What can Busselton population health surveys tell us about asthma in older people?

Alan L James, Matthew W Knuiman, Helen C Bartholomew and Arthur (Bill) W Musk

ABSTRACT

What we need to know

- Do the characteristics of asthma differ in people older than 55 years compared with younger people with respect to risk factors (atopy, airway hyper-responsiveness and genetic variation), smoking, lung function and other illness?
- How do inflammation and remodelling of airways vary with age and with duration and severity of asthma?

What we need to do

- Continue collecting prevalence data for asthma and its risk factors.
- Assess (i) period and cohort effects on asthma and its risk factors and (ii) interactions between age, smoking, severity and duration of asthma, lung function and airway responsiveness, and other concurrent disease.
- Measure airway responsiveness and exhaled nitric oxide to detect airway abnormalities in older people and relate this to the diagnoses of asthma and other diseases.

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Asthma occurs from infancy to very old age. In older people with asthma, the presence of other disease may confound the diagnosis, and this may account for undiagnosed asthma.[1] However, it is known that asthma persists from infancy or adolescence and may arise in advanced age, and that mortality from asthma is increased in older people.[2] We also know a great deal about the features of asthma in older people. One study found that symptoms or treatment requirements were not related to duration of disease, although people with a long duration of asthma had poorer lung function and more often had a previous history of allergic disease.[3] Another study found that 6.5% of people aged 70 or older in a whole community study of a single Welsh town had asthma.[4] Data from the Normative Aging Study have shown that cat allergy preceded airway hyper-responsiveness and diagnosis of asthma in older men;[5] in another study, cockroach allergy was associated with more severe asthma in older people.[6] Cross-sectional studies of asthma in older people suggest that duration of asthma may be related to more severe abnormalities of lung function but not to severity of symptoms.[3,7]

What do we need to know in the context of the current evidence?

We need to know more about the interactions of asthma with its risk factors and symptoms, and its relationship with lung function, age and severity. We need to examine markers of remodelling (lung function, decline in lung function and non-specific airway responsiveness) and airway inflammation (such as exhaled nitric oxide) in relation to asthma at different ages.

The Busselton population health surveys

Respiratory and general health data have been collected in the town of Busselton, WA, since 1966. Cross-sectional community studies have been undertaken at 3-year intervals in adults from 1966 to 1981 and again in 1990, and in children from 1968 to 1983. In 1992, 250 families with two or more children took part in a genetics study.[8] A follow-up survey of all available previous attendees (2279 men and 2730 women aged 20–89 years) was undertaken in 1994–1995 at centres in Perth and in Busselton (Box 1). A further survey of respiratory symptoms and illness, conducted in a random sample of adults and children, will commence in Busselton in mid-2005. Therefore, data from the Busselton health surveys are available for cross-sectional and longitudinal analysis.

At each survey, participants completed an extensive questionnaire on demography, cigarette smoking, alcohol intake, use of medications, previous and current medical history and current respiratory symptoms. Standing height and weight were measured, and skinprick tests to common inhaled allergens were performed. Spirometry was performed with dry spirometers (McDermott) from 1966 to 1969, wedge spirometers (Vitalograph) from 1972 to 1981 and pneumotachographs (Welch-Allyn) in the 1994–1995 survey. Forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC) were measured and expressed as percentages of predicted value.[9] In selected participants in the 1981 and 1990 surveys and all participants in the 1994–1995 survey, the dose of inhaled methacholine that provoked a 20% fall in FEV_1 (PD_{20}) was calculated to assess airway responsiveness. Atopy was defined as any skinprick test response that produced a weal of more than 2 mm, and airway hyper-responsiveness was defined as a PD_{20} of less than 4 μmol. Secular trends in the prevalence of doctor-diagnosed asthma were age-corrected. Cross-sectional data were analysed by decade only for participants who were older than 18 years at the time of the study.

Asthma

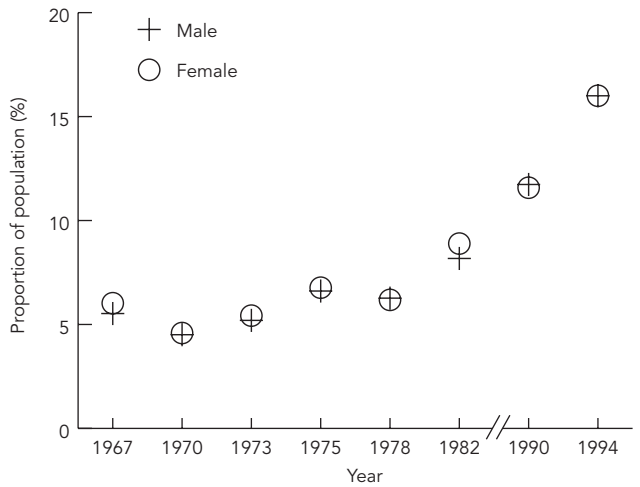
Age-corrected prevalence of asthma ever diagnosed by a doctor increased in adults from 8% in 1966 to 16% in the follow-up survey in 1994 (Box 2). The prevalence of doctor-diagnosed

West Australian Sleep Disorders Research Institute, Sir Charles Gairdner Hospital, Perth, WA.
Alan L James, MB BS, FRACP, MD, Respiratory Physician; and Clinical Assocate Professor, University of Western Australia.
Department of Public Health, University of Western Australia, Perth, WA.
Matthew W Knuiman, BSc, PhD, Deputy Director; Helen C Bartholomew, BSc, GradDipComp, Data Programmer and Analyst; Arthur (Bill) W Musk, MB BS, FRACP, PhD, Respiratory Physician.
Correspondence: Associate Professor Alan L James, West Australian Sleep Disorders Research Institute/Department of Pulmonary Physiology, Queen Elizabeth II Medical Centre, Level 5, G Block, Hospital Avenue, Nedlands, WA 6009. ajames@it.net.au

1 Doctor-diagnosed asthma in 5009 Busselton population health survey adults (aged 20–89 years) in 1994–1995

Age (years)	Men	Women
20–29	317 (27%)	333 (27%)
30–39	483 (15%)	617 (21%)
40–49	459 (14%)	564 (18%)
50–59	371 (14%)	400 (18%)
60–69	326 (13%)	398 (18%)
70–79	229 (15%)	312 (16%)
80–89	94 (20%)	106 (17%)
Total	2279	2730

2 Prevalence of doctor-diagnosed asthma in the Busselton population health surveys from 1966 to 1994*



* Data are age-corrected except for the follow-up survey of 1994–1995.

3 Smoking habits in Busselton population health survey adults (aged 20–89 years) in 1994–1995

	Men		Women	
Age (years)	Ever smoked	Former smoker	Ever smoked	Former smoker
20–29	44%	11%	44%	17%
30–39	52%	24%	50%	30%
40–49	54%	27%	41%	27%
50–59	59%	35%	38%	24%
60–69	71%	39%	41%	30%
70–79	71%	42%	45%	33%
80–89	77%	49%	36%	26%

asthma in the 1994–1995 survey decreased after age 30 and then remained steady even into the eighth decade (Box 1). At each decade, doctor-diagnosed asthma was more frequent in people with atopy and airway hyper-responsiveness. The prevalence of atopy decreased with age. Airway hyper-responsiveness, which was consistently more prevalent in women than men, decreased from around 20% for people in their 20s to around 10% for those in their 40s and 50s, then increased again to 20% for those in their 70s. The increased prevalence of airway hyper-responsiveness in older participants and in women may reflect the effects of decreased airway calibre. In the same survey, asthma — defined as airway hyper-responsiveness *and* wheeze within the last 12 months — decreased with age from 13% in men and 14% in women in their 20s to 6% (men) and 8% (women) in their 30s, and further to 2%–4% for those in their 60s and 70s. Wheeze (in the last 12 months) itself decreased from 31% (women) and 36% (men) in their 20s to 19% (women) and 20% (men) in their 30s, with little further change with age in either sex.

Lung function, asthma and cigarette smoking

FEV_1 as a percentage of the predicted value was lower in all participants with asthma compared with those without asthma, and the difference between these groups increased with age. In men aged under 55 years, the mean FEV_1 was 94% of the predicted value in those with asthma and 104% in those without asthma. For men aged 70–79 years, the corresponding values were 76% and 97%, respectively. In women aged under 55 years, the mean FEV_1 was 102% in those with asthma and 109% in those without asthma. For women aged 70–79 years, the corresponding values were 93% and 117%, respectively. In 1994, the rate of cigarette smoking (ever) was similar in men and women up to age 40, after which it increased with age in men but remained much the same in women (Box 3). In men, the proportion of those currently smoking was similar in participants with and without asthma in those aged under 55 years (22% v 26%) and aged over 55 years (27% v 24%). Likewise, in women, the proportion currently smoking was similar in those with and without asthma in the under-55-years age group (19% v 16%) and the over-55-years age group (8% v 6%). Compared with non-smoking participants without asthma, those with asthma or who smoked showed a greater decline in FEV_1 from age 18, and there was an additive effect on decline in FEV_1 in those with asthma who also smoked cigarettes.[10]

These data show an increase in doctor-diagnosed asthma with calendar time, but a decrease with age, suggesting a period effect, as was seen with cigarette smoking in men. In other words, although the prevalence of asthma (corrected for age) increased from 1966 to 1995, the greatest cumulative prevalence of asthma (doctor-diagnosed [ever]) was in younger participants in the cross-sectional study of 1994–1995. If the prevalence of asthma stayed the same across age groups, the cumulative prevalence might be expected to increase. Instead it remained much the same from 30 to 80 years. In addition, "current asthma" — defined as airway hyper-responsiveness and recent wheeze (within the last 12 months) — decreased, despite variations in the prevalence of wheeze and increases in other illnesses such as cardiovascular disease with age. This demonstrates the usefulness of other measures of airway abnormality, such as airway responsiveness. The prevalence of other diseases (cardiovascular diseases and diabetes) increased with age, and the cumulative effects of cigarette smoking were likely to confound the diagnosis and pathology of asthma in older age groups.

What we need to do in the context of the current evidence

We need to carry out further analyses of the current data with respect to changes in risk factors (including weight, smoking, occupation and atopy) and markers of oxidant load (such as white cell count) with time, as well as the effects of genetic factors on lung function, atopy and asthma at different ages. Future studies will need to characterise the nature of airway disease in older people with regard to inflammation and remodelling, as there may be confounding influences from cumulative exposures, asthma and other diseases. Airway wall remodelling may be increased in older people with asthma. The extent to which markers of inflammation (numbers of eosinophils and neutrophils, and exhaled nitric oxide) and factors related to airway remodelling (such as matrix metalloproteinases and tissue inhibitors of metalloproteinases, and matrix proteoglycans) differ in older people with asthma is largely unknown. Assessing these factors may help to more specifically identify older people with asthma in future studies.

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