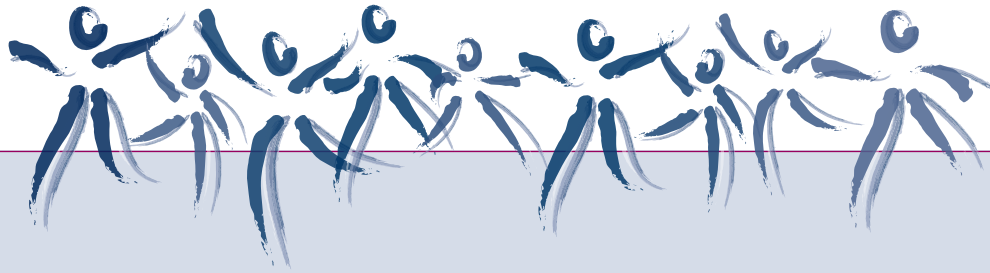
7. Tobacco smoke and occupation as risk factors for asthma



Key points

- People with asthma continue to smoke at least as commonly as people without asthma, despite the known adverse effects.
- The prevalence of smoking is higher among younger people with asthma than older people with asthma.
- Socioeconomic position is a strong determinant on the risk of smoking among people with asthma.
- An estimated 11% of children with asthma reside in homes where smoking occurs inside the home.
- Nearly 10% of adult-onset asthma is caused by occupational exposures and, hence, could be avoided if exposure to triggering agents in the workplace was eliminated.
- Occupational asthma is the one truly preventable form of the disease.

Introduction

While the underlying causes of asthma are still not well understood, environmental and lifestyle factors, as well as constitutional factors such as an allergic tendency, may increase the risk of developing asthma. Among those with the condition, airway narrowing and symptoms can be triggered by a wide range of exposures and other factors. These include specific allergens, such as house dust mites, pollens, mould spores, animal dander and occupational allergens, viral infections, irritants, such as tobacco smoke and other air pollutants, exercise and some food additives.

The environmental causes of asthma have been extensively investigated and reviewed (NSW Health Dept 1997; Peat 1994; Rural and Regional Health and Aged Care Services Division 2004). The subject remains controversial with conflicting evidence on the effects of exposure to pets and other allergen sources, the protective effects of breastfeeding and other aspects of diet and feeding, overweight and obesity, and the role of infections in childhood. A number of randomised controlled trials evaluating the effects of specific interventions for the prevention of asthma have been conducted but the findings are either negative or inconclusive. Without clear evidence of an important, avoidable causal role in asthma, these factors are not suitable targets for surveillance and have not been included in this report. Apart from environmental tobacco smoke exposure in children and smoking in adults, this publication does not report on these factors.

On the other hand, exposure to occupational allergens has been conclusively linked both to the development of asthma, *de novo*, and to progression of the disease. Since this is a potentially avoidable cause of asthma, exposure to occupational allergens and the occurrence of occupational asthma are important targets for surveillance.

In this chapter, we present data on smoking among people with asthma and exposure to environmental tobacco smoke among children with asthma. We also discuss occupational exposure as a risk factor for the development of asthma in adulthood.

7.1 People with asthma who smoke

The harmful effects of both active and passive smoking are well known. People with asthma who smoke have particular problems (Siroux et al. 2000) and find their asthma more difficult to control than non-smokers. In part, this may be because smoking impairs the effectiveness of inhaled corticosteroids (Chalmers et al. 2002), even at high doses (Pedersen et al. 1996). In addition, both smoking and asthma accelerate the rate of decline in lung function with age (James et al. 2005).

7.1.1 Prevalence

In 2004–05, the prevalence of smoking at least once a week in people aged 18 years and over was 24.5% among those with current asthma and 22.3% among those without current asthma (Table 7.1). Survey data from the states and territories confirm that the rate of smoking among people with asthma (15.8–35.5%) is the same, if not higher than, the rate among people without asthma (Table 7.1). The proportion of ex-smokers is generally similar among those with and without asthma.

Table 7.1: Smoking status among people with and without current asthma, most recent survey results, 2004–2007

Population (study)	Year	Age (years)	Smoking status	People with asthma		People without asthma	
				Rate (%)	95% CI	Rate (%)	95% CI
Australia (1)	2004–05	18 and over	Current smoker	24.5	22.3–26.7	22.6	21.9–23.4
			Ex-smoker	31.6	29.1–34.0	29.8	29.0–30.6
			Never smoked	43.4	40.6–46.1	47.2	46.3–48.1
				(*n* = 2,053)		(*n* = 17,448)	
New South Wales (2)	2006	16 and over	Current smoker	19.0	16.1–21.8	n.a.	n.a.
				(*n* = 1,522)			
Victoria (3)	2006	18 and over	Current smoker[(a)]	15.8	12.4–19.3	21.1	19.6–22.5
			Ex-smoker	22.1	18.4–25.7	24.4	23.0–25.7
			Non-smoker	62.1	57.5–66.8	54.6	52.9–56.3
Queensland (4)	2006	18 and over	Daily smoker	30.4	23.4–38.4	15.4	13.2–17.8
			Occasional smoker	5.1	2.4–10.2	4.7	3.5–6.4
			Ex-smoker	18.7	13.9–24.6	27.5	25.0–30.2
			Ex-smoker (tried few times)	6.3	3.6–10.8	12.1	10.2–14.3
			Never smoked	39.6	32.2–47.6	40.4	37.4–43.4
				(*n* = 215)		(*n* = 1,305)	
Western Australia (5)	2006–07	16 and over	Current smoker	15.8	11.7–21.1	15.8	14.4–17.4
			Ex-smoker	14.5	11.1–18.8	15.2	13.9–16.6
			Never smoked	69.7	63.8–75.0	69.0	67.1–70.8
				(*n* = 619)		(*n* = 5,312)	
South Australia (6)	2006–07	16 and over	Current smoker	17.1	14.6–19.9	16.7	15.7–17.8
			Ex-smoker	36.3	33.0–39.8	35.8	34.5–37.2
			Never smoked	46.6	43.0–50.1	47.4	46.0–48.8
				(*n* = 764)		(*n* = 4,935)	

n.a. Not available

(a) Includes people who reported smoking daily or occasionally.

Note: CI = confidence interval.

Sources: (1) Australian Centre for Asthma Monitoring (ACAM) analysis of the Australian Bureau of Statistics (ABS) National Health Survey 2004–05 confidentialised unit record files; Data excludes people who smoked less than weekly (0.6% of people with asthma and 0.4% of people without asthma). (2) New South Wales (NSW) Population Health Survey, Centre for Epidemiology and Research, NSW Health; (3) Department of Human Services, Victorian Population Health Survey 2006 (unpublished data); (4) Queensland Omnibus, Epidemiology Services Unit, Queensland Health; (5) WA Health and Wellbeing Surveillance System, Epidemiology Branch, Department of Health, Government of Western Australia; (6) South Australian Monitoring and Surveillance System (SAMSS), Population Research and Outcome Studies, Health Intelligence, Department of Health, Government of South Australia.

A South Australian study incorporating over 10 years of aggregated omnibus data found that asthma was associated with ex-smoking status. The prevalence of asthma among ex-smokers was 1.29 times (95% CI 1.15–1.44) as high as the prevalence of asthma among non-smokers (Wilson et al. 2006). Furthermore, the prevalence of asthma among female smokers was 1.27 times (95% CI 1.08–1.51) as high as the prevalence among non-smokers. Similarly, among males, the prevalence of asthma among ex-smokers was 1.47 times (95% CI 1.20–1.80) as high as the prevalence among non-smokers.

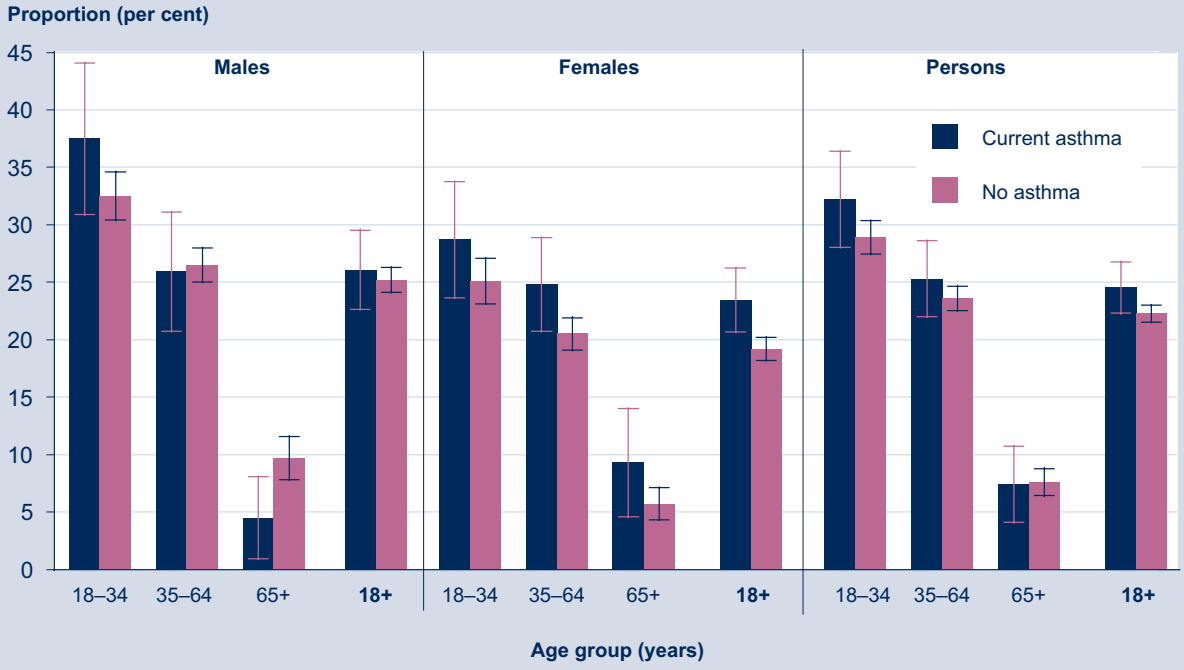
It is clear that many people with asthma continue to smoke. However, it is also possible that the very high rates of smoking (and particularly ex-smoking status) reflect the causal pathway. In other words, some people may have asthma-like symptoms or have been diagnosed with asthma due to the adverse effects of smoking.

7.1.2 Population subgroups

Age and sex

The prevalence of current smoking in adults decreases with age but, among men, the prevalence of ever having smoked increases with age. As a consequence, and as expected, a far higher proportion of older people, particularly men, are ex-smokers (ACAM 2007a). In 2004–05, nearly 38% of young men aged 18–34 years with asthma continued to smoke despite their illness (Figure 7.1).

The relatively high, or at least similar, rates of smoking in people with asthma compared to people without asthma is observed in both males and females (Figure 7.1). Among adult females, 23.5% of those with asthma compared to 19.2% of those without asthma reported being current smokers. Among adult males, 26.1% of those with asthma were smokers compared to 25.2% of those without the condition.



Note: Age-standardised to the Australian population as at June 2001.

Source: Australian Centre for Asthma Monitoring (ACAM) analysis of Australian Bureau of Statistics (ABS) 2004–05 National Health Survey confidentialised unit record files.

Figure 7.1: Prevalence of current smoking among adults aged 18 years and over, by asthma status, age group and sex, 2004–05

Socioeconomic disadvantage

The prevalence of smoking among people with asthma in 2004–05 was over three times higher (40.0%) among those living in more socioeconomically disadvantaged areas than among those living in less disadvantaged localities (12.4%) (rate ratio 3.2; 95% CI 2.4–4.2) (Figure 7.2). This differential was less marked among people without asthma (rate ratio 2.0; 95% CI 1.8–2.2).

Among those living in the most disadvantaged localities, the prevalence of smoking among people with asthma (40.0%) was substantially higher than that observed among people without the condition (31.0%). However, there were no statistically significant differences in the prevalence of smoking among people with and without asthma living in the least disadvantaged areas.

Notes: Age-standardised to the Australian population as at June 2001; SEIFA = Socio-economic Indexes for Areas.

Source: Australian Centre for Asthma Monitoring (ACAM) analysis of Australian Bureau of Statistics (ABS) 2004–05 National Health Survey confidentialised unit record files.

Figure 7.2: Prevalence of current smoking among adults aged 18 years and over, by asthma status and socioeconomic status, 2004–05

7.2 Passive smoke exposure in children with asthma

Exposure to environmental tobacco smoke, commonly referred to as 'passive smoke', in childhood is a risk factor for the development of asthma symptoms and also for the worsening of pre-existing asthma. It has been shown that exposure to environmental tobacco smoke increases the risk of wheezing illness in young children (Martinez et al. 1992; Young et al. 2000) and that the association between exposure and childhood wheezing illness is most consistent at high levels of environmental tobacco smoke exposure (NHMRC 1997). These findings are supported by evidence from international studies which conclude that parental smoking is associated with more severe asthma in children (Pattenden et al. 2006; Strachan & Cook 1998) and that exposure to environmental tobacco smoke after birth is a likely cause of wheezing or other acute respiratory illness in young children (Strachan & Cook 1997).

Cohort studies have shown that children with pre-existing asthma who are exposed to environmental tobacco smoke have increased morbidity and asthma symptoms (Murray & Morrison 1989), more frequent exacerbations (Chilmonczyk et al. 1993), more severe asthma symptoms (Murray & Morrison 1993; Strachan & Cook 1998), impaired lung function (Chilmonczyk et al. 1993; Murray & Morrison 1989) and increased airway reactivity (Murray & Morrison 1989; Oddoze et al. 1999) or peak flow variability (Fielder et al. 1999; Frischer et al. 1993). There is also evidence that children exposed to environmental tobacco smoke are more likely to attend emergency departments with asthma (Evans et al. 1987). It has been shown that prevention of indoor smoking leads to a reduction in hospital admissions in children with asthma (Gurkan et al. 2000). Recovery after hospitalisation, measured by use of reliever medication and number of symptomatic days, is also impaired in children exposed to passive smoke (Abulhosn et al. 1997).

A Brisbane study conducted between 1981 and 1998 showed that 14-year-old girls, but not boys, had an increased risk of having asthma symptoms (odds ratio 1.96; 95% CI 1.25–3.08) if their mother reported smoking heavily (defined as 20 or more cigarettes per day) both during pregnancy and 6 months after the birth of their daughter (Alati et al. 2006). Smoking during pregnancy was the most important risk factor.

There is some evidence that early life exposure to tobacco smoke may have long-term consequences. A recent study has reported that as much as 17% of adult-onset asthma is attributable to maternal smoking in childhood (Skorge et al. 2005). Several other international studies have reported associations with passive smoke exposure in childhood and asthma in adulthood. A Swedish study showed that the prevalence of adult asthma among people who never smoked was higher among subjects who had been exposed to environmental tobacco smoke as a child (Larsson et al. 2001). Findings from the European Community Respiratory Health Survey showed a higher prevalence of respiratory symptoms and poorer lung function among adults whose mother smoked during pregnancy or had childhood exposure to maternal smoking (Svanes et al. 2004).

Other studies have reported that subjects who were exposed to passive smoke during their childhood are more likely to take up smoking themselves (Cook & Strachan 1999; Larsson et al. 2001) and this may increase their risk of developing asthma.

The large body of evidence regarding the harmful consequences of passive smoke exposure has resulted in the introduction of smoking bans in many public areas. Recent legislative changes in Australia prohibit smoking in places such as bars, cafes and restaurants, shopping centres, entertainment venues and the workplace. South Australia recently became the first state to ban smoking in cars carrying children under the age of 16 years. In New South Wales in 2006, 88% of adults reported that their car was smoke-free (Centre for Epidemiology and Research 2007). Unfortunately, young children, who are most vulnerable to the effects of passive smoke exposure, are most likely to be exposed to passive smoke in their home, where smoking bans do not apply. There is evidence, though, that the proportion of homes in which smoking was *not permitted* inside the house increased in Australia from 71.6% in 1999 to 80.1% in 2004 (Valenti et al. 2005).

This section provides data on the proportion of children with asthma who live in homes where smoking occurs inside the home.

7.2.1 Exposure to passive smoke inside the home

Australian children with asthma continue to be exposed to environmental tobacco smoke in the home despite the known adverse effects. In 2004–05, 39.1% of children aged 0–14 years with asthma *lived with one or more cigarette smokers*. This proportion was marginally higher than that observed among children without asthma (36.2%). Furthermore, 11.0% of children with asthma were residing in homes where *smoking occurred inside the home* (Table 7.2). This rate was significantly higher than that observed for children without asthma (9.4%; $p = 0.04$). Results from health surveys conducted in Victoria, Western Australia and South Australia support these findings (Table 7.2).

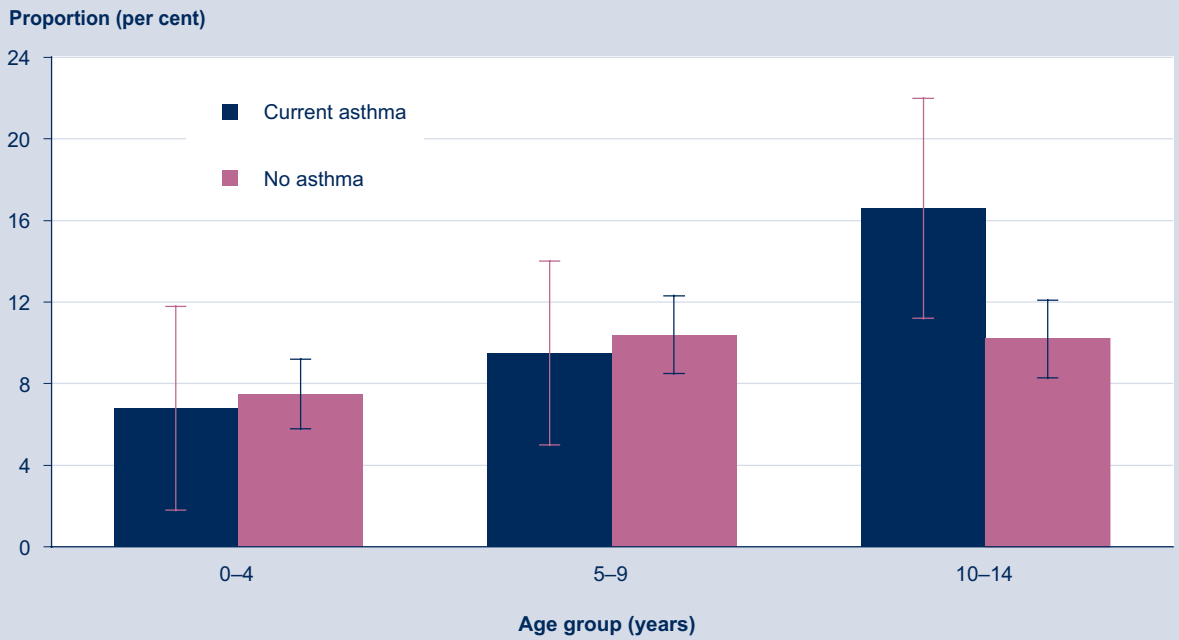
Table 7.2: Exposure to passive smoke among children, 2004–2007

Population (study)	Year	Age (years)	Household smoking status	Children with asthma		Children without asthma	
				Rate (%)	95% CI	Rate (%)	95% CI
Australia (1)	2004–05	0–14	One or more regular smokers usually smoke inside the house	11.0	8.1–13.9	9.4	8.3–10.4
Victoria (2)	2006	1 to under 13	No smoker	65.0	60.7–69.4	68.1	66.3–69.9
			Regular smoker/s	35.0	30.6–39.3	31.9	30.1–33.6
				(*n* = 652)		(*n* = 3,933)	
Victoria (2)	2006	1 to under 13	Always or usually smoke outside the house	88.1	83.0–93.1	85.8	83.4–88.2
			Sometimes smoke inside and sometimes smoke outside	9.4	4.8–14.1	10.0	8.1–11.8
			Usually smoke inside and sometimes smoke outside	2.5	0.3–4.6	4.2	2.6–5.7
				(*n* = 255)		(*n* = 1,287)	
Western Australia (3)	2006–07	Under 16	My home is smoke-free	97.3	94.0–98.8	96.0	94.1–97.3
			People occasionally smoke inside the house	2.2	0.9–5.6	2.0	1.2–3.3
			People frequently smoke inside the house	0.5	0.2–1.7	2.0	1.1–3.7
				(*n* = 127)		(*n* = 1,051)	
South Australia (4)	2006–07	2–15	My home is smoke-free	92.5	87.9–95.5	95.2	93.8–96.3
			People occasionally smoke inside the house	1.9	0.7–5.0	3.3	2.4–4.5
			People frequently smoke inside the house	5.5	3.1–9.8	1.5	0.9–2.4
				(*n* = 189)		(*n* = 1,087)	

Note: CI = confidence interval.

Sources: (1) Australian Centre for Asthma Monitoring (ACAM) analysis of Australian Bureau of Statistics (ABS) National Health Survey 2004–05 confidentialised unit record files; (2) Victorian Child Health Survey, Child Outcomes Monitoring, Statewide Outcomes for Children Branch, Office for Children, Department of Human Services; (3) Epidemiology Branch, Analysis and Performance Reporting Directorate, Department of Health, Government of Western Australia; (4) South Australian Monitoring and Surveillance System (SAMSS), Population Research and Outcome Studies, Health Intelligence, Department of Health, Government of South Australia.

High rates of exposure to environmental tobacco smoke were observed for children of all ages with and without asthma (Figure 7.3).

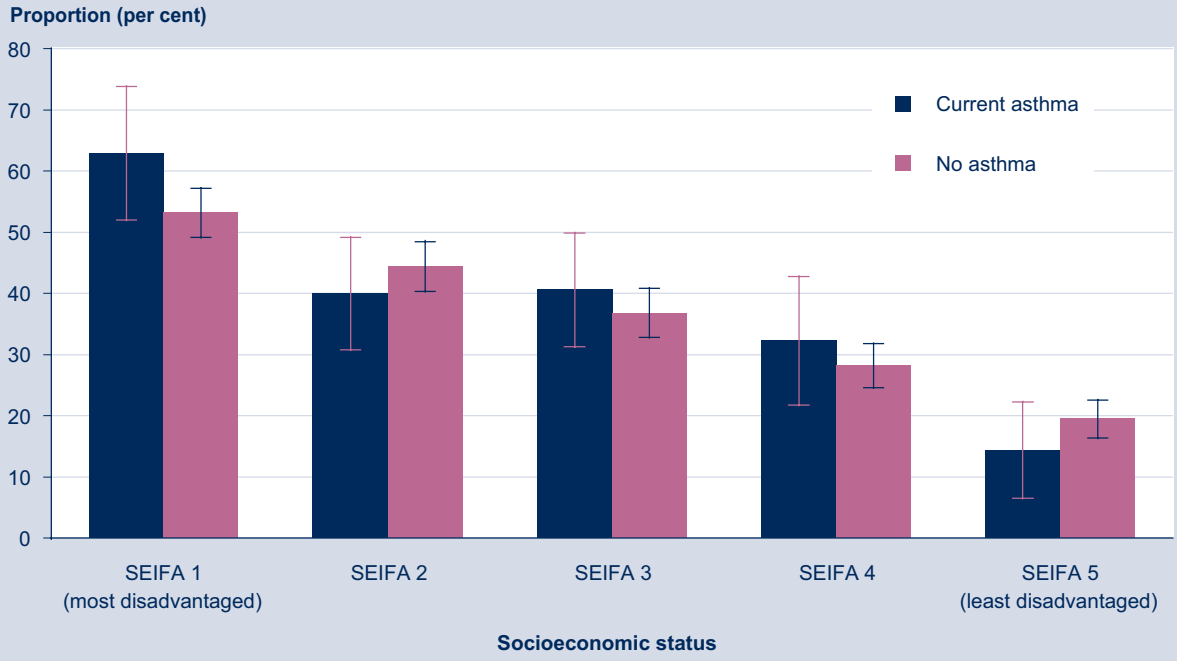


Source: Australian Centre for Asthma Monitoring (ACAM) analysis of Australian Bureau of Statistics (ABS) 2004–05 National Health Survey confidentialised unit record files.

Figure 7.3: Proportion of children aged 0–14 years residing with one or more cigarette smokers who usually smoke inside the house, by age group and current asthma status, 2004–05

Socioeconomic disadvantage

In the most socioeconomically disadvantaged localities, nearly two-thirds of children with current asthma reside with a smoker. This proportion declines to 14% in the least socioeconomically disadvantaged localities (Figure 7.4).



Notes: Age-standardised to the Australian population as at June 2001; SEIFA = Socio-economic Indexes for Areas

Source: Australian Centre for Asthma Monitoring (ACAM) analysis of Australian Bureau of Statistics (ABS) 2004–05 National Health Survey confidentialised unit record files.

Figure 7.4: Proportion of children aged 0–14 years with one or more cigarette smokers in the household, by socioeconomic status and current asthma status, 2004–05

7.3 Occupational asthma

Occupational asthma represents the most prevalent occupational lung disease in the developed world (Nicholson et al. 2005). The term refers to asthma caused, or made worse, by exposures in the workplace. International studies suggest that 9–15% of cases of asthma in adults of working age are either caused or aggravated by occupational factors (Nicholson et al. 2005).

There are over 400 substances that are recognised as triggers for asthma in the workplace including various chemicals used in paints, manufacturing and cleaning products, latex gloves, animals and dusts from grain, flour and wood (Nicholson et al. 2005). These agents pose most risk for people employed in the plastics, rubber and chemical industries, nurses, timber workers and welders, and jobs involving painting (particularly spray painting), dyeing, cleaning, baking and food processing, farming, laboratory work and working with animals (NAC 2006; Nicholson et al. 2005).

The importance of occupational asthma is that it is relatively common and it is preventable. A population-based study in Canada (Johnson et al. 2000) concluded that the removal of exposure to known triggers could prevent as much as 18% of adult-onset asthma in that country, although a

subsequent, similar study in Australia found a lower proportion of cases attributable to workplace exposures (see section on 'Prevalence of occupational asthma' below). Early removal from exposure is important for treatment and preventing persistent disease. Reducing or eliminating exposure to the triggering agent(s) will usually reduce the severity of symptoms or, in some cases of early intervention, it may eliminate symptoms completely. Persons who remain exposed are more likely to have persistent and troublesome asthma. The AIHW has recently published a review of occupational asthma (AIHW 2008e) that summarises known occupational risk factors for asthma, current knowledge about incidence and prevalence and approaches to prevention and disease monitoring, with particular reference to the Australian context. Here we present a brief coverage of those issues.

7.3.1 Current surveillance

Since occupational exposure represents a potentially avoidable cause of asthma, exposure to occupational allergens and the occurrence of occupational asthma are important targets for surveillance. Therefore, the prevalence of occupational asthma has been identified as one of the 24 national health indictors for asthma (AIHW: Baker et al. 2004). The intent of the indicator is to:

- monitor exposure to, and the impact of, occupational risk factors for asthma
- evaluate population health interventions to prevent the onset and exacerbations of asthma (in the occupational setting)
- monitor the provision of a safe environment for people with asthma.

In spite of its identification as an asthma indicator, there is no consistent, thorough and reliable scheme to monitor the prevalence of occupational asthma in Australia at the present time.

Conventional monitoring for chronic diseases is largely based on measures of late-stage events such as hospitalisation and mortality and on cross-sectional prevalence surveys. Unfortunately, both of these sources provide very limited and potentially biased evidence about the impact of occupational asthma.

Causal exposures are very rarely recorded in hospitalisation and mortality data and, hence, there is virtually no information on the contribution of occupational exposures to these outcomes of asthma. Furthermore, these outcomes represent only the 'tip of the iceberg' of this issue.

A more fundamental problem is that the disease may be transient and the main impact may be to cause someone to leave his or her job. People who find that their work is causing or aggravating asthma (or other symptoms) tend to seek alternative employment or leave the workforce altogether. Some cross-sectional surveys have been performed to estimate the prevalence of occupational asthma. These have generally asked respondents about previous employment or exposures that may have caused asthma or asthma-like symptoms. Australian data from two surveys are reported below in the section entitled 'prevalence of occupational asthma'.

Since some industries are at particularly high risk for cases of occupational asthma, there may be value in conducting surveillance in specific workplaces. However, workplace-based cross-sectional surveys are particularly likely to underestimate the burden of the disease since many of the affected workers will have left the workplace. The remaining workers will tend to be the healthy ones. This bias is a major problem in surveillance for occupational disease and is known as the 'healthy worker effect'.

In order to accurately estimate the impact of occupational asthma in the community or in a specific workplace, it is necessary to measure the incidence of asthma in a cohort followed over time. Well-conducted cohort studies will not be affected by the healthy worker bias described above. Some examples of measures of the incidence of occupational asthma in Australia are cited in the section below entitled 'Incidence of occupational asthma'.

7.3.2 Prevalence

It has been estimated that occupational exposures cause of 9.5% of cases of adult-onset asthma in New South Wales (Johnson et al. 2006). This estimate was based on data from a self-completed postal questionnaire administered to a randomly selected sample of adults in New South Wales. Information on adult-onset asthma and ever being employed in occupations identified as being of high risk for the development of occupational asthma were collected. The triggering agents associated with the greatest risk of adult-onset asthma were exposure to ammonia (odds ratio 2.54; 95% CI 1.72–3.78) and photographic development (odds ratio 2.25; 95% CI 1.04–4.85). One of the strengths of this study is its population-based design, which allows for the inclusion of people who have had occupational asthma and have left the workplace.

In 2001, 1.6% (95% CI 1.0–2.2%) of respondents with asthma in the NHS aged 15 years and over stated that their asthma was work-related. In 2004–05, the prevalence of work-related asthma among those with asthma was 2.2% (95% CI 1.5–2.8%). Among those aged 35–64 years, 3.1% (95% CI 1.9–4.3) of all asthma cases in 2004–05 were attributed to work while among those aged 15–34 years, 0.7% (95% CI 0.1–1.3) of people with asthma reported that their condition was work-related.

The estimate for the proportion of adult-onset asthma attributable to occupational exposures in New South Wales falls towards the lower end of the range observed in international studies (Nicholson et al. 2005).

7.3.3 Incidence

Population-based surveillance for incident cases of occupational asthma has been established in three Australian states.

The Surveillance of Australian Workplace-Based Respiratory Events (SABRE) is a voluntary notification scheme that has been in operation in Victoria and Tasmania since 1997 and in New South Wales since 2001. In this scheme, respiratory physicians, occupational physicians and, in the case of New South Wales, accredited general practitioners report newly diagnosed cases of occupational respiratory diseases.

Since the scheme started, the incidence of occupational asthma was 5 cases per million employed people per year in Tasmania and Victoria combined and 2 cases per million employed people per year in New South Wales (Hannaford-Turner et al. 2007). On an international scale, this is a relatively low incidence rate.

Unfortunately, the voluntary nature of this scheme means that this is almost certainly an underestimate of the true incidence. As there is no legislative requirement to report benign occupational lung disease (as there is for certain infectious diseases and for cancer, for example) and there is no comprehensive compensation scheme for people with occupational asthma, there is no incentive for patients or health-care professionals to notify new cases. Furthermore, the notification scheme does not impose a standard for the diagnosis of occupational asthma. Hence, estimates of the incidence of occupational asthma based on these notifications may be inaccurate. The net effect is likely to be an underestimate.

The lower incidence in New South Wales compared with other settings may be attributable to underestimation. For example, in Finland, physicians are required by law to report all cases of known or suspected work-related disease to a national register. In addition, all employees in Finland must carry insurance for occupational diseases. Reports of disease and accident diagnoses, recorded by the insurance companies, are provided to the national register. It has been reported that the mean annual incidence of occupational asthma in Finland, where all cases of occupationally-related disease are captured in the Finnish Registry of Occupational Diseases, is 174 cases per million employed workers (Karjalainen et al. 2000). International estimates of the incidence of occupational asthma average around 47 cases per million workers (range 12–174 cases per million workers) (Karjalainen et al. 2000; Nicholson et al. 2005).

7.3.4 Improving surveillance

Cross-sectional community-based population studies do provide some valuable information on the prevalence of occupational asthma in the population. However, they are only successful if they are truly population-based—that is, they include individuals who have left the workforce and they include a detailed historical record of respondents' occupational exposures. Since this is very time-consuming, it is best achieved using a nested survey design in which this information is only sought from respondents with adult-onset asthma and a sample of controls without asthma.

Workplace and community surveillance for incident cases is the 'gold standard' for monitoring the impact of occupational asthma and also for managing the problem in real time. For the reasons outlined above, community-level surveillance has been difficult to achieve in Australia. Improved rates of notification require incentives in the form of a legislative requirement or, preferably, a link to compensation payments, and the application of standards for the diagnosis.

Summary

Despite the known adverse effects of smoking, people with asthma continue to smoke at least as commonly as people without asthma. Smoking is more common among younger people with asthma than older people with asthma. These results imply that developing asthma does not immediately encourage people to quit smoking, which probably reflects the highly addictive qualities of nicotine products. It is also plausible that some of the observed association between smoking and self-reported asthma is attributable to the association with smoking-related respiratory disease, including chronic obstructive pulmonary disease.

Socioeconomic position has an important effect on the risk of smoking among people with asthma. Although smoking is more common in all people living in more disadvantaged localities than those living in more advantaged localities, this discrepancy is much greater in the population with asthma. Disadvantaged localities may benefit from a targeted approach to health promotion aimed at reducing smoking.

Children with asthma continue to be exposed to passive smoke in their home. Almost 40% of children with asthma lived with smokers and an estimated 11% of children with asthma were living in homes where smoking occurred inside the home. There was a substantial socioeconomic gradient in exposure to environmental tobacco smoke. Children living in areas where socioeconomic position was lower were more likely to be exposed to environmental tobacco smoke, and this association was strongest among children who had asthma.

Asthma caused or aggravated by exposures at work is the one truly preventable form of the disease. It is estimated, based on data from New South Wales, that around 9.5% of adult-onset asthma is caused by occupational exposures and, hence, could be avoided if exposure to triggering agents in the workplace was eliminated. There are limited surveillance data on occupational asthma in Australia and there is a need to improve the completeness of notification to existing voluntary schemes in Victoria, Tasmania and New South Wales.