to estimate the cumulative effects of the pollutant in the adopted outcome. Also, to assess potential differences in TSP effects between burning and non-burning periods, a model was constructed using an interaction term for period.

To explore the presence of thresholds in TSP effect, additional analyses were conducted using categories (quintiles) for daily concentrations, instead of using a continuous variable. The effect of air pollution was presented as a percentage increase or relative risk and 95% CI in asthma hospital admission owing to a $10 \mu\text{g}/\text{m}^3$ increase in TSP. The statistical analyses were performed in SPSS V.11 and Splus 4.5.

RESULTS

Table 1 shows the descriptive analysis of the variables used in the study for the entire period, and for the burning and non-burning periods. Mean TSP concentration remained below the air quality standard during the burning period and despite this, the mean TSP value was twice that observed during non-burning periods ($p < 0.001$).

Asthma admissions varied substantially between the two periods. There were 640 asthma hospital admissions during the 493 days of the study. As observed for TSP concentrations, during the burning period (318 days) asthma hospital admissions were 50% higher than those observed during the 175 days of the non-burning period (477 and 163 asthma hospital admissions, respectively; $p < 0.001$).

Weather variables showed that Araraquara is located in a region with moderate temperature and very dry days during winter.

Figure 1 shows daily TSP concentrations and asthma hospital admissions within the study period. Both variables increased during the burning period and in 2004, they seem to be more closely correlated than in 2003.

Asthma hospital admissions showed a low but positive and statistically significant correlation with TSP concentrations (Pearson's correlation coefficient: 0.13; $p < 0.001$) but the same was not observed for weather variables.

Figure 2 shows the estimated lag structure for the effect of TSP on total asthma hospital admissions using a polynomial distributed lag model. There was a lagged effect starting one day after TSP concentrations increased, which remained almost unchanged until the fifth day after exposure. Other estimates were done for longer periods (up to 14 days after exposure). However, there was no statistically significant effect after day 5.

Once the lag structure was identified, we used moving averages ranging from 2 to 7 days to estimate the cumulative effects of TSP on asthma hospital admissions (table 2). Because effects were positive from lag 0 to lag 7 (in the polynomial distributed lag models), an increase in the cumulative effect until the 6-day moving average is totally plausible.

The 7day cumulative effect of a $10 \mu\text{g}/\text{m}^3$ increase in TSP concentrations on asthma hospital admissions represented twice the effect observed on the day of the increase and the day after (table 2). When lags 0 and 6 were excluded from initial effect estimates, the cumulative effect from lag 1 to lag 5 reached almost 12%. When the analyses were stratified to non-burning and burning periods, it was observed that for the same variation of $10 \mu\text{g}/\text{m}^3$ in TSP concentration, asthma hospital admissions increased by 9.7% (95% CI 2.6 to 17.2) and 12.7% (95% CI 2.2 to 24.3), respectively.

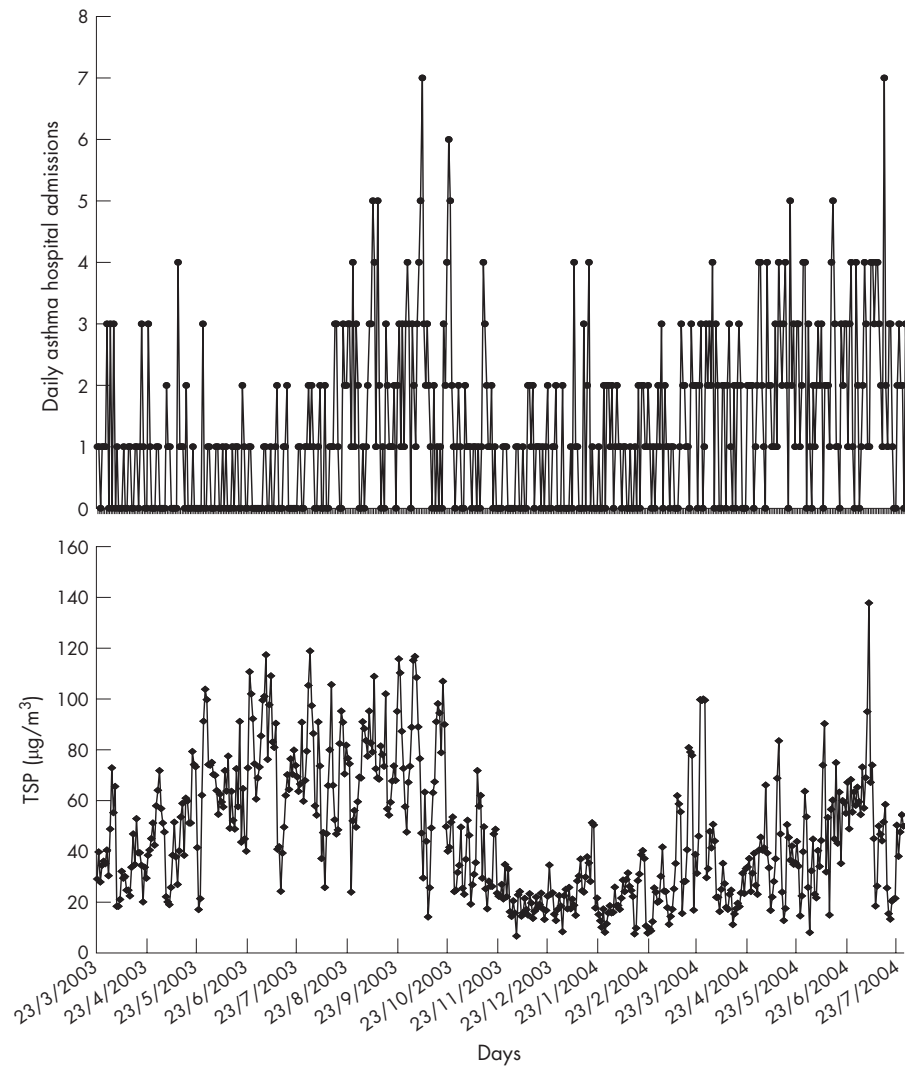


Figure 1 Daily number of asthma hospital admissions and daily concentrations of total suspended particles (TSP).

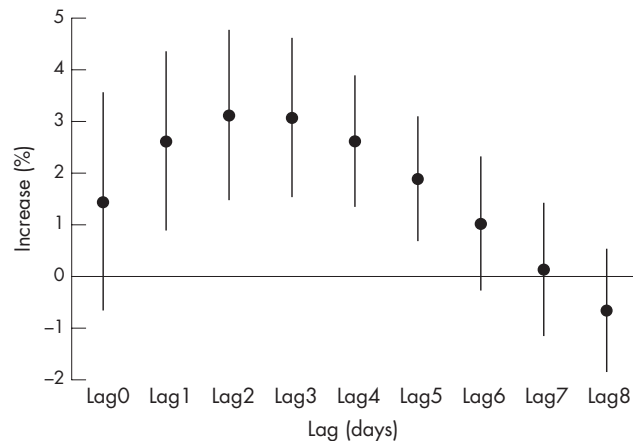


Figure 2 Percentage increases and 95% CI in asthma hospital admissions in the concurrent and six subsequent days following a 10 µg/m³ increase in total suspended particle concentrations.

Models including categories (quintiles) of TSP moving averages with different ranges instead of continuous variables were also considered. Table 3 shows the results for the quintiles of the 5-day moving average (lag 1–lag 5).

As observed for the moving averages actually adopted in the study (tables 1 and 2), as well as for the one used in table 3 (5-day moving average one day lagged), the relative risk of asthma admissions had a dose–response relationship with the pollutant, without any trace of threshold.

Table 2 Percentage increases and 95% CIs for asthma hospital admissions using moving averages of total suspended particle concentrations

TSP moving average	Percentage increase (95% CI)
2-day (lag0–lag1)	6.96 (1.4 to 12.86)
3-day (lag0–lag2)	9.09 (3.12 to 15.40)
4-day (lag0–lag3)	10.28 (4.05 to 16.90)
5-day (lag0–lag4)	11.63 (5.46 to 19.18)
5-day (lag1–lag5)	11.63 (5.42 to 17.73)
6-day (lag0–lag5)	12.61 (5.68 to 20.00)
7-day (lag0–lag6)	12.56 (5.47 to 20.13)

TSP, total suspended particles.

Table 3 Relative risk (RR) and 95% CIs of the total suspended particles 5-day moving average (lag 1–lag 5) quintiles

TSP moving average	RR (95% CI)
5-day (lag1–lag5)($\mu\text{g}/\text{m}^3$)	
9.25–28.45	1.00
28.46–48.85	1.55 (0.45 to 5.77)
48.86–69.06	2.46 (1.08 to 5.60)
69.07–88.44	2.77 (1.32 to 5.84)
88.45–108.91	2.94 (1.48 to 5.85)

TSP, total suspended particles.

DISCUSSION

This study shows that in a region with characteristics similar to those of Araraquara, where periodic preharvest biomass burning is common practice, TSP concentrations and asthma hospital admissions are very likely to be higher during the burning periods. Moreover, there is a statistically significant association between asthma hospital admissions and TSP concentrations. Also, the effect was acute, occurred 1 day after the increase in TSP concentrations, and remained for almost a week. These results show almost the same lag structure seen for respiratory diseases in our other studies.^{14 19 20}

The all-ages approach used in this study, as opposed to stratified age group analysis of asthma hospital admissions, was taken because, as shown in the descriptive analysis above, these are not common events and stratifying by age could possibly decrease, distort or even hide the true picture. Instead, working with the whole spectrum of patients with asthma may provide a much closer estimate of the effect of TSP generated from burning sugar cane in a specific community.

Health data were collected from the city's main hospital and the precision of asthma diagnosis was assured by its permanent, well-trained staff. Moreover, the diagnosis of all patients admitted to hospital is reviewed by a senior physician. In Brazil, respiratory diseases, including asthma, are more prevalent throughout the country during autumn and winter, both in large urban centres where air pollution is generated mainly by fossil fuel combustion, and in areas where the main source of pollutants is biomass burning. Weather and seasonality have been recognised as confounders of the association between air pollution and respiratory disease, and effective control for these confounding factors has been matter of concern among environmental epidemiologists. This study uses different sets of controls for weather and season, which are the standard approaches and have been extensively used by other authors.^{14 19 21} In a recent study²⁰ we used the method of stratification by burning/non-burning periods. In it, we found a remarkable difference in the total number of hospital admission for respiratory ailments in each of these periods. In the present study, we used an interaction term for period and the results showed at least a 30% greater effect during the burning period.

Our statistical analyses comprised a widely adopted time-series method that is robust and less sensitive to effect estimate bias.^{19 20} Some authors have recently chosen to use case-crossover analysis to estimate the acute effects of air pollution. Results, however, have shown that the appropriate use of either case-crossover or time-series techniques produces similar results for estimates.²² Hence, the adoption of generalised linear models in our study cannot be considered a weakness.

Some recent studies on the adverse health effects derived from particulate matter have looked at fine instead of coarse particle or TSP data. This was not possible in this study as the region has no monitoring stations and PM_{10} measurements are rarely conducted by the São Paulo State Environmental Agency

(CETESB) and, when conducted, are of short duration. To be adequately performed, a time-series analysis requires a long period of regular data collection for both exposure and outcome. This being so, we considered that using daily TSP concentrations instead would be a better choice. Moreover, previous studies have shown that most particles emitted during biomass burning fall into the ultrafine category ($<0.1 \mu\text{m}$).¹¹ The relationship between particulate matter with diameter $<10 \mu\text{m}$ and total particulate emission in agricultural burning emissions is approximately 90%.²³ Ward *et al*²⁴ found that total particulate matter emitted by flaming-combustion forest fires is composed of 80–95% fine particles ($\text{PM}_{2.5}$). Therefore it is reasonable to assume that daily TSP variations can be used as a proxy of daily variation of fine and ultrafine particles, which are widely recognised as inducing damage in the airways of patients with asthma.⁵

Several toxicological studies have shown that particles can cause inflammatory reactions both in vivo and in vitro. Particles collected directly from the natural environment cause inflammatory reactions in rat lungs, rat cell lines and human cell lines.^{25–27} Free radical activity or the oxidative capacity of particulate matter plays an essential role in provoking these inflammatory responses. It has also been suggested that the inflammatory properties of ultrafine particles are mediated by their large number, small size and high penetration rate into the interstitium, independently of their chemical composition.²⁸

Patients with pre-existing respiratory or cardiac illnesses seem to be at particular risk of the most severe adverse health effects on exposure to inhalable particles, including those with asthma. Previous studies from several countries have shown a relationship between hospital admissions for asthma and fossil-fuel-generated particulate matter, independently of the size of the particles and the age of the population.^{29–36} Although there are very few epidemiological studies on this subject, it is possible to subdivide them into three groups: (1) residential wood burning, (2) agricultural residuals and (3) forest fires. Lipsett *et al*³⁷ working in Santa Clara County showed a 6% increase in asthma-related emergency room visits after increases of $10 \mu\text{g}/\text{m}^3$ in ambient wintertime PM_{10} . In this case the main source of PM_{10} was residential wood smoke. In 1993, wood burning was found to be the dominant source of PM_{10} pollution in Seattle, ranging from 60% in summer to 90% in winter. Schwartz *et al*³⁸ found a 3.7% increase in emergency room visits for every $10 \mu\text{g}/\text{m}^3$ rise in PM_{10} , and the positive association remained for an average lag of 0–4 days, suggesting a delayed response. Norris *et al*,³⁹ again in Seattle, found that for every $11 \mu\text{g}/\text{m}^3$ increase in PM_{10} , there was a 1.15% increase in the relative risk of asthma-related emergency room visits. Also in Seattle, Sheppard *et al*⁴⁰ showed an association among PM , PM_{10} and $\text{PM}_{2.5}$, and asthma hospital admissions in patients aged <65 years, with a 1 day lag.

Long *et al*⁴¹ evaluated 428 subjects surveyed for their respiratory symptoms following exposure to combustion emissions from agricultural burning (straw and stubble). During the 24 h period of exposure, average PM_{10} concentrations increased from $15\text{--}40 \mu\text{g}/\text{m}^3$ to $80\text{--}110 \mu\text{g}/\text{m}^3$; 63% of those surveyed experienced worsening symptoms owing to the air pollution. Subjects with asthma and chronic bronchitis were most affected.

Forest fires are usually brief events in which particulate matter reaches extremely high concentrations and a concomitant increase in asthma-related emergency room visits has been observed.^{42 43} Occasionally, forest fires are long lasting such as the one in Southeast Asia, which lasted from July to October 1997. During this period asthma-related emergency room visits and hospital admissions in many countries in the region were higher than usual.¹¹ A study performed at that time on young

What is already known

- Indoor biomass burning is strongly associated with high exposure to particulate matter and this situation leads to increases in adverse health effects.
- Episodic vegetation fire is also associated with adverse health effects.
- Some regions face regular episodes of biomass burning due to agricultural activities and with lower concentration of particulate matter. Sugar cane preharvest burning was already associated with increases in emergency room visits and hospital admissions, mainly in elderly and children.

What this paper adds

- This is one of the few available manuscripts analysing the adverse health effects of regular biomass burning in communities surrounded by sugar cane plantations.
- Most of the existing studies on biomass burning deal just with episodic events in which there was a very high concentration of particulate matter.
- This study shows that, in urban centres, the effects of particles produced by burning biomass rival those produced by the burning of fossil fuel.

Policy implications

- The population and authorities have devoted much more of their attention to analysing the effects of air pollution generated from fossil fuel combustion than evaluating air pollution caused by biomass burning, which they have associated mainly with wood burning or episodic events such as forest fires.
- If alcohol is to be considered an alternative to fossil fuel, it is important to show that cities surrounded by sugar cane plantations where preharvest burning is routine practice have been paying an extremely high price in terms of adverse public health.
- We also recommend that mechanised harvesting is introduced as soon as possible and that biomass burning is banned to balance better the advantages and disadvantages of producing fuel alcohol.

and healthy military recruits in Singapore showed an association between air pollution and the increase in peripheral blood cell counts,⁴⁴ linking this specific kind of air pollution with systemic inflammatory responses.

Johnston *et al*⁵⁵ conducted a time-series ecological study in Darwin, Australia, during a bushfire (7 months) in 2000, and showed a significant increase in asthma hospital admissions owing to increases in PM₁₀ concentrations. However, the effect was not linear, only occurring on days with PM₁₀ concentrations above 40 µg/m³.

All of these studies reported episodic events showing adverse respiratory effects associated with relatively brief haze episodes. In the cities surrounded by the sugar cane plantations, exposure to air pollution generated by biomass burning is continuous for at least 6 months each year but particle concentrations are lower, and the effects were very similar to

those observed in other regions with episodic events. Analysis of specific toxicity or of the elemental composition of the particles is required to shed more light on the size of the effect.

The main concern about alcohol production is the need to burn the sugar cane before manual harvesting, a process that reduces the cost of production. To minimise adverse effects from this pollution, researchers and doctors have recommended that the population avoid overexposure during burning periods and restrict physical activity. The weather forecast also provides information on weather quality and pollutant dispersion, allowing people to plan their activities. São Paulo state is gradually implementing mechanised harvesting, with the aim to be fully mechanised by 2031.⁴⁶ However, we have concerns about the length of time required to mechanise given the adverse health effects of the burning process.

Biomass burning is not just a Brazilian problem and has been a common practice in other countries. In Brazil, however, the problem is more acute as the country is the biggest producer of sugar and alcohol in the world. The problems faced in Brazil from this specific kind of air pollution may help to consolidate the relevance of biomass burning as a public health issue, leading to more restrictive policies in Brazil and other countries planning to implement alcohol-based fuel programmes.

In summary, increased TSP concentrations were associated with asthma hospital admissions in Araraquara and, despite the benefits of alcohol produced from sugar cane in reducing air pollution from automotive sources in large Brazilian urban centres, the cities where sugar cane is harvested have paid a high price in terms of public health. We recommend the banning of preharvest sugar cane burning to balance better the advantages and disadvantages of producing fuel alcohol.

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REFERENCES

- 1 **International Study of Asthma and Allergies in Childhood (ISAAC) Steering Committee.** Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. *Lancet* 1998;**351**:1225–32.
- 2 **Magnus P, Jaakkola JJK.** Secular trend in the occurrence of asthma among children and young adults: critical appraisal of repeated cross sectional surveys. *BMJ* 1997;**314**:1795–9.
- 3 **Centers for Disease Control and Prevention.** Surveillance for asthma—United States, 1960–1995. *CDC Surveill Summ* 1998;**47**:1–28.
- 4 **National Academy of Sciences.** *Clearing the air: asthma and indoor air exposures.* Washington DC: National Academy Press, 2000.
- 5 **Etzel RA.** How environmental exposures influence the development and exacerbation of asthma. *Pediatrics* 2003;**112**:233–9.
- 6 **Wardlaw AJ.** The role of air pollution and asthma. *Clin Exp Allergy* 1993;**23**:81–96.
- 7 **Koenig JQ.** Air pollution and asthma. *J Allergy Clin Immunol* 1999;**104**:717–22.

- 8 **Samet JM**, Zeger SL, Dominici F, et al. The national morbidity, mortality and air pollution study. Part II: morbidity and mortality from air pollution in the United States. *Res Health Eff Inst* 2000;**94**:5–70.
- 9 **Goldsmith CW**, Kobzik L. Particulate air pollution and asthma: a review of epidemiological and biological studies. *Rev Environ Health* 1999;**14**:121–34.
- 10 **Committee of Environmental and Occupational Health Assembly of the American Thoracic Society**. Health effects of outdoor air pollution. *Am J Respir Crit Care Med* 1996;**153**:3–50.
- 11 **Schwela DH**, Goldammer JG, Morawska LH, et al. *Health guidelines for vegetation fire events—guideline document*. Geneva: WHO, 1999.
- 12 **Massad E**, Saldiva PHN, Saldiva, CD, et al. Toxicity of prolonged exposure to ethanol and gasoline auto engine exhausts gases. *Environ Res* 1986;**40**:479–86.
- 13 **Lara LBLS**, Artaxo P, Martinelli LA, et al. Chemical composition of rainwater and anthropogenic influences in the Piracicaba river basin. *Atm Environ* 2001;**35**:4937–45.
- 14 **Arbex MA**, Bohm GM, Saldiva PHN, et al. Assessment of the effects of sugar cane plantation burning on daily counts of inhalation therapy. *J Air Waste Manage Assoc* 2000;**50**:1745–9.
- 15 **McCullagh P**, Nelder JA. *Generalized linear models*, 2nd edition. London: Chapman and Hall, 1989.
- 16 **Green PJ**, Silverman BW. *Non parametric regression and generalized linear models, A roughness penalty approach*. London: Chapman and Hall, 1994.
- 17 **Akaike H**. Information theory and an extension of the maximum likelihood principal. In: Petrov BN, Csaki F, eds. *2nd International Symposium on Information Theory*. Budapest: Akademiai Kiado, 1973:267–81.
- 18 **Schwartz J**. The distributed lag between air pollution and daily deaths. *Epidemiology* 2000;**11**:320–6.
- 19 **Martins MCH**, Fatigati FL, Véspoli TC, et al. Influence of socioeconomic conditions on air pollution adverse health effects in elderly people: an analysis of six regions in Sao Paulo, Brazil. *J Epidemiol Comm Health* 2004;**58**:41–6.
- 20 **Cançado JED**, Saldiva PHN, Pereira LAA, et al. The impact of sugar cane burning emissions on the respiratory system of children and elderly. *Environ Health Perspect* 2006;**114**:725–9.
- 21 **Lin CA**, Pereira LAA, Conceição GMS, et al. Association between air pollution and ischemic cardiovascular emergency room visits. *Environ Research* 2003;**92**:57–63.
- 22 **Lin M**, Chen Y, Burnett RT, et al. The influence of ambient coarse particulate matter on asthma hospitalization in children: case-crossover and time-series analyses. *Environ Health Perspect* 2002;**110**:575–81.
- 23 **US EPA**. Air quality criteria for particulate matter (October 2004). http://oaspub.epa.gov/eims/eimscomm.getfile?p_download_id=435945 (accessed 4 February 2007).
- 24 **Ward D**. Review of smoke components in United States Department of Agriculture (USDA): health hazards of smoke. Recommendations of the Consensus Conference. Missoula, 1997.
- 25 **Vicent R**, Bjarnason SG, Adamson IYR, et al. Acute pulmonary toxicity of urban particulate matter and ozone. *Am J Pathol* 1997;**151**:1563–70.
- 26 **Becker S**, Soukup JM, Gilmour MI, et al. Stimulation of human and rat alveolar macrophages by urban air particulates: effects on oxidant radical generation and cytokine production. *Toxicol Appl Pharmacol* 1996;**141**:637–48.
- 27 **Carter JD**, Ghio AJ, Samet JM, et al. Cytokine production by human airway epithelial cells after exposures to an air pollution particle is metal dependent. *Toxicol Appl Pharmacol* 1997;**146**:180–8.
- 28 **Hosiokangas J**, Kikas U, Pekkanen J, et al. Identifying an quantifying air pollution sources in Kuopio by receptor modeling. *J Aerosol Sci* 1995;**26**:S423–4.
- 29 **Bates DV**, Sizto R. Hospital admissions and air pollutants in southern hospital Ontario: the summer acid haze effect. *Environ Res* 1987;**43**:317–31.
- 30 **Pope CA III**. Respiratory disease associated with community air pollution and a steel mill, Utah Valley. *Am J Public Health* 1986;**79**:623–8.
- 31 **Pope CA III**. Respiratory hospital admissions with PM₁₀ pollution in Utah, Salt Lake and Cache Valleys. *Arch Environ Health* 1991;**46**:90–7.
- 32 **Atkinson RW**, Anderson HR, Sunyer J, et al. Acute effect of particulate air pollution on respiratory admissions. Results of APHEA 2 project. *Am J Respir Crit Care Med* 2001;**164**:1860–6.
- 33 **Anderson HR**, Ponce de Leon A, Bland JM, et al. Air pollution, pollens, and daily admissions for asthma in London 1987–92. *Thorax* 1998;**53**:842–8.
- 34 **Tseng RY**, Li CK, Spinks JA. Particulate air pollution and hospitalization of asthma. *Ann Allergy* 1992;**68**:425–32.
- 35 **Pönkä A**. Asthma and low level air pollution in Helsinki. *Arch Environ Health* 1991;**46**:262–70.
- 36 **Migliaretti G**, Cadum E, Migliore E, et al. Traffic air pollution and hospital admission for asthma: a case control approach in a Turin (Italy) population. *Int Arch Occup Environ Health* 2005;**78**:164–9.
- 37 **Lipsett M**, Hurley S, Ostro B. Air pollution and emergency room visits for asthma in Santa Clara County, California. *Environ Health Perspect* 1997;**105**:216–22.
- 38 **Schwartz J**, Slater D, Larson TV, et al. Particulate air pollution and hospital emergency room visits for asthma in Seattle. *Am Rev Respir Dis* 1993;**147**:826–31.
- 39 **Norris G**, YoungPong SN, Koenig JQ, et al. An association between fine particles and asthma emergency department visit for children in Seattle. *Environ Health Perspect* 1999;**107**:389–93.
- 40 **Sheppard L**, Levy D, Norris G, et al. Effects of ambient air pollution on non-elderly asthma hospital admissions in Seattle, 1987–1994. *Epidemiology* 1999;**10**:23–30.
- 41 **Long W**, Tate RB, Neuman M, et al. Respiratory symptoms in a susceptible population due to burning of agricultural residue. *Chest* 1998;**113**:351–7.
- 42 **Chew FT**, Ooi BC, Hui JK, et al. Singapore's haze and acute asthma in children. *Lancet* 1995;**346**:1427.
- 43 **Duclos P**, Sanderson LM, Lipsett M. The 1987 forest fire disaster in California: assessment of emergency room visits. *Arch Environ Health* 1990;**45**:53–8.
- 44 **Tan CW**, Qiu D, Lian BL, et al. The human bone marrow response to acute air pollution caused by forest fire. *Am J Respir Crit Care Med* 2000;**161**:1213–17.
- 45 **Johnston FH**, Kavanagh AM, Bowman DMJS, et al. Exposure to bushfire smoke and asthma: an ecological study. *The Med J Aust* 2002;**176**:536–8.
- 46 **State of São Paulo**. State law N° 11241 on gradual ban of sugar cane burning from 19 September 2002 (In Portuguese). www.cetesb.sp.gov.br/licenciamentoo/legislacao/estadual/leis/2002_Lei_Est_11241.pdf (accessed 12 Jan 2007).