Eur J Epidemiol (2013) 28:79–85
DOI 10.1007/s10654-013-9761-7

RESPIRATORY EPIDEMIOLOGY

Obstructive pulmonary disease in old age among individuals born preterm

Eva Berggren Broström · Olof Akre · Miriam Katz-Salamon · David Jaraj · Magnus Kaijser

Received: 1 May 2012 / Accepted: 7 January 2013 / Published online: 23 January 2013
© Springer Science+Business Media Dordrecht 2013

Abstract There are only few studies of the association between preterm birth and risk of chronic lung disease in old age. The aim of this study was to assess the association between poor fetal growth, preterm birth, sex and risk of asthma and Chronic Obstructive Pulmonary Disease (COPD) in adulthood. We have followed up a cohort of all infants born preterm (<35 weeks) or with low birth weight (<2,000 and <2,100 g for girls and boys, respectively) and an equal number of controls in a source population of 250,000 individuals born from 1925 through 1949 in Sweden (6,425 subjects in total). Cases of asthma and COPD were identified through the Swedish Patient Register and we considered cohort subjects as cases if they had a main or additional discharge diagnosis of asthma or COPD. For any obstructive airways disease, there was a statistically significant increase in risk with decreasing birth weight and gestational duration among women but not among men. Compared to women born at term, women born before 32 weeks of gestation had a hazard ratio for any obstructive airways disease and asthma of 2.77 (95 % CI 1.39–5.54) and 5.67 (1.73–18.6), respectively. Low birth weight and preterm birth are risk factors for obstructive airways disease also among the old, but the importance of these risk factors differs between the sexes.

Keywords Preterm birth · Low birth weight · Obstructive pulmonary disease

E. B. Broström
Department of Women's and Children's Health (KBH), K6, Child, Division of Neonatology, Karolinska Institutet (KI), Karolinska Universitetssjukhuset, Q2:07, 171 76 Stockholm, Sweden

O. Akre · D. Jaraj · M. Kaijser
Clinical Epidemiology Unit, Department of Medicine Solna, Karolinska Institutet, 171 76 Stockholm, Sweden

O. Akre · D. Jaraj
Department of Urology, Karolinska University Hospital, 171 76 Stockholm, Sweden

M. Katz-Salamon
Department of Woman and Child Health, Neonatal Unit, Karolinska Institutet, Elevhemmet H102, 171 76 Stockholm, Sweden

M. Kaijser (✉)
Department of Neuroradiology, R3:00, Karolinska University Hospital, 171 76 Stockholm, Sweden
e-mail: magnus.kaijser@ki.se

Introduction

There is a growing concern that preterm birth may have implications for long-term respiratory function [1]. Infants born prematurely often develop respiratory insufficiency and bronchopulmonary dysplasia (BPD) [2]. Although symptoms tend to improve by age, prematurely born infants are at greatly increased risk of respiratory problems such as coughing, wheezing, and impaired lung function later in childhood and adolescence [3–7].

Several studies have assessed adolescent and adult lung function in relation to birth weight [8]. According to a meta-analysis of 9 studies [8], there is an association between birth weight and forced expiratory volume in one second (FEV_1). This notion has been supported also in more recent studies [9–11]. In addition, low birth weight has been reported to be associated with adult asthma, respiratory infections and respiratory failure, especially in females [12]. Low birth weight can be due either to short gestational duration or poor fetal growth, or a combination of both, but few studies have assessed to what extent the

association between low birth weight and lung function is due to preterm birth [4, 6, 13], or if the association between preterm birth and risk of chronic lung disease remains also in old age [14].

The aim of this study was to assess the association between poor fetal growth, preterm birth, and sex and risk of asthma and Chronic Obstructive Pulmonary Disease (COPD) in adulthood and old age. We have followed up a cohort of all infants born preterm (<35 weeks) or with low birth weight (<2,000 g and <2,100 g for girls and boys, respectively) in a source population of 250,000 individuals born from 1925 through 1949 in Sweden.

Methods

Study cohort

The source population for this cohort study was all births from 1925 through 1949 at four major delivery units in Sweden (Allmänna BB and Södra BB in Stockholm, Uppsala University Hospital, and Sundsvalls County Hospital). By manually examining the approximately 250,000 births records during this period, we identified an exposed cohort by selecting all newborn infants with a gestational duration of less than 35 weeks and/or a birth weight of less than 2,000 g for girls and 2,100 g for boys. The study was initially performed to assess risk of cancer among individuals with a history of preterm birth and/or low birth weight. Since boys on average weight 100 g more at birth than do girls, and we wanted to create a cohort with an equal distribution of boys and girls, we arbitrarily chose the cut off points 2,000 and 2,100 g. Subjects who emigrated or deceased prior to 1987 were excluded.

As unexposed cohort members, we selected subjects who neither were born preterm nor with low birth weight. For convenience, we selected the first child of same sex and hospital of birth born after each exposed subject.

Perinatal definitions and categorization

We used last menstrual period to estimate gestational duration, which was categorized into 4 groups: 32 completed weeks or less (very preterm), 33–36 (moderately preterm), 37–42 (term), and 43 weeks or more (postterm). Birth weight was categorized in 7 groups of 500 g intervals, from less than 1,500 g up to 4,000 g or more.

In estimation of birth weight for gestational age (a proxy for fetal growth), we used the Swedish reference curve for estimated intra-uterine fetal growth, based on ultrasound estimations of fetal weights in normal pregnancies ending at term [15]. Birth weight for gestational age was categorized in five groups according to their distance from average in numbers of standard deviations (≤ -2 standard deviations [SD], > -2 to -1 SD, > -1 to $+1$ SD, $>+1$ to $+2$ SD, and $>+2$ SD).

The socioeconomic status of the family was assessed by the father's or single mother's occupation using three categories: high (college education), medium (white-collar workers and farm owners with no college education), and low (blue collar workers and farm hands).

Follow-up and analysis

Follow-up started in January 1st, 1987 and continued to December 31, 2006. Asthma and COPD was defined from the Hospital Discharge Registers and Cause of Death Register. The starting date of follow –up was chosen when national coverage in the Hospital Discharge Registers was available. We used the Register of Population and Population Changes to ascertain emigration and the Cause of Death Register to ascertain death during this time. During the period 1987 through 1996 diagnoses were categorized according to the 9th revision of the International Classification of Disease, and 1997 onwards according to the 10th revision. We considered cohort subjects as cases if they had a main diagnosis of asthma or COPD with diagnostic codes 493 or 491, 492, or 496, respectively, before 1997 (9th revision of International Classification of Disease) and diagnostic codes J45 or J41-J44, respectively, after 1997 (10th revision of International Classification of Disease).

Since COPD is strongly associated with smoking, we also assessed the risk of lung cancer (162 according to the 7th revision of International Classification of Disease) in the cohort in order to validate our findings. Risk of lung cancer was assessed through the Swedish Cancer Registry.

Data were modeled through conditional Cox' regression, using the TPHREG procedure in SAS Statistical Software, version 9.1 (SAS Institute Inc., Cary, NC, USA). All analyses were conditioned by calendar period of birth and socioeconomic status. Missing information on socioeconomic status was treated as a separate category. The study was approved by the ethics committee of the Karolinska Institutet.

Results

At start of follow up, there were 6,425 subjects in the cohort (Fig. 1). There were 2,176 subjects in the cohort with low birth weight (<2,500 g), and 151 had a birth weight of less than 1,500 g. In all, 2,931 subjects were born preterm (<37 weeks), and 986 had a gestational duration of 32 weeks or less. The distributions of birth weight, gestational duration, and fetal growth are presented in further detail in Table 1.

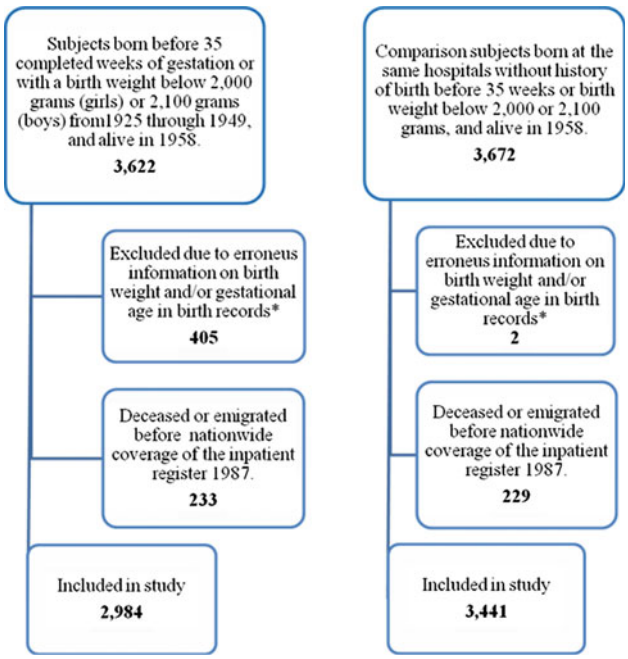

Fig. 1 Study inclusion flow chart. *Birth weight for gestational age of more than 4 standard deviations above or below mean birth weight for gestational age according to Swedish reference curves [15]

Table 1 Number of subjects by birth weight, gestational duration and Gestational age adjusted birth weight

	Gestational duration				
	≤32	33–36	37–42	≥43	Total
Birth weight					
<1,500	132	19	0	0	151
1,500–1,999	403	392	39	1	835
2,000–2,499	307	716	161	6	1,190
2,500–2,999	144	454	377	25	1,000
3,000–3,499	0	252	1,045	70	1,367
3,500–3,999	0	110	1,105	94	1,309
≥4,000	0	2	494	77	573
Total	986	1,945	3,221	273	6,425
Gestational age adjusted birth weight					
≤ −2 SD	26	256	255	39	576
> −2 to −1 SD	94	240	522	86	942
> −1 to +1 SD	427	816	2,016	137	3,396
> +1 to +2 SD	140	218	348	9	715
> +2 SD	299	415	80	2	796
Total	986	1,945	3,221	273	6,425

During follow up, 150 subjects were hospitalized with a principal diagnosis of any obstructive airways disease (Table 2). Among these 150 subjects, there were 17 who were treated with both asthma and COPD as main diagnoses at different occasions, yielding 45 cases of asthma (Table 3) and 122 cases of COPD (Table 4). For any obstructive airways disease, there was a statistically significant increase in risk with decreasing birth weight when males and females were analyzed together (p for trend = 0.046, Table 2). When analyzing the two sexes separately, there was no significant association between birth weight and risk among males, whereas there was a more than fourfold increase for any obstructive airways disease among women with a birth weight of less than 1,500 g compared to women with a birth weight of 3,000–3,499 g (HR = 4.57, 95 % Confidence Interval = 1.43–14.6; Table 2). When analyzing gestational duration and gestational age adjusted birth weight—we found preterm birth, i.e. birth before 37 completed weeks of gestation, being associated with an increased risk for obstructive airways disease among women, but not among men. Compared to women born at term, women born before 33 weeks of gestation had an almost threefold increase in risk for any obstructive airways disease. For age adjusted birth weight, the association was u-shaped rather than linear (Table 2).

When analyzing asthma and COPD separately, there was a significant increase in risk of asthma with decreasing birth weight when both sexes where analyzed together (p for trend = 0.006). When separating the sexes, the association remained unaltered among women (p for trend = 0.0013) but not among men (p = 0.91) (Table 3). When analyzing the two determinants of birth weight, preterm birth was a strong risk factor for asthma (Table 3) when both sexes were analyzed together. We found no association between risk of asthma and age adjusted birth weight (Table 3). When excluding the 17 patients who had been treated both for asthma and for COPD in the analysis, results were essentially unaltered (data not shown). For COPD, we found a significant increase in risk with decreasing birth weight in women (p for trend = 0.02) but that was not seen in men or when men and woman were analyzed together. When we excluded patients with a history of a main diagnosis of both asthma and COPD, this test of trend was not statistically significant (p = 0.08), albeit the point estimates were essentially unaltered (data not shown). Hazard ratio for women born <32 weeks were 2.15 (CI 0.96–4.81). There was a U-shaped association between fetal growth and risk of disease (Table 4).

We found no increase in risk for lung cancer among subjects born preterm or with poor fetal growth. On the contrary, there was a statistically significant positive association between lung cancer and both birth weight and gestational duration (Table 5).

Discussion

We found that low birth weight and short gestational duration are risk factors for obstructive airways disease in

Table 2 Risk of obstructive airways disease (ICD9 = 491–493, 496, ICD10 = J41–J45) by sex, birth weight, gestational duration, and Gestational age adjusted birth weight

	All			Men			Women		
	# cases	HR*	95 % CI**	# cases	HR*	95 % CI**	# cases	HR*	95 % CI**
Birth weight									
<1,500	6	2.34	(0.96–5.70)	1	0.81	(0.11–6.08)	5	4.57	(1.43–14.6)
1,500–1,999	30	1.86	(1.10–3.15)	12	1.57	(0.76–3.24)	18	2.79	(1.15–6.73)
2,000–2,499	23	0.97	(0.55–1.70)	12	0.80	(0.39–1.64)	11	1.45	(0.55–3.77)
2,500–2,999	27	1.40	(0.82–2.41)	16	1.22	(0.63–2.38)	11	1.89	(0.72–4.93)
3,000–3,499	26	1	Reference	19	1	Reference	7	1	Reference
3,500–3,999	26	1.07	(0.62–1.84)	21	1.11	(0.60–2.07)	5	0.89	(0.28–2.81)
≥4,000	12	1.13	(0.57–2.25)	11	1.32	(0.63–2.79)	1	0.39	(0.05–3.16)
P for trend			0.046			0.87			0.0004
Gestational duration (weeks)									
<32	31	1.48	(0.96–2.27)	11	0.92	(0.48–1.77)	20	2.77	(1.39–5.54)
33–36	50	1.23	(0.85–1.78)	29	1.06	(0.67–1.68)	21	1.95	(1.00–3.83)
37–42	65	1	Reference	50	1	Reference	15	1	Reference
≥43	4	0.79	(0.29–2.19)	2	0.59	(0.14–2.45)	2	1.44	(0.33–6.31)
P for trend			0.047			0.84			0.0052
Gestational age adjusted birth weight									
≤ −2 SD	20	1.91	(1.15–3.18)	8	1.27	(0.59–2.72)	12	2.98	(1.44–6.13)
> −2 to −1 SD	24	1.42	(0.89–2.29)	14	1.26	(0.69–2.32)	10	1.76	(0.82–3.77)
> −1 to +1 SD	60	1	Reference	40	1	Reference	20	1	Reference
> +1 to +2 SD	22	1.70	(1.04–2.77)	17	1.85	(1.05–3.28)	5	1.15	(0.43–3.12)
> +2 SD	24	1.54	(0.96–2.48)	13	1.25	(0.67–2.35)	11	1.95	(0.91–4.15)
P for trend			0.93			0.44			0.27
Total	150			92			58		

* *HR* hazard ratio, ** *CI* confidence interval

adulthood and old age. We also found that the importance of these risk factors may differ between the sexes. The data in this study are unique in several ways: First, the over sampling of preterm births and subjects with a low birth weight enabled us to study the effect of these exposures with an unprecedented precision. Second, we were able to follow up these subjects for chronic lung disease into old age. Third, whereas most other studies relied on questionnaire-based information on the birth characteristics, our data were registered at the time of birth, thus ensuring measurement accuracy. For this reason, we also have the best available information (at that time) on gestational duration, and we were thus able to distinguish preterm birth from low birth weight for gestational age; two essentially distinct exposures that were rarely separated in previous long-term follow-up studies.

A limitation is the definition of the outcomes. First, we use the inpatient register to assess cases of asthma of COPD. This means that less severe cases treated in outpatient care are not included, and that we have a selection in our study of more severe cases. This per se should not put in question the validity of our diagnoses, but one could imagine that other diseases associated with preterm birth and low birth weight increase the risk for hospitalization and, thus, to a diagnosis of asthma or COPD in the register. By including only main diagnoses and not auxiliary diagnoses in the Inpatient Register, however, we have tried to reduce the risk of such case ascertainment bias to a minimum. Whether or not our results should be different if our study included also cases of asthma and COPD treated only as outpatient can, however, not be told. Second, in the clinics these diagnoses are overlapping so that patients with COPD may be diagnosed with asthma rather than the inverse. This is reflected by the fact that 17 patients were treated with main diagnoses of both asthma and COPD. Excluding these patients with both diagnoses had little impact on the results, but if our results would have been different with a more thorough diagnostic differentiation between asthma and COPD can not be told. A concern about the study is that those surviving premature birth and retarded fetal growth in the early 1900s, are markedly different from today's patients at the neonatology ward. This problem of non-comparability is inherent in all long term follow-up studies and can never be solved.

Table 3 Risk of asthma (ICD9 = 493, ICD10 = J45) by sex, birth weight, gestational duration and gestational age adjusted birth weight

	All			Men			Women		
	# cases	HR*	95 % CI**	# cases	HR*	95 % CI**	# cases	HR*	95 % CI**
Birth weight									
<1,500	2	2.07	(0.44–9.70)	0	n.a.*	n.a.*	2	6.34	(0.88–45.6)
1,500–1,999	12	2.35	(0.98–5.61)	1	0.39	(0.05–3.20)	11	6.09	(1.33–27.8)
2,000–2,499	10	1.33	(0.54–3.29)	5	0.96	(0.30–3.06)	5	2.47	(0.47–12.9)
2,500–2,999	4	0.60	(0.19–1.96)	3	0.63	(0.16–2.45)	1	0.61	(0.05–6.80)
3,000–3,499	9	1	Reference	7	1	Reference	2	1	Reference
3,500–3,999	6	0.74	(0.26–2.09)	3	0.44	(0.11–1.72)	3	1.87	(0.31–11.3)
≥4,000	2	0.54	(0.12–2.48)	2	0.65	(0.13–3.13)	0	n.a.*	n.a.*
P for trend			0.0060			0.91			0.0013
Gestational duration (weeks)									
<32	11	2.29	(1.05–5.01)	1	0.39	(0.05–3.06)	10	5.67	(1.73–18.6)
33–36	19	2.14	(1.08–4.22)	9	1.53	(0.63–3.72)	10	3.83	(1.18–12.4)
37–42	15	1	Reference	11	1	Reference	4	1	Reference
≥43	0	n.a.*	n.a.*	0	n.a.*	n.a.*	0	n.a.*	n.a.*
P for trend			0.0064			0.90			0.0016
Gestational age adjusted birth weight									
≤ −2 SD	6	1.66	(0.67–4.13)	1	0.69	(0.09–5.42)	5	2.21	(0.76–6.43)
> −2 to −1 SD	4	0.66	(0.22–1.91)	2	0.69	(0.15–3.17)	2	0.64	(0.14–2.89)
> −1 to +1 SD	21	1	Reference	10	1	Reference	11	1	Reference
> +1 to +2 SD	7	1.50	(0.63–3.52)	6	2.62	(0.94–7.28)	1	0.41	(0.05–3.21)
> +2 SD	7	1.26	(0.53–2.98)	2	0.79	(0.17–3.63)	5	1.60	(0.54–4.79)
P for trend			0.80			0.54			0.78
Total	45			21			24		

* *HR* hazard ratio, ** *CI* confidence interval

In our cohort, approximately 45 % of the subjects born before 33 weeks of gestation had a birth weight for gestational age that was above +1 standard deviation above the mean, whereas only 12 % had a birth weight that was below −1 standard deviation below the mean. This skewed distribution may have several explanations. First, selective survival favoring subjects with normal or above average birth weight for gestational age is likely. In a study of subjects from the same area born during the period 1915–1930, higher mortality was found among children with low birth weight [16]. Childhood mortality declined steeply in Sweden during the period when our subjects were born (1925 through 1949). Second, whereas we used information on the last menstrual period to estimate gestational age, our reference curve for fetal growth was based on ultrasonographically dated pregnancies from the 1990s.

Smoking is the leading environmental cause of COPD. It may also be correlated to birth weight through maternal smoking, and there is thus a theoretical risk of confounding by smoking in the data. We were unable to obtain information on smoking status for the subjects. In the results presented in Table 5, however, we found a slight positive association between birth weight and gestational age on the one hand, and lung cancer on the other. It can therefore be inferred that any bias caused by smoking should dilute the association between our study variables and asthma and COPD. As mentioned above, smoking is considered the major risk factor to develop COPD, but 25 % of COPD occur in non-smokers [17]. In one population based study only 15 % of the men were not current smokers while 39 % of the women had never smoked [18]. Other identified risk factors described are pneumonia, heart disease, malignancies, osteoporosis, musculoskeletal disorders, insomnia, peptic ulcer, migraine, sinusitis and depression while prematurity is not mentioned [17–20]. One could argue that even though smoking among the subjects themselves is unlikely to explain our findings, our results could, at least in part, be due to antenatal and childhood exposure to maternal smoking. It is important to remember, however, that although smoking among women became rather common in the second half of the twentieth century, it was still fairly uncommon during the time when our subjects were born [21]. For the majority of our study subjects, the maternal smoking rate is likely to have been

Table 4 Risk of COPD and chronic bronchitis (ICD9 = 491, 492, 496, ICD10 = J41–J44) as main or additional discharge diagnosis by sex, birth weight, gestational duration and fetal growth

	All			Men			Women		
	# cases	HR*	95 % CI**	# cases	HR*	95 % CI**	# cases	HR*	95 % CI**
Birth weight (grams)									
<1,500	4	2.31	(0.78–6.84)	1	1.31	(0.17–10.2)	3	3.10	(0.76–12.7)
1,500–1,999	24	2.09	(1.13–3.85)	12	2.42	(1.09–5.40)	12	2.06	(0.76–5.55)
2,000–2,499	18	1.08	(0.56–2.07)	11	1.14	(0.50–2.60)	7	1.05	(0.35–3.16)
2,500–2,999	24	1.80	(0.98–3.33)	14	1.68	(0.78–3.64)	10	2.02	(0.73–5.61)
3,000–3,499	18	1	Reference	12	1	Reference	6	1	Reference
3,500–3,999	22	1.32	(0.71–2.46)	19	1.61	(0.78–3.32)	3	0.64	(0.16–2.56)
≥4,000	12	1.69	(0.81–3.51)	11	2.14	(0.94–4.87)	1	0.50	(0.06–4.19)
P for trend			0.26			0.97			0.020
Gestational duration (weeks)									
<32	24	1.33	(0.82–2.15)	11	1.04	(0.54–2.03)	13	2.15	(0.96–4.81)
33–36	39	1.11	(0.73–1.67)	24	1.02	(0.62–1.68)	15	1.63	(0.75–3.51)
37–42	55	1	Reference	43	1	Reference	12	1	Reference
≥43	4	1.00	(0.36–2.76)	2	0.73	(0.18–3.05)	2	1.80	(0.40–8.09)
P for trend			0.26			0.77			0.11
Gestational age adjusted birth weight									
≤ −2 SD	16	2.01	(1.13–3.55)	8	1.56	(0.72–3.39)	8	2.94	(1.21–7.18)
> −2 to −1 SD	22	1.76	(1.06–2.93)	13	1.48	(0.78–2.83)	9	2.46	(1.05–5.77)
> −1 to +1 SD	45	1	Reference	32	1	Reference	13	1	Reference
> +1 to +2 SD	20	2.06	(1.22–3.50)	16	2.15	(1.18–3.93)	4	1.49	(0.48–4.62)
> +2 SD	19	1.61	(0.94–2.76)	11	1.31	(0.66–2.60)	8	2.23	(0.91–5.44)
P for trend			0.82			0.53			0.51
Total	122			80			42		

* *HR* hazard ratio, ** *CI* confidence interval

Table 5 Risk of lung cancer (ICD7 = 162) by birth weight, gestational duration and fetal growth

	All			Men			Women		
	# cases	HR	CI	# cases	HR	CI	# cases	HR	CI
Birth weight									
<1,500–2,499	17	0.67	(0.36–1.22)	10	0.59	(0.28–1.25)	7	1.23	(0.39–3.91)
2,500–3,499	28	1	Reference	23	1	Reference	5	1	Reference
≥3,500	30	1.46	(0.87–2.45)	21	1.12	(0.62–2.02)	9	3.06	(1.02–9.17)
P for trend			0.01			0.11			0.08
Gestational duration (weeks)									
<37	26	0.59	(0.36–0.98)	18	0.70	(0.39–1.28)	8	0.49	(0.20–1.20)
37–42	39	1	Reference	27	1	Reference	12	1	Reference
>42	10	1.05	(0.52–2.11)	9	1.42	(0.66–3.05)	1	0.32	(0.04–2.43)
P for trend			0.04			0.08			0.57
Gestational age adjusted birth weight									
≤ −1 SD	13	0.69	(0.37–1.29)	11	1.10	(0.57–2.13)	0	0	n.a.*
> −1 to 1	42	1	Reference	28	1	Reference	14	1	Reference
> +1 SD	20	0.98	(0.58–1.68)	13	0.94	(0.49–1.82)	7	1.06	(0.43–2.64)
P for trend			0.35			0.68			n.a.*

15 % or lover. It is therefore unlikely that our results should be due to study subjects exposure to maternal smoking. Nevertheless, the overall lack of data on smoking in this study is a major limitation, and further studies are needed to assess to what extent the association between preterm birth and low birth weight and Asthma and COPD are attributable to smoking.

Apart from smoking, there are several other factors important for the development for asthma and COPD in adulthood that we have no information on, including maternal body mass index, history of asthma, number of siblings and pets in the household. To what extent our results are influenced by or mediated through any of these factors can not be told.

Our data suggests that for asthma and COPD, the importance of preterm birth and low birth weight varies between the sexes. These differences were not extected a priori, and although the study is one of the larger in its kind, the number of subjects with asthma and COPD in each stratum is low. Therefore, even though there can be several mechansisms that would explain differences between the sexes, our results need corroboration from other studies before speculation of underlying mechanisms are of value.

Conclusions

Our data suggest that low birth weight and preterm birth are risk factors for obstructive airways disease also among the old, and that the importance of these risk factors differs between the sexes. This may have implications for the future, since improvements in neonatal care of the preterm have lead to infants surviving at a lower gestational age and thus to an increasing total number of infants with pulmonary impairment at risk to develop chronic obstructive pulmonary disease.

References

1. Bentham JR, Shaw NJ. Some chronic obstructive pulmonary disease will originate in neonatal intensive care units. Paediatr Respir Rev. 2005;6(1):29–32. doi:10.1016/j.prrv.2004.11.005.
2. Jobe AH, Bancalari E. Bronchopulmonary dysplasia. Am J Respir Crit Care Med. 2001;163(7):1723–9.
3. Baraldi E, Carraro S, Filippone M. Bronchopulmonary dysplasia: definitions and long-term respiratory outcome. Early Hum Dev. 2009;85(10 Suppl):S1–3. doi:10.1016/j.earlhumdev.2009.08.002.
4. Brostrom EB, Thunqvist P, Adenfelt G, Borling E, Katz-Salamon M. Obstructive lung disease in children with mild to severe BPD. Respir Med. 2010;104(3):362–70. doi:10.1016/j.rmed.2009.10.008.
5. Bush A. Update in pediatric lung disease 2007. Am J Respir Crit Care Med. 2008;177(7):686–95. doi:10.1164/rccm.200801-028UP.
6. Bush A. COPD: a pediatric disease. COPD. 2008;5(1):53–67. doi:10.1080/15412550701815965.
7. Halvorsen T, Skadberg BT, Eide GE, Roksund OD, Carlsen KH, Bakke P. Pulmonary outcome in adolescents of extreme preterm birth: a regional cohort study. Acta Paediatr. 2004;93(10):1294–300.
8. Lawlor DA, Ebrahim S, Davey Smith G. Association of birth weight with adult lung function: findings from the British Women's Heart and Health Study and a meta-analysis. Thorax. 2005;60(10):851–8. doi:10.1136/thx.2005.042408.
9. Hancox RJ, Poulton R, Greene JM, McLachlan CR, Pearce MS, Sears MR. Associations between birth weight, early childhood weight gain and adult lung function. Thorax. 2009;64(3):228–32. doi:10.1136/thx.2008.103978.
10. Tennant PW, Gibson GJ, Parker L, Pearce MS. Childhood respiratory illness and lung function at ages 14 and 50 years: childhood respiratory illness and lung function. Chest. 2010;137(1):146–55. doi:10.1378/chest.09-0352.
11. Tennant PW, Gibson GJ, Pearce MS. Lifecourse predictors of adult respiratory function: results from the Newcastle Thousand Families Study. Thorax. 2008;63(9):823–30. doi:10.1136/thx.2008.096388.
12. Walter EC, Ehlenbach WJ, Hotchkin DL, Chien JW, Koepsell TD. Low birth weight and respiratory disease in adulthood: a population-based case-control study. Am J Respir Crit Care Med. 2009;180(2):176–80. doi:10.1164/rccm.200901-0046OC.
13. Barker DJ, Godfrey KM, Fall C, Osmond C, Winter PD, Shaheen SO. Relation of birth weight and childhood respiratory infection to adult lung function and death from chronic obstructive airways disease. BMJ. 1991;303(6804):671–5.
14. Villamor E, Iliadou A, Cnattingius S. Is the association between low birth weight and asthma independent of genetic and shared environmental factors? Am J Epidemiol. 2009;169(11):1337–43. doi:10.1093/aje/kwp054.
15. Niklasson A, Ericson A, Fryer JG, Karlberg J, Lawrence C, Karlberg P. An update of the Swedish reference standards for weight, length and head circumference at birth for given gestational age (1977–1981). Acta Paediatr Scand. 1991;80(8–9):756–62.
16. Leon DA, Lithell HO, Vagero D, et al. Reduced fetal growth rate and increased risk of death from ischaemic heart disease: cohort study of 15000 Swedish men and women born 1915–29. BMJ. 1998;317(7153):241–5.
17. Garcia Rodriguez LA, Wallander MA, Tolosa LB, Johansson S. Chronic obstructive pulmonary disease in UK primary care: incidence and risk factors. COPD. 2009;6(5):369–79. doi:10.1080/15412550903156325.
18. Lundback B, Eriksson B, Lindberg A, et al. A 20-year follow-up of a population study-based COPD cohort-report from the obstructive lung disease in Northern Sweden studies. COPD. 2009;6(4):263–71. doi:10.1080/15412550903061483.
19. Soriano JB, Visick GT, Muellerova H, Payvandi N, Hansell AL. Patterns of comorbidities in newly diagnosed COPD and asthma in primary care. Chest. 2005;128(4):2099–107. doi:10.1378/chest.128.4.2099.
20. van Manen JG, Bindels PJ, Ijzermans CJ, van der Zee JS, Bottema BJ, Schade E. Prevalence of comorbidity in patients with a chronic airway obstruction and controls over the age of 40. J Clin Epidemiol. 2001;54(3):287–93.
21. Pettersson A, Kaijser M, Richiardi L, Askling J, Ekbom A, Akre O. Women smoking and testicular cancer: one epidemic causing another? Int J Cancer. 2004;109(6):941–4.