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ORIGINAL ARTICLE

Risk factors for reduced lung function in Australian Aboriginal children

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Aim: To determine the influence of perinatal and childhood exposures on lung function in a cohort of Australian Aboriginal children.

Methods: This was a cross-sectional study of 547 Northern Territory Aboriginal children, aged 8–14 years, belonging to a birth cohort. Assessment included physical examination and spirometry as well as retrospective review of centralised hospital records. The effect of select perinatal and childhood exposures on lung function outcomes (forced expiratory volume in 1 s (FEV1), forced vital capacity (FVC) and forced expiratory flow between 25 and 75 s (FEF25–75)) adjusted for age, sex, height and other measures of size was examined using multiple regression.

Results: Non-urban residence (FEV1 –5% (95% confidence interval, CI 0.91–0.99), FVC –9% (95% CI 0.87–0.95)), current cough (FEV1 –6% (95% CI 0.91–0.97), FVC –4% (95% CI 0.93–0.97), FEF25–75 –8% (95% CI 0.86–0.98)) and hospitalisations for respiratory disease (FEV1 –10% (95% CI 0.86–0.95), FEF25–75 –12% (95% CI 0.70–0.87)) all had significant negative effects on adjusted lung function measures. Children with a non-Aboriginal ancestor had significantly better lung function. No perinatal exposure other than neonatal lung disease had any significant effect on adjusted lung function.

Conclusions: For Northern Territory Aboriginal children factors related to the childhood environment are more important than perinatal factors in determining childhood lung function.

Key words: Aboriginal; child; lung function; risk factor; spirometry.

Respiratory disease is an important cause of morbidity and mortality among Australian Aborigines. Compared with non-Aboriginal children, Aboriginal children in Alice Springs were admitted to hospital 70 times more frequently for pneumonia and, in Western Australia, they were six times more likely to have a discharge diagnosis of bronchiolitis.[1] In the Northern Territory (NT) overall respiratory mortality for Aboriginal adults and children is at least five times that for non-Aborigines and respiratory illness is the commonest reason for hospitalisation in children under 5 years.[2]

Although there are six published studies of lung function in Aboriginal children[3–8] the factors that influence lung function in this population, and their effect on long-term respiratory health, are largely unknown.

We performed a cross-sectional examination of lung function in a cohort of Aboriginal children in late childhood with the aim of determining the influence of various perinatal and childhood exposures on spirometry outcomes.

Key Points

1. Northern Territory Aboriginal children have high rates of hospitalisation for respiratory disease, particularly pneumonia.
2. Northern Territory Aboriginal children experience significant reductions in lung function that are not explained by the usual biological predictors of size, age and gender.
3. Factors relating to the childhood environment, in particular, poor living conditions associated with non-urban dwelling, a history of respiratory hospitalisations and inter-current cough are more important than antenatal and perinatal factors in affecting lung function in Northern Territory Aboriginal children in late childhood.

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Methods

Subjects

The subjects were Aboriginal children, aged 8–14 years, belonging to a birth cohort. The recruitment and follow-up of this cohort has been previously published.[9,10] Briefly, 686 live-born singleton babies delivered at Royal Darwin Hospital (RDH) were recruited at birth, between January 1987 and March 1990. Seventy per cent were routine deliveries from the Darwin Health Region (120 000 km^2 of the 'Top End' of the NT) and 30% were high-risk in utero referrals from adjacent areas. At follow-up between December 1998 and June 2001, 547 of the 686 children (80%) underwent respiratory assessment. Thirty-one children were unable to be traced, 18 had died (9

owing to preterm birth), 64 were traced but unable to be examined because of weather and access difficulties on the day of assessment and 26 were examined but did not have a respiratory assessment due to unavailability of the investigator.[9] In this study sample, 10% of the children were preterm, 25% small for gestational age (birthweight <10th percentile for gestational age (SGA10) and sex using an Australian reference standard[11]) and 17% low birthweight (birthweight <2.5 kg).

Procedure

Children were seen at schools, hospitals, health centres and private homes in over 70 locations throughout the NT, north-western and south Australia. Assessment included physical examination, spirometry and hospital record review. Current residence was classified as urban (a suburban situation), remote (a rural community with an Aboriginal council) or other (included other communities and camps).[12]

Physical examination

Height to the nearest centimetre was measured with a portable wall-mounted stadiometer. Weight, with light clothing and no shoes, was recorded to the nearest 0.1 kilogram on digital electronic (Tanita model 5810) scales. A paediatrician (IB) assessed each child's pubertal status and noted the presence of skin sores, purulent nasal discharge or discharging ears. Triceps and subscapular skinfold were measured to the nearest millimetre using a Harpenden calliper. Children were recorded as having a 'current cough' if they were able to cough (productive or otherwise) on demand or if a cough was heard during testing. They were examined for digital clubbing and auscultation was performed at lung bases.

Spirometry

All spirometry was conducted by one paediatrician (IB) using a Vitalograph 2120 hand-held closed system attached to a computer with a visual incentive and display of the manoeuvres. Children performed at least three blows with encouragement to exhale for as long as possible and/or until a plateau was seen on the volume – time curve. American Thoracic Society (ATS) criteria were used to determine reproducibility and acceptability of tests,[12] however, a minimum exhalation of 3 s was used as most children were unable to exhale for 6 s (ATS end-of-test criterion).[13] Three spirometric outcomes were recorded: forced expiratory volume in 1 s (FEV1), forced vital capacity (FVC) and forced expiratory flow between 25 and 75 s (FEF25–75). The highest FEV1 and FVC were selected from all acceptable, but not necessarily the same, blows.[14] FEF25–75 was taken from the blow with the highest sum of FVC and FEV1. The spirometer was recalibrated with a 1 L portable calibration syringe every 4 h and whenever environmental temperature varied by more than 2 degrees Celcius. Barometric pressure was not recorded.

Medical record review

Medical records were examined from RDH, Katherine and Gove hospitals, the only two regional hospitals in the Top End. Information was recorded about neonatal lung disease (NNLD), admissions for pneumonia, bronchiolitis, asthma, malnutrition and cardiac disease; all diagnoses were cross-checked against clinical notes and radiology reports. NNLD was defined as 'significant' if the baby required oxygen for 2 days or more. This captured all babies who required any mechanical respiratory support. Malnutrition was defined as weight-for-age <80% of the median using the 1978 CDC/WHO growth reference curves. Information about maternal smoking and any non-Aboriginal maternal ancestor was collected at the time of subject recruitment.[9]

The Joint Institutional Ethics Committee of RDH and the Menzies School of Health Research, including the Aboriginal Ethical subcommittee, approved the study. Written consent was obtained from the care givers of all children

Statistical methods

Perinatal exposures examined included birthweight, gestation, birthweight below the 10th percentile (SGA10), maternal smoking, significant NNLD and non-Aboriginal ancestry. Childhood exposures included place of residence, current cough, onset of puberty, hospitalisations for respiratory illness and for malnutrition, and a composite variable encompassing ear, nose and skin infections (ENS).

Gestation, birthweight (in 500 g increments) and age were treated as continuous variables. All other exposures were dichotomous. Pubertal status was classified as pre-pubertal or commenced puberty, maternal smoking was classified as non-smoker or smoker, and the ENS variable was classified as positive if 1 or more sites of infection were present.

All non-normal continuous data (FEV1, FVC, FEF25–75 and some measures of body size) were transformed to the natural logarithm (ln) to yield a normal distribution. Stepwise modelling with linear regression was used to examine perinatal and childhood exposures that were independently associated with FEV1, FVC and FEF25–75 after adjusting for age, sex and measures of size (height, ln weight, ln triceps and subscapular skinfolds). The regression results are expressed as per cent differences in lung function for exposed children compared with unexposed children (i.e. the exponential of the regression coefficients minus 100%). Data analysis was undertaken using STATA 7.0.[15]

Results

Respiratory assessment was undertaken on 547 children; 259 girls and 288 boys, aged 8.9–13.8 years. Characteristics of children at recruitment and follow-up are shown in Table 1.

Ability to perform spirometry

A total of 417 of the 547 children (76%) successfully performed spirometry according to ATS criteria and exhaled for at least 3 s. Only 54 children sustained expiration for 6 s. Children whose tests were excluded were lighter, at birth and at testing, and were less likely to live in an urban setting.

Hospitalisation history and physical examination

Only 21 children had significant NNLD and only 9 were ventilated. Twenty-five per cent of children had at least one respiratory hospitalisation, almost 75% of these for pneumonia, but only 5% of children were admitted for asthma (Fig. 1). Seventeen children had

Table 1 Characteristics of children seen in the study

Characteristics	Total ($n = 547$)	Girls ($n = 259$)	Boys ($n = 288$)
	Mean (SD)	Mean (SD)	Mean (SD)
Birthweight (g)	3043 (636)	2964 (597)	3115 (662)*
Gestational age (weeks)	38.8 (2)	38.8 (1.8)	38.9 (1.8)
Current age (years)	11.6 (1.2)	11.5 (1.2)	11.7 (1.1)
Height adjusted for age	–0.49 (1.1)	–0.47 (1.1)	–0.50 (1)
Weight adjusted for age	–0.61 (1.3)	–0.55 (1.3)	–0.66 (1.3)
Height (cm)	144.3 (10.8)	144.5 (11.0)	144.0 (10.5)
	Median (IQR)	Median (IQR)	Median (IQR)
Weight (kg)	33.1 (27.1–42.1)	34.1 (27.1–42.9)	32.1 (27.9–40)
FEV1 (L)	1.9 (1.6–2.2)	1.8 (1.5–2.2)	1.9 (1.6–2.2)
FVC (L)	2.2 (1.8–2.6)	2.1 (1.7–2.5)	2.2 (1.9–2.6)*
FEF25–75 (L/s)	2.3 (1.8–2.7)	2.3 (1.9–2.9)	2.2 (1.8–2.7)
	n (%)	*n* (%)	*n* (%)
Commenced puberty	257 (51)	155 (64)	102 (38)*
Live in remote community	395 (72)	194 (75)	201 (70)
Small for gestational age	138 (25)	63 (24)	25 (26)
Non-Aboriginal maternal ancestor	98 (18)	42 (16)	56 (20)

*$P < 0.05$. FEF25–75, forced expiratory flow between 25 and 75 s; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity; IQR, interquartile range; SD, standard deviation.

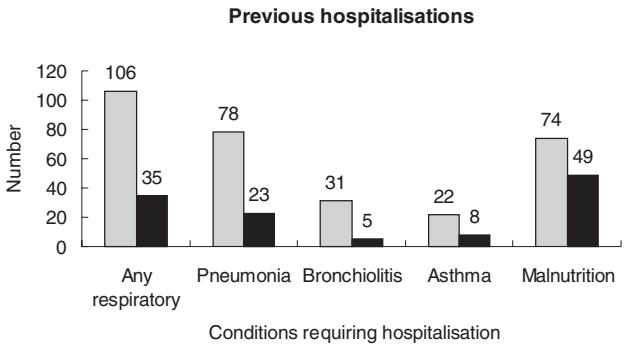

Fig. 1 Respiratory and malnutrition hospitalisation history of children in the study. (□) One hospitalisation; (■) two or more hospitalisations.

congenital or rheumatic heart disease. On examination 122 (22%) children had a cough (six with wheeze) but only 18 children had inspiratory crepitations and/or rhonchi. Two children with cardiac disease were clubbed.

Predictors of lung function

As expected, height, age and sex were significant predictors ($P < 0.05$) of all lung function measures. Together they explained over 70% of the variance of FEV1 and FVC. Other statistically significant size standardising terms (weight and skinfolds) explained less than an additional 1% of the variance each.

After adjusting FEV1, FVC and FEF25–75 for age, sex and body size all perinatal and childhood exposures except SGA10, puberty, maternal smoking and ENS were significantly associated with at least one of these outcomes. The effect of each of these significant exposures on the adjusted lung function outcomes, in the presence of the other significant independent predictors, is shown in Table 2.

Compared with urban children, non-urban children had a significant reduction in FVC and FEV1. Cough was associated with a reduction of all lung function measures and children with at least two respiratory hospitalisations had significant reduction in FEV1 and FEF25–75. When admissions for asthma, bronchiolitis and pneumonia were analysed separately, children hospitalised for asthma had significant reductions in FEV1 (0.92, 95% confidence interval, CI 0.87–0.96) and FEF25–75 (0.80, 95% CI 0.71–0.89) and children hospitalised for pneumonia had reduced FEV1 (0.96, 95% CI 0.93–0.99) and FVC (0.96, 95% CI 0.93–0.99). Those hospitalised for bronchiolitis had a small reduction in FEF25–75 (0.91, 95% CI 0.83–0.99) only. Of the perinatal exposures, only NNLD and non-Aboriginal maternal ancestry had any significant impact on any of the adjusted lung function outcomes.

Discussion

This is the largest study of respiratory health in Australian Aboriginal children and the first to examine the effect of multiple risk factors for reduced childhood lung function in any Indigenous population.

Ethnicity is a known biological determinant of lung function but the mechanism for its effect remains poorly understood; adjusting for body measurements inconsistently and incompletely accounts for the differences.[16] Although our study was confined to Aboriginal children, those whose mother had a non-Aboriginal ancestor had significantly better lung function even after adjusting for size. In this context mixed maternal ancestry may be a partial surrogate for better living conditions as including the ancestry variable into the regression model attenuated the association between non-urban residence and poorer lung function outcome.

Table 2 Per cent differences in adjusted lung function for exposed children compared to unexposed children

	FEV1 % (95% CI)	FVC % (95% CI)	FEF25–75 (95% CI)
Place of residence			
Rural	0.95* (0.91–0.99)	0.91** (0.87–0.95)	
Other	0.96 (0.91–1.01)	0.93* (0.89–0.98)	
Birthweight (500 g)	1.01 (0.99–1.02)		1.02 (0.99–1.05)
Gestational age			1.00 (0.98–1.02)
Presence of cough			
Cough	0.94** (0.91–0.97)	0.96** (0.93–0.97)	0.92** (0.86–0.98)
Respiratory hospitalisations			
One	0.97 (0.93–1.00)	0.97 (0.94–1.00)	0.96 (0.89–1.03)
Two or more	0.90** (0.86–0.95)	0.96 (0.91–1.01)	0.78** (0.70–0.87)
Malnutrition hospitalisations			
One	0.99 (0.95–1.03)	0.98 (0.94–1.01)	
Two or more	0.97 (0.92–1.02)	0.96 (0.92–1.00)	
Neonatal lung disease			
Diseased	0.92* (0.84–0.99)		0.86 (0.72–1.03)
Maternal ancestry			
Non-Aboriginal ancestor	1.06** (1.01–1.10)	1.02 (0.97–1.06)	1.12** (1.03–1.21)

*$P < 0.05$; **$P < 0.01$. Lung function outcomes (FEV1, FVC, FEF25–75) are adjusted for age, sex, height, weight, skinfolds and independent predictors of lung function outcomes. Data are shown only where the exposure was significant for the particular lung function outcome in the crude analysis. CI, confidence interval; FEF25–75, forced expiratory flow between 25 and 75 s; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity.

Poor living environments have been highlighted as key to respiratory ill-health among Australian Aborigines.[17,18] In our study non-urban residence was associated with a reduction in FVC of up to 9% and a lesser reduction in FEV1. Although non-urban children were shorter, lighter, thinner and had higher rates of superficial infection,[12] differences in lung function remained even after controlling for these factors. These rural–urban differences may be confounded by mobility, but the majority of children living in rural and remote areas at birth were still in those areas at follow-up.[12] Other features of non-urban Aboriginal communities including poor hygiene and sanitation, smoke exposure, overcrowding and low socio-economic class were not examined. Any of these may explain our finding as each can affect respiratory health both in the short- and long-term.

The children in our study with cough had significant reductions in all three spirometry outcomes, consistent with the reduction in FEV1/FVC found in Aboriginal children with cough in a study in Western Australia.[4] We were unable to separate children with an acute cough from those with chronic cough as the young, frequently unaccompanied, children were unable to reliably report their medical histories and the information was otherwise unavailable. It is conceivable that the reduction in lung function seen in some of these children may only reflect temporary lung dysfunction owing to acute infection, however, chronic cough is common in apparently well Aboriginal children[4,17] and may be the only clinical indication of chronic suppurative lung disease.[17]

Previous studies have documented the high rate of hospitalisations of Aboriginal children with respiratory illness[1,19] but none have examined the relationship between hospitalisations and spirometry. By 8–14 years of age almost 20% of children from the cohort had been hospitalised at least once for respiratory illness, the majority of admissions occurring before the children were 2 years of age. Admissions with pneumonia were four times more common than admissions for asthma or bronchiolitis. Children with at least two respiratory hospitalisations had significantly reduced FEV1 and FEF25–75 and a non-significant reduction in FVC. Numerous studies in non-Indigenous populations have demonstrated an association between early childhood respiratory infections and abnormal lung function in both child and adulthood.[20] Similar to our study, the deficit in FEV1 ranges from 3% to 16% but is generally less than 10%.[21] In our cohort the trend for reduction in lung function owing to asthma hospitalisations was greater than those for pneumonia hospitalisations. This was borne out by the 'obstructive pattern' of the reduction, namely reduction in FEV1 and FEF25-75, without significant reduction in FVC. Despite the apparent greater effect of asthma hospitalisations on lung function, asthma is relatively uncommon among NT Aboriginal children[22] and few children in our study had clinical signs of asthma.

There was no relationship between either birthweight or intrauterine growth retardation (SGA 10) and lung function in our study, consistent with the only other study of Aboriginal children to examine this relationship.[3] The 'fetal origins hypothesis' proposes that fetal under-nutrition results in permanent structural alterations in lung development that cause persistent deficits in lung function.[20] This hypothesis is supported by studies in other populations,[20,23] however, only one of these, from urban India, was conducted in the developing world.[23]

Although maternal smoking in pregnancy is known to be associated with reduced childhood lung function,[24] there was no such association in our cohort. The method of assessment of maternal smoking (retrospective self-reporting) may have been insufficiently robust or, alternatively, the heavy environmental smoke exposure[4,5]

that is experienced by many of these children from an early age may be more influential than antenatal cigarette exposure in this population.

Studies have repeatedly shown a relationship between preterm birth and reduced lung function,[25] however, most have focused on very low birthweight babies, many of whom have ventilator-related lung disease. In our study, although only six children were delivered before 32 weeks gestation and only two were ventilated for more than 7 days, those with NNLD still had significantly reduced FEV1 and a trend towards reduced FEF25–75. These very small numbers prohibit any definite conclusions about this relationship.

Strengths of our study include the cohort size and the high follow-up achieved in spite of population mobility.[10] The NT medical referral system meant we could use centralised hospital records to assess history instead of relying on recall, which would have been difficult given the inconstant presence of parents and language barriers. The majority of admissions, and all serious illness, have been captured by this method as there is a low threshold for admission in this population. We analysed only respiratory illnesses severe enough to result in hospitalisation. Including outpatient attendances or illnesses not requiring medical attention might have altered the associations, however, these could not be reliably assessed.

Another limitation was the inability of most children to comply with the six second expiration manoeuvre recommended by the ATS[13] although the feasibility of this criterion in children has been questioned.[14] As spirometry was frequently conducted in difficult field conditions and the duration of exhalation was significantly related to age, more controlled conditions may have yielded better results, particularly for the younger children. For consistency with others[15] we used 3-s exhalation as a minimum criterion for acceptability. This led to the exclusion of 84 otherwise acceptable tests. There were minimal differences when data were reanalysed including these 84 results.

In this study, non-urban residence, current cough, a history of hospitalisations for respiratory disease and NNLD were independent predictors of reduced lung function at 8–14 years of age. Although the effects of each of the significant exposures on lung function in this study were relatively modest, even small (3–4%) differences in FEV1 and FVC may have a significant public health impact.[26] While antenatal factors may influence lung development, the greater influence of poor living conditions and infections overrides any detectable effects of antenatal factors in this population. For the Australian Aboriginal population, improving living conditions as well as prevention and better treatment of childhood respiratory infections and asthma may be the most effective ways to improve childhood lung function and to reduce the burden of respiratory disease and its consequences in Aboriginal adults.

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References

1 Hanna J, Torzillo PJ. Acute respiratory infections in Australian Aboriginal children; current knowledge and future requirements. *PNG Med. J.* 1991; **34**: 204–10.
2 Dempsey KE, Condon JR. *Mortality in the Northern Territory 1979–1997.* Darwin: Epidemiology Branch Territory Health Services, 1999.
3 Chandler D, Dugdale AE. Respiratory function tests in Aboriginal children on Mornington Island. *Med. J. Aust.* 1980; **1**: 10.
4 Bremner PR, de Klerk NH, Ryan GF *et al.* Respiratory symptoms and lung function in Aborigines from tropical Western Australia. *Am. J. Respir. Crit. Care Med.* 1998; **158**: 1724–9.
5 Veale AJ, Peat JK, Salome CM, Thompson JE, Woolcock AJ. 'Normal' lung function in rural Australian Aborigines. *Aust. N. Z. J. Med.* 1997; **27**: 543–9.
6 Watson DS, Watson RM, Siskind V. Respiratory function in Aboriginal children. *Med. J. Aust.* 1986; **144**: S11–13.
7 Maxwell GM. Chronic chest disease in Australian Aboriginal children. *Arch. Dis Child* 1972; **47**: 897–901.
8 Chang AB, Masel JP, Boyce NC, Wheaton G, Torzillo P. Non-CF bronchiectasis: clinical and HRCT evaluation. *Paediatr. Pulmonol.* 2003; **35**: 477–83.
9 Sayers S, Powers J. Risk factors for Aboriginal low birthweight, intrauterine growth retardation and premature birth in the Darwin health region. *Aust. N. Z. J. Public Health* 1997; **21**: 524–9.
10 Sayers S, Mackerras DE, Singh G, Bucens I, Flynn K, Reid A. An Australian Aboriginal Birth Cohort: a unique resource for a life course study of an Indigenous population. A study protocol. *BMC Int. Health Hum. Rights* 2003; **3**: 1.
11 Guaran RL, Wein P, Sheedy M, Walstab J, Beischer NA. Update of growth percentiles for infants born in an Australian population. *Aust. N. Z. J. Obstet. Gynaecol.* 1994; **34**: 39–50.
12 Mackerras DE, Reid A, Sayers SM, Singh GR, Bucens IK, Flynn KA. Growth and morbidity in children in the Aboriginal Birth Cohort Study: the remote-urban differential. *Med. J. Aust.* 2003; **178**: 56–60.
13 American Thoracic Society. Standardisation of spirometry 1994 update. *Am. J. Respir. Crit. Care Med.* 1995; **152**: 1107–36.
14 Sly PD, Robertson CF. A review of pulmonary function testing in children. *J. Asthma* 1990; **27**: 137–47.
15 StatCorp. *Stata Statistical Software: Release 7.0*. College Station, TX: StatCorp LP, 2001.
16 American Thoracic Society. Lung Function testing: selection of reference values and interpretive strategies. *Am. Rev. Respir. Dis.* 1991; **144**: 1202–18.
17 Maxwell GM, Elliott RB, McCoy WT, Langsford WA. Respiratory infections in Australian Aboriginal children: a clinical and radiological study. *Med. J Aust.* 1968; **30**: 990–3.
18 Torzillo PJ, Pholeros P. Household infrastructure in Aboriginal communities and the implications for health improvement. *Med. J. Aust.* 2002; **176**: 502–3.
19 Gracey M, Anderson CM. Hospital admissions for infections of Aboriginal and non-Aboriginal infants and children in Western Australia 1981–1986. *Aust. Paediatr. J.* 1989; **25**: 230–5.
20 Barker DJP, Godfrey KM, Fall C, Osmond C, Winter PD, Shaheen SO. Relation of birthweight and childhood respiratory infection to adult lung function and death from chronic obstructive airways disease. *BMJ* 1991; **303**: 671–5.

21 Samet JM, Tager IB, Speizer FE. The relationship between respiratory illness in childhood and chronic airflow obstruction in adulthood. *Am. Rev. Respir. Dis.* 1983; **127**: 508–23.
22 Whybourne AM, Lesnikowski CL, Ruben AR, Walker AC. Low rates of hospitalisation for asthma among Aboriginal children compared to non-Aboriginal children of the Top End of the Northern Territory. *J. Paediatr. Child Health* 1999; **35**: 438–41.
23 Stein CE, Kumaran K, Fall CHD, Shaheen SO, Osmond C, Barker DJP. Relation of fetal growth to adult lung function in South India. *Thorax* 1997; **52**: 895–9.
24 Tager IB, Weiss ST, Munoz A, Rosner B, Speizer FE. Longitudinal study of the effects of maternal smoking on pulmonary function in children. *N. Engl. J. Med.* 1983; **309**: 699–703.
25 Doyle LW, Cheung MMH, Ford GW, Olinsky A, Davis NM, Callanan C. Birth weight <1501 g and respiratory health at age 14. *Arch. Dis. Child* 2001; **84**: 40–4.
26 Kunzil N, Ackermann-Liebrich U, Brandili O, Tschopp JM, Schindler C, Leuenberger P. Clinically "small" effects of air pollution on FVC have a large public health impact. Swiss Study on Air Pollution and Lung Disease in Adults (SAPALDIA) team. *Eur. Respir. J.* 2000; **15**: 131–6.