

CHRONIC RESPIRATORY DISEASES IN SOUTH AFRICA

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This chapter is dedicated to the memory of Neil Walton White,^c 1954-2004

1. INTRODUCTION

Scope

Epidemiological research into chronic respiratory disease in South Africa has grown over the last decade with the recognition of the burden of such disease on the health of the population. The aim of this chapter is to review the findings of such published research, mainly from 1994-2005, and their implications for prevention. The development of South African guidelines to improve the management of these diseases is also covered.

The most common chronic respiratory diseases in South Africa are asthma and chronic bronchitis/chronic obstructive pulmonary disease (COPD), and these are covered in detail. In addition, attention is given to air pollution and occupation as risk factors for chronic respiratory disease in South Africa. The chapter ends with recommendations for policy action and research.

Definitions

The lack of standard definitions for COPD and asthma historically makes it difficult to compare different studies and sources of information. A feature of the last decade has been the development of international consensus guidelines on the definition of asthma and COPD in an attempt to standardise diagnosis and treatment, notably the Global Initiative for Chronic Obstructive Lung Disease (GOLD)¹ and the Global Initiative for Asthma (GINA).²

GOLD has defined COPD as "a disease state characterised by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases".¹ The guidelines refer to both typical symptoms and a history of exposure to risk factors as a basis for suspecting the presence of the condition, while spirometry is required to confirm the diagnosis. Chronic bronchitis, i.e. chronic cough with phlegm, may be present with or without airflow limitation. The GOLD definition treats chronic bronchitis without airflow limitation as "Stage 0" ("at risk"), while COPD proper requires evidence of airflow limitation, with or without symptoms.¹ A post-bronchodilator ratio of forced expiratory volume in one second (FEV₁) to forced vital capacity (FVC) of less than 70% is the necessary spirometric criterion for COPD proper, while the value of post-bronchodilator FEV₁ as a percentage of its predicted reference value determines the staging of severity of COPD from I ("mild") to Stage IV ("very severe").³

A recent consensus document by the European Respiratory Society and American Thoracic Society served to unify global definitions and solidified the departure from older definitions by recognising COPD as a disease resulting from an inflammatory process.⁴

For purposes of questionnaire-based epidemiological surveys the original British Medical Research Council definition of chronic bronchitis - "cough with phlegm for three successive months for at least two successive years" - has been frequently used.⁵ This definition has the advantage of being relatively simple and now has sufficiently long usage to allow population prevalences to be compared. The disadvantage, in the absence of spirometry, is that the prevalence of COPD cannot directly be inferred from the prevalence of chronic bronchitis.⁶

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GINA defines asthma as “a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role. The chronic inflammation causes an associated increase in airway hyperresponsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment.”²

As in the clinical setting, it is not easy to distinguish clearly between asthma and COPD in epidemiological studies, particularly in smokers and older people. Epidemiological studies rely heavily on symptom questionnaires. While some of these questionnaires have been standardised and validated among adults and children in developed countries, their validity in South Africa has not been established. In particular, studies specifically comparing standardised questionnaires with spirometry are lacking. However, for asthma, questionnaire-based symptom definitions have been shown elsewhere to perform better in studying comparative prevalence than an objective test such as non-specific bronchial hyperresponsiveness (BHR). This is because questionnaires offer an optimal balance of sensitivity (identifying cases) and specificity (excluding non-cases), whereas tests of BHR tend to be specific but not sensitive.⁷

Burden of mortality and morbidity

Worldwide in 2001, COPD was the fifth most common cause of death,⁸ responsible for 4.7% of deaths and 2.0% of disability adjusted life years (DALYs).⁹ A 30-year projection from 1990 predicted a steady rise in the number of COPD deaths to the third most common cause worldwide by 2020.¹⁰ Most of the projected DALY burden will fall on developing countries.¹⁰ The actual burden in middle and low income countries is difficult to assess because of under-recognition of COPD as well as the underdevelopment of routine data systems and a paucity of surveys.^{11,12} The reasonable prediction is that the prevalence of COPD will rise as the populations age in these countries and as tobacco use increases.¹² There is already evidence from East Asia that the actual burden of disease is higher than the WHO estimates.¹² There is, however, a scarcity of data from Africa.

In South Africa, respiratory disease as a group, but excluding tuberculosis, was ranked as the seventh most important cause of DALYs (4.7%) in 2000.¹³ COPD alone was responsible for 2.3% of all deaths in 2000, although only 1.1% of years of life lost, indicating its concentration at older ages.¹⁴ The true proportion of deaths to which COPD contributes is almost certainly higher, since COPD is likely to be under-certified as an underlying cause where the immediate cause is stated as respiratory infection.

Asthma is not ranked among the top 15 causes of death globally, as it is largely an adequately controlled disease in developed countries. It ranked only at 30th as a cause of DALYs in 2001 (WHO, 2002),⁹ responsible for one percent of DALYs lost (comparable to diabetes)² and 0.4% of all deaths.⁹ In South Africa, asthma ranked somewhat higher, at 13th as a cause of death (1.5% of all deaths) and 18th as a cause of years of life lost (0.9%).¹⁴

It is striking that while South Africa is ranked 25th worldwide in the prevalence of asthma (estimated at 8.1% over all ages), it runs fourth in asthma mortality rates in the 5-34-year age group, at approximately 1.5 per 100 000, falling between Turkmenistan and Uzbekistan.¹⁵ Similarly, the asthma case fatality rate in South Africa is reported as being the fifth highest in the world at 18.5 per 100 000 asthmatics.¹⁵

Causes of COPD and asthma

COPD as understood in developed countries is primarily caused by tobacco smoking, and prevention activities are appropriately directed at tobacco control. By contrast, the causes of asthma are still poorly understood although research has become increasingly rich in hypotheses. Health system activity in asthma is thus largely directed at secondary prevention through early diagnosis and appropriate management.

In South Africa, the pattern of asthma and COPD reflects the structure of society with its high degree of industrialisation, high rates of smoking among some sections of the population, extensive urban and rural poverty, and the persistence of epidemic infectious diseases. In addition to tobacco smoking as a cause of COPD, post-tuberculous lung damage, occupational exposures, indoor and outdoor domestic air pollution and cannabis smoking have been identified as playing a role in chronic airflow limitation in South Africa, and are discussed in this chapter. Causes of asthma are more complex. Childhood asthma has to date been more closely studied than adult asthma, and gender, family history, urban residence, proximity to sources of pollution, allergic sensitisation, body mass index (BMI) and passive smoking have all emerged as predictors of asthma in cross-sectional studies of prevalence. However, it should be borne in mind that cross-sectional studies have difficulty distinguishing underlying causes of asthma from aggravating factors.

Paradoxically, infection may play an important role in determining the pattern of chronic “non-communicable” lung disease in a country such as South Africa. For example, a history of pulmonary tuberculosis has been shown to be a significant predictor of chronic respiratory symptoms in the general population.^{16,17} By contrast, a decline in infectious challenge in early childhood has been hypothesised to explain the rise of allergic asthma incidence over time and the positive rural to urban gradient of asthma.^{18,19}

2. COPD

Prevalence and risk factors

Since Wicht *et al.*²⁰ published their study on the “Diffuse Obstructive Pulmonary Syndrome” in 1977, few studies have investigated COPD in general populations in South Africa (Table 11.1). There is only one published study and one abstract reporting spirometry.^{21,22} Table 11.1 thus lists mainly studies which have reported self-reported chronic bronchitis as an outcome.

The South African Demographic and Health Survey 1998 (SADHS)²³ was the first study to provide national prevalence figures for chronic bronchitis,¹⁶ viz. 2.3% in men and 2.8% in women > 14 years of age, predictably lower than found in workforces in Africa exposed to dust and other respiratory hazards.²⁴ Prevalences in the age group 15-43 years in the SADHS were low compared to those from European studies, while comparable national prevalences were not available for the older group. The slight female excess was surprising given that national figures for current smoking in the SADHS were 42% among men and 11% among women.²³

Table 11.1. Prevalence of COPD and chronic bronchitis in population surveys in South Africa

Study (yr published)	Population, (N, age range)	Outcome measure	Age stratum (yr)	Prevalence (%)	
				Male	Female
Wicht ²⁰ (1977)	Northern Suburbs, Cape Town (507, 20-80 yr)	FEV ₁ /VC < 70%	< 40	9.0	14.0
		Chronic bronchitis	> 40	37.0	21.0
			< 40	4.3	1.8
			> 40	12.2	4.7
Nriagu (1999) ²⁶	South-central Durban* (693, > 17 yr)	Chronic phlegm > 3 months in past year	> 17	31	
Jithoo ^{21,27} (2003, 2005)	Ravensmead, Uitsig, Cape Town (3 512, ≥ 15 yr)	Chronic bronchitis	≥ 15	7.5	
		Chronic bronchitis	≥ 40	12.6	9.2
		COPD**	≥ 40	30.3	19.0
Ehrlich ¹⁶ (2004)	National sample (13 468, > 14 yr)	Chronic bronchitis	15-43	1.5	1.9
			≥ 44	4.2	4.3

FEV₁: Forced expiratory volume in one second. VC: Vital capacity

* Area in vicinity of petrochemical refineries and other industry

** Jithoo²²

In statistical modelling to mutually adjust the effects of different risk factors, smoking and a past history of tuberculosis emerged as predictors of chronic bronchitis in both men and women.¹⁶ Reported occupational exposure (to smoke, dust, fumes, strong smells, or work underground in a mine, > 1 year) was associated with chronic bronchitis in men, while domestic exposure to smoky fuels (coal, wood or other biomass) was associated with chronic bronchitis in women. Further analysis (R Ehrlich, unpublished data) confirmed that the highest stratified prevalence of chronic bronchitis, adjusted for age and smoking, occurred in rural African* women (3.2%). As the prevalence of smoking daily or occasionally in this group was only 7.5%, exposure to domestic fuel pollution is the most likely explanation for this.

The results of two community studies emphasise the importance of environmental conditions in the aetiology of chronic bronchitis. Jithoo *et al.*,²¹ studied a low- to middle-income community in Cape Town with very high prevalences of current smoking (58.9% in men and 42.9% in women) and history of tuberculosis (9.7%). The prevalence of chronic bronchitis was 9.2% in women and 12.6% in men over age 40 years.²¹ These local data report prevalences that are much higher than the

* Race groups are reported as used in the original data source and are understood primarily as reflecting a complex social stratification.

national prevalence and are reflective of the high smoking prevalences, heavy tuberculosis burden and poor working and living conditions in this community. The study by Nriagu *et al.*,²⁶ which found very high respiratory symptom prevalences in a south-central Durban community, was carried out because the population had identified pollution by local industry as a source of respiratory illness.

Population studies are useful for estimating population attributable fractions, i.e. the proportion of the disease occurrence in the population attributable to the risk factor, on the assumption that the association is causal. Table 11.2 from the study by Ehrlich *et al.*,¹⁶ illustrates that while tobacco remains a major contributor to chronic bronchitis in South Africa, the combined burden of past tuberculosis and occupational exposure in men is equal to that of smoking, while in women the combined burden of past tuberculosis and domestic fuel exposure exceeds that of smoking.

In the Cape Town study of chronic bronchitis,²¹ the population attributable fraction for tobacco was 30.7% for smoking 1-14 cigarettes per day and 14.5% for smoking ≥ 15 cigarettes per day. In addition, occupational exposure contributed 22.2%, cannabis smoking 19.3%, and tuberculosis 5.5%. Smoky domestic fuel use was rare as most households used electricity. The population attributable fraction for cannabis is striking. Research on the impact of cannabis on respiratory disease is lacking in South Africa, and the significant pulmonary toxicity of cannabis smoking is very poorly publicised. Despite the illegal status of the drug it is widely used. Twelve percent of the sample in the Cape Town study population reported ever having used the drug and seven percent reported current use. As self-reported usage is likely to be understated owing to the sensitive nature of such questions, it is suspected that true prevalence of usage is slightly higher.

Table 11.2. Population attributable fractions for modifiable risk factors for chronic bronchitis in South Africa¹⁶

	Men (N=5 671)			Women (N=7 929)		
	P	POR	PAF	P	POR	PAF
Past tuberculosis	2.9	4.9	10	2.0	6.6	10
Occupational exposure	26.8	1.6	14	-	-	-
Smoky domestic fuel	-	-	-	33.8	1.5	14
Current smoking, 1-14/day	30.6	1.5	13	8.7	2.3	10
Current smoking, 15+/day	8.7	2.5	12	1.9	1.8	1

P: Population prevalence of exposure. POR: prevalence odds ratio. PAF: population attributable fraction (rounded)

Both the SADHS²³ and the Cape Town^{21,27} studies found tuberculosis to be associated with chronic bronchitis. The association of past tuberculosis with both chronic cough and sputum and impairment of lung function has been noted locally in South African gold miners.^{28,29} The loss of lung function increases incrementally with the number of previous episodes of tuberculosis.²⁸ Post-tuberculous lung function loss does not seem to meet the GOLD definition of the airflow limitation of COPD as "usually ... associated with an abnormal inflammatory response of the lungs to noxious particles or gases".¹ However classified, tuberculosis appears to be an important cause of chronic lung disease in South Africa.

Guidelines for COPD management

South African consensus guidelines for the management of COPD were published in 2004,³⁰ incorporating GOLD recommendations and a recognition of the cost constraints on drug prescription in South African health care. There was also recognition of domestic and occupational exposures and previous lung infections, such as tuberculosis, as risk factors for COPD in addition to tobacco consumption. Treatment guidelines were directed at prevention of exacerbations and improvement of quality of life of patients with COPD as well as at smoking cessation efforts. Recommended treatment modalities reflected the development of long-acting beta₂ agonist and anticholinergic drugs and emerging evidence on a limited role for inhaled corticosteroids in the treatment of COPD.

Spirometry is central to the diagnosis and management of COPD. An investigation in 1991 found very poor quality spirometry practice among 45 medical practitioners, including 26 physicians.³¹ Knowledge of international spirometry standards, spirometer working mechanisms and calibration ranged from poor to completely unsatisfactory in most practices, as did the standard of test quality assessment and interpretation of results. Quality control of spirometry has thus long been a concern and a number of guidelines on the subject have been published, including two applicable to the occupational health arena where routine spirometry is common.³²⁻³⁴ The reviews concur on the

elements of good quality spirometry, viz. an understanding of the indications for the test, calibrated equipment meeting international performance standards, trained health personnel performing the test, adequate subject preparation and proper interpretation. There are no recent published data on what proportion of routine spirometry outside of specialist centres meets these quality criteria.

3. ASTHMA

Prevalence and risk factors in children and adolescents

Asthma in children and adolescents has received more attention than adult asthma because of lack of confounding by COPD or tobacco-related disease in children, and because school studies provide access to children and allow good estimates of community prevalence (Table 11.3).

The most common questionnaire measures reported are wheeze in the past 12 months and self-reported asthma ("asthma ever") whether based on a doctor's diagnosis or not. Answers to these questions were in fact found to be the most reproducible of a number of questions in a study of Cape Town schoolchildren.³⁵ From Table 11.3 it is striking that the prevalence of self-reported asthma in South Africa is relatively consistent across studies – between 10% and 13%. There is much more variation in wheeze in the past 12 months, at least partly attributable to different age groups studied, as the prevalence of wheezing declines with age.

The CHAMP (Chestiness in Childhood Asthma in Mitchell's Plain) study is the only local study to report prevalence in pre-school children.⁴² The study found a high prevalence of symptoms in children aged 2-6 years, with 36.7% reporting wheeze in the past 12 months. Asthma diagnosis was a little more common in this pre-school age group (13.1%) than in the school-going group (11.2%). Prevalence of diagnosis was slightly higher than reported from a pre-school study in the United Kingdom³⁶ but much lower than that reported in an Australian study.³⁷ The threshold for paediatric diagnosis of asthma in pre-schoolers is thus likely to vary between countries for given symptom prevalences. This is perhaps not surprising as the interpretation of wheezing in pre-school children is more difficult than in schoolchildren, given the various forms of early wheezing, including transient wheezing, viral induced wheezing and persistent wheezing with atopy.³⁸

Table 11.3. Prevalence of reported asthma and recent wheezing in child and adolescent populations in South Africa

Study (yr published)	Population (N, age range)	Outcome measure	Prevalence (%)	
Burr ⁴⁰ (1994)	Southern Suburbs, Cape Town (1 180, 12 yr)	Wheeze past 12 months	17.8	
		Asthma ever	11.5	
Ehrlich ³⁵ (1995)	Mitchell's Plain, Cape Town (1 955, 6-10 yr)	Wheeze past 12 months	26.8	
		Asthma ever	10.8	
Nriagu ²⁶ (1999)	South-central Durban* (367, < 17 yr)	Shortness of breath with wheeze past 12 months	16.0	
		Asthma ever	10.0	
Poyser ⁴¹ (2000)	Cape Town (5 178, 13-14 yr)	Wheeze past 12 months	16.0	
		Wheeze past 12 months (video**)	6.4	
		Asthma ever	13.3	
Pather ⁴² (2002)	Mitchell's Plain, Cape Town (17 446, 2-15 yr)	Wheeze past 12 months 2-6 yr	36.7	
		Asthma ever 7-12 yr	27.5	
			2-6 yr	13.1
			7-12 yr	11.2
White ⁴³ (2003)	Northwest suburbs, Cape Town* (3 162, 9-15 yr)	Wheeze past 12 months	33.0	
		Wheeze past 12 months (video**)	18.3	
		Asthma ever	23.7	
Obihara ⁴⁴ (2005)	Low-income area, Cape Town (861, 6-14 yr)	Asthma ever	12.3	

* Residential areas in close proximity to petrochemical refineries and other industry

** Respondents wrote down answers in response to video scenes of symptomatic children

The International Study of Asthma and Allergy in Childhood (ISAAC) is a worldwide study using a common method to study asthma prevalence and risk factors, including a video prompted questionnaire of scenes depicting asthma symptoms.³⁹ Cape Town participated in Phase I³⁸ and Phase II (H. Zar, unpublished data), studying a random sample of Cape Town adolescents.⁴¹ In international comparison of the ISAAC Phase I results, the prevalence of wheezing in this Cape Town population lay in the mid-range internationally.³⁹

Tests of non-specific bronchial hyperresponsiveness (BHR) (Table 11.4) offer an objective measure of a trait associated with asthma, although BHR is not a necessary condition for the identification of asthma for epidemiological purposes.⁷ Prevalences of BHR should thus not be compared directly with questionnaire prevalences. Three findings stand out in Table 11.4. The first is the positive rural urban gradient, confirming that asthma is at least partly a disease of urbanisation.⁴⁵⁻⁴⁷ The second is the secular increase in the prevalence of BHR between the late 1970s⁴⁵ and the late 1990s,⁴⁷ although part of the difference may be attributable to a more liberal criterion for positivity in the latter study. This increase appears to have been relatively much greater in rural children. Finally, there is the remarkably high prevalence among Durban schoolchildren.⁴⁸ This is apparently attributable to their living in proximity to industrial and petrochemical industry emissions, but the absence of a control area should be noted.

On adjusting the rural urban gradient in BHR (to exercise) for a number of potential confounders, Calvert⁴⁹ showed this gradient could be fully explained by a combination of greater atopy, evidence of *Ascaris* infection, greater BMI and the absence of an animal in the house among urban children (Table 11.5). He speculated that an increase in nutritional status as reflected in a higher BMI that accompanied urbanisation allowed a greater expression of atopy in the form of skin prick test reactivity.

Independently, the protective effect of having animal presence in the house (in this case farm animals in the rural sample) is consistent with a version of the "hygiene hypothesis", viz. that the microbial environment, including the presence of endotoxin, rather than specific clinical infections, is important in the programming of the immune system in early life.¹⁸ Obihara *et al.*,⁴⁴ in a cross-sectional study of low-income Cape Town children and adolescents found tuberculin skin test response (> 10 mm) to be inversely related to the history of allergic disease in general (asthma, hayfever and eczema). This "protective" effect of tuberculin response was strongest for hayfever (odds ratio 0.40, 95% CI: 0.21-0.77).

Table 11.4. Prevalence of non-specific bronchial hyperresponsiveness in child and adolescent populations in South Africa

Study (yr published)	Population (N, age range)	Outcome measure	Prevalence (%)
Van Niekerk ⁴⁵ (1979)	Transkei (671, 6-9 yr); Gugulethu, Cape Town (694, 6-9 yr)	≥ 15% ↓ in FEV ₁ or PEF after exercise	0.14 rural 3.17 urban
Vermeulen ⁵⁰ (1990)	Transkei (1 014, 8-16 yr)	≥ 20% ↓ FEV ₁ after histamine	14.2 rural
Terblanche ⁵¹ (1990)	Northern suburbs, Cape Town (1 192, 6-19 yr)	> 10% ↓ in FEV ₁ after exercise	5.1 urban
Nagel ⁵² (1992)	Southern suburbs, Cape Town (1 180, 12 yr)	> 15% ↓ FEV ₁ after exercise	4.1 urban
Steinman ⁴⁶ (2003)	Transkei; Urban Cape Town: (a) informal, (b) middle class (418, 10-14 yr)	≥ 20% ↓ FEV ₁ after histamine	17 rural 34.4 urban (a) 33 urban (b)
Calvert ⁴⁷ (2005)	Transkei (1 671, 8-12 yr); Khayelitsha, Cape Town (1 651, 8-12 yr)	> 15% ↓ FEV ₁ or ≥ 26% ↓ FEF ₂₅₋₇₅ after exercise	8.9 rural 14.9 urban
Mashalane ⁵³ (2003)	Thokoza (475, 9-10 yr)	> 15% ↓ PEF after exercise	7.2
Robins ⁴⁸ (2005)	South-central Durban* (222, grades 3-6)	> 20% ↓ FEV ₁ after methacholine	50 urban

FEV₁: Forced expiratory volume in one second. PEF: Peak expiratory flow. FEF₂₅₋₇₅: Forced expiratory flow between 25% and 75% of the forced vital capacity.

* Residential areas in close proximity to petrochemical refineries and other industry.

The association between asthma and passive smoking, particularly maternal smoking in pregnancy and in the child's early life, is now well established,^{54,55} although the mechanism remains elusive. Local studies have confirmed this association. A large cross-sectional study of primary schoolchildren in a heavily smoking Cape Town population found a robust association between childhood asthma/wheezing and maternal smoking in pregnancy, the number of smokers in the household and the concentration of cotinine (a nicotine metabolite) measured in the child's urine.⁵⁶ Maternal smoking was by far the largest contributor.⁵⁷ Exposure to maternal smoking in pregnancy was reported in 53% of the children with asthma/wheeze and 36% of controls. Nationally, 9% of pregnancies involved foetal exposure to passive smoking.²³ The association of a range of upper and low respiratory symptoms with maternal smoking was confirmed by Richards⁵⁸ in a study of adolescents in the Vaal triangle. The stronger effects of maternal smoking may be because of longer or closer exposure of children to smoking by mothers and/or possible in utero initiation of respiratory tract damage.

In a study of aggravation of asthma by maternal smoking, Ehrlich *et al.*⁵⁹ found that the mean FEV₁ was lower among those asthmatic children whose mothers smoked. Surprisingly, frequency of BHR to histamine was lower among these children when compared to asthmatic children of non-smoking mothers, especially if the mother smoked ≥ 15 cigarettes per day. Maternal adjustment of smoking habits in response to smoking may be an explanation of this counterintuitive finding. However, the mechanism of aggravation of asthma by passive smoking remains obscure, particularly as passive smoking does not appear to be associated with differences in atopy.⁶⁰

Table 11.5. Predictors of bronchial hyperresponsiveness to exercise among 8-12-year-olds in Transkei and Khayelitsha, Cape Town⁴⁹

Predictor		Odds ratio (95% CI)*
Residence:	Rural	1.00
	Urban	0.52 (0.25-1.09)
Atopy: quantiles of wheal diameter	0 mm	1.00
	0.75-2.3 mm	1.29 (0.74-2.24)
	2.4-4 mm	1.19 (0.66-2.12)
	4.1-7 mm	2.70 (1.46-5.01)
Ascaris infection:	No	1.00
	Yes	1.87 (1.15-3.06)
Body mass index tertile:	Lowest	1.00
	Middle	1.37 (0.90-2.07)
	Highest	2.36 (1.54-3.69)
Animals "come into" house:	No	1.00
	Yes	0.66 (0.44-0.99)

CI: confidence interval

* Adjusted for education of head of household and number of household assets owned

Asthma in adults

Adult asthma (Table 11.6) has been less studied than among children and has relied entirely on questionnaire self-report. In the study based on the SADHS,¹⁷ the sociodemographic predictors of wheeze among adults in the past 12 months in multivariate analysis were female sex, older age (> 44 yrs), having less education and (independently) being white. Environmental risk factors were a history of tuberculosis, smoking and occupational exposures. This combination of risk factors and their similarity to the findings for chronic bronchitis (see above) suggest that the wheeze symptom in adults may be unable to distinguish between asthma and COPD. Even when the analysis was restricted to asthma ever, similar environmental risk factors emerged as for wheezing (with the addition of not having medical aid), suggesting that even a reported diagnosis of asthma may be non-specific.¹⁷

Table 11.6. Prevalence of self-reported asthma and wheezing in adult populations in South Africa

Study (yr published)	Population, (N, age range)	Outcome measure	Prevalence %	
			Male	Female
Wicht ²⁰ (1977)	Northern Suburbs, Cape Town (507, 20-80 yr)	Self-reported asthma	7.7	11.9
		Ever wheeze with shortness of breath	9.6	8.9
Nriagu ²⁶ (1999)	South-Central Durban* (693, ≥ 17 yrs)	Shortness of breath with wheeze past 12 months	28	
		Asthma ever	12	
Jithoo ²⁷ (2003)	Ravensmead/Uitsig, Cape Town (3 512, ≥ 15 yr)	Wheeze past 12 months	12.7	
		Doctor diagnosed asthma	7.9	
Ehrlich ¹⁷ (2005)	National sample (13 826, > 14 yr)	Wheeze past 12 months	14.4	17.6
		Asthma ever	3.7	3.8

* Residential areas in close proximity to petrochemical refineries and other industry

Asthma severity, hospitalisation and mortality

The 1995 ISAAC study showed that while the prevalence of lifetime or recent wheezing symptoms was higher in adolescents from well-off suburbs than from poor suburbs, poorer children who did wheeze were more likely to suffer frequently.⁶¹ Living in a socio-economically deprived suburb has also been shown to be strongly correlated with admission to an intensive care unit for asthma and to asthma mortality.⁴¹

Thus, while asthma prevalence is not necessarily associated with poverty, the complications of asthma are. The high global ranking of South Africa in asthma death rates and case fatality rates relative to population prevalence described earlier¹⁵ is likely to reflect both living conditions conducive to aggravation of asthma and inadequate health services.

Zar *et al.*⁶² reported that between 1980 and 1997 there had been a steady decline in asthma deaths in South Africa and a decline in paediatric near-fatal asthma episodes in Cape Town. Adult ICU admissions and the proportion requiring ventilation had remained unchanged. Possible factors mentioned by the authors as responsible for the decline in mortality were improved access to medical care and treatment of acute asthma, use of inhaled corticosteroids and asthma education. However, the same study found that the white population had half the asthma death rate of the coloured population, a situation that had not changed since an earlier study.⁶³ Almost three-quarters of the deaths occurred outside a health facility. Thus, while asthma care may have improved overall in Cape Town, the data suggest sharp persisting socio-economic disparity in access and/or quality of care.

In a study of factors distinguishing cases of near-fatal asthma from other cases of acute asthma presenting to hospital, no difference in health-care access or medication supply could be found between cases and controls.⁶⁴ The cases did show lower mean beta-agonist usage in the 24 hours prior to admission and a greater frequency of previous tuberculosis. Modifiable factors characterising acute asthmatics at risk of death thus remain somewhat elusive, but may include impaired perception of severity. The association with previous tuberculosis is interesting in the light of the findings discussed earlier between previous tuberculosis and chronic respiratory symptoms.

Studies of asthma care

With recognition of the secular rise in asthma prevalence in a number of countries came the realisation that asthma was under-diagnosed and under-treated. A large Cape Town study found that only 53% of children with multiple current asthma symptoms were recognised as having asthma.⁶⁵ Recognition was associated with access to private care, to being on inhaled therapy and maintenance therapy and to having used a peak flow meter.⁶⁵ In the SADHS, regular recent medical treatment among those with a history of asthma or symptoms (based on demonstration to the interviewer of medications kept in the home) was less likely among women, Africans, those with smoky domestic fuel and with fewer household assets (as markers of rural residence and poverty respectively).¹⁷ The apparently lower treatment rate of women was surprising given that women had a higher prevalence of recent wheezing than men.

Recent guidelines for asthma management have emphasised the use of inhaled adrenergic and corticosteroid agents.^{66,67} The SADHS revealed an apparent preference among respondents for oral medication, with 39% of respondents using oral xanthines and 28% oral adrenergics for "asthma and chronic bronchitis", in contrast to 15% using inhaled adrenergics and 15% inhaled glucocorticoids.²³ This preference may be that of the prescribers or consumers for the oral medication, but either way

there is cause for concern because of the lower benefit:cost ratio of oral than inhaled medication for a given dosage, the greater difficulty of controlling dosage and the larger potential for side effects.²

Similar preferences regarding medication were found in a study of knowledge, attitudes and practice of parents of asthmatic children in a low-income Cape Town community with high asthma prevalence.⁶⁸ Resistance to inhaled therapy was reported, with reliance on syrups and home remedies. There was low compliance with prescribed medication. Parents also complained of poor levels of service offered by public sector clinics. The authors concluded that there was an urgent need for asthma education of parents and improvement in the quality of public service asthma care.

The CHAMP study (see above) used academic detailing by a pharmacist targeted at private general practitioners as an intervention to improve childhood asthma management in an urban setting in Cape Town.⁶⁹ General practices were randomised and children with asthma symptoms linked to practices via a large school survey.⁷⁰ At one-year follow-up the children who were patients of the intervention practices had a small but statistically significant greater improvement in an asthma symptom score than children who were patients of the control practices.

Treatment of lung diseases in the public sector was targeted in a randomised controlled trial in the Free State, named the Practical Approach to Lung Disease in South Africa (PALSA).⁷¹ Combined syndromic evidence based guidelines with two to six training sessions were aimed at primary-care nurses in resource-poor public-sector clinics, enabling them to diagnose and treat common lung diseases like asthma. WHO guidelines were adapted to suit local circumstances. The adapted guideline was validated prospectively, confirming that it performed well as a screening, diagnostic and treatment tool when compared to specialist diagnosis.⁷¹ Some aspects of respiratory care were significantly improved in the intervention group compared to the control group, in particular, the provision of inhaled corticosteroids (odds ratio 1.90, 95% CI: 1.14-3.18).⁷²

Guidelines for treatment of asthma

Updated guidelines for the management of asthma in adults⁶⁷ and children⁶⁶ were published in 2000. As with earlier guidelines, emphasis was laid on appropriate staging of severity and early use of inhaled corticosteroid therapy. The guidelines incorporated the improved understanding of the role of long-acting beta agonists and the introduction of leukotriene receptor antagonists. However, cost of medication is recognised as a significant barrier to optimal asthma care in South Africa, particularly within public-sector budgets,⁷³ as is the case generally in low-income settings.⁷⁴

4. CHRONIC RESPIRATORY DISEASE AS A RESULT OF ENVIRONMENTAL AIR POLLUTION

There have been relatively few studies of air pollution since the large Vaal Triangle studies undertaken in the early 1990s that were reviewed in the previous Report,⁷⁵ and elsewhere.⁷⁶

Indoor air

South Africa embarked on a large-scale electrification programme during the 1990s, with an increase in the proportion of households electrified from 36% in 1994 to about 68% (of 10 million households) at the end of 1999.⁷⁷ A much higher percentage of urban households (80%) than rural (46%) were electrified by that date. A large proportion of the population is thus still dependent on highly polluting biomass fuel (wood, grass or dung) and fossil fuels (coal) for their indoor cooking and heating requirements. Rural homes in South Africa have been measured as having a higher level of respirable particulate matter and carbon monoxide than their electrified counterparts.⁷⁸ In a Cape Town peri-urban area use of paraffin was associated mainly with an elevated carbon monoxide concentration.⁷⁹

Poorly ventilated combustion of biomass fuel by itself is well established in poor rural settings in other countries as a cause of chronic bronchitis.⁸⁰⁻⁸² The SADHS found that 32.6% of men and 38.2% of women were exposed to smoky domestic fuel (wood, coal or dung).⁸³ There was a very strong association between exposure to smoky domestic fuel and markers of poverty as well as rural residence. The same survey found an association between chronic bronchitis and smoky domestic fuel only among women.¹⁶ Rural African women, the group most exposed to this type of indoor air pollution, are thus a major contributor to the higher prevalence of chronic bronchitis overall in women than men in South Africa despite lower smoking prevalences.⁸⁴ (See text under COPD above).

Outdoor air pollution

Two recent investigations have focused on localised air pollution in the vicinity of industrial areas in close proximity to residential suburbs, one in Durban and one in Cape Town. In the study in south-central Durban, Nriagu *et al.*²⁶ found very high prevalences of respiratory symptoms. Surprisingly, only 10% of children and 12% of adults reported doctor-diagnosed asthma, no different from that reported elsewhere in the country, suggesting under-diagnosis of these conditions. Asthma prevalence was very strongly correlated with school absenteeism (odds ratio 44, 95% CI 13-141), highlighting the adverse impact of asthma on quality of life and presumably on learning in children.

A high prevalence of respiratory symptoms was also found by White *et al.*⁴³ in a study of children in the northwest suburbs of Cape Town in the vicinity of a petrochemical refinery, much higher than reported in a previous asthma symptom survey in greater Cape Town.⁶¹ In this mainly middle-class study area, 23.7% of children reported that they had ever had asthma and 64.6% reported that they had ever had hayfever. Using the local petrochemical refinery as the putative point source, symptoms were associated with a meteorologically estimated exposure incorporating wind direction and speed and distance from the refinery. The study was not able to test any hypothesis regarding the specific emission that might be responsible. Previous monitoring of sulphur dioxide levels in the area, for example, had shown relatively low concentrations. However, the results were compatible with the hypothesis that petrochemical refinery emissions were an important risk factor for asthma or asthma exacerbation among children in this area.

5. CHRONIC RESPIRATORY DISEASE AS A CONSEQUENCE OF OCCUPATION

The contribution of occupational exposures to chronic bronchitis and asthma symptoms at the general population level has been mentioned above. The specific chronic occupational lung diseases that have received the most attention over the past decade have been diseases caused by mineral dusts, particularly in gold miners, and occupational asthma.

Chronic lung disease caused by mineral dusts

Workers inhaling silica dust are at risk of silicosis, tuberculosis, COPD and lung cancer.⁸⁵ Prior to 1994, very few studies had been published on black mineworkers, who traditionally have held dustier jobs than white mineworkers, veiling the significant epidemic of silicosis in the gold mining industry.^{86,87} The true toll of occupational lung disease in black gold miners is now emerging. Studies of ex-miners in labour sending areas of the Eastern Cape⁸⁸ and Botswana⁸⁹ found very high prevalences of radiological silicosis, of the order of 25%. In 2004, Churchyard *et al.*⁹⁰ published the first study using dust exposure data of exposure-response relations for silicosis in active black South African gold miners. Almost 20% of older, longer-service gold miners had developed silicosis, confirming the large burden of the disease in this group. Assuming stable dust counts over a long period, this burden occurred despite a mean quartz exposure well below the recommended occupational exposure limit for respirable quartz of 0.1mg/m³. The findings are consistent with the view that the lengthening of the average duration of service on the mines, together with failure to control dust to an adequate degree over the past three decades, have contributed to a rising prevalence of silicosis.^{86,91}

Both silica exposure and silicosis are risk factors for pulmonary tuberculosis.⁹² While the incidence of this disease among gold miners was high prior to the HIV pandemic, HIV co-infection has led to a fourfold increase in the incidence rate among gold miners, reaching approximately 2000 per 100 000 miners in one group of mines in 2000.⁹³ Corbett *et al.*⁹⁴ have shown in another group of mines that while HIV infection and silicosis increased the incidence of tuberculosis in gold miners fivefold and threefold, respectively, the combined effect of HIV and silicosis was to increase the incidence of tuberculosis by 15 times. In the light of the above findings, the need to control silica exposure should be a priority of the mining industry and government.

Chronic lung disease in coal miners has been barely studied in South Africa. A recent study demonstrated a relatively low prevalence of radiological coalworkers' pneumoconiosis, i.e. 2-4% (depending on x-ray reader).⁹⁵ An autopsy study of coal miners found length of service to be associated not only with silicosis and coal workers' pneumoconiosis defined pathologically but also with emphysema after controlling for smoking.⁹⁶

With regard to COPD in gold miners, an early study of mortality in white miners showed that smoking and silica exposure acted synergistically in increasing the risk of death from COPD.⁹⁷ In a study of lung function loss, the same author found that the average loss of FEV₁, attributable to the

effects of 25 years of gold mining was 236 ml in comparison with the average loss attributable to smoking one packet of cigarettes per day over 30 years of 552 ml.⁹⁸ Past tuberculosis is also a potent cause of lung function loss in miners, with the proportion with chronic airflow limitation rising from 18.4% among those with one previous episode to 27.1% among those with two previous episodes of pulmonary tuberculosis.²⁸ While smoking is prevalent among certain groups of mineworkers, this is not universally so, with the prevalence ranging between 10% and 75%, depending on age, country of origin and previous employment.⁹⁹ As smoking becomes more common among black mineworkers, the prevalence and severity of COPD in this group can be expected to rise.

Occupational asthma

A recent authoritative international review of the proportion of adult asthma attributable to occupation put the figure at 15%.¹⁰⁰ A study of acute asthma at a hospital casualty department in Cape Town found that 13% of cases were consistent with occupational asthma, within the range of the above review.¹⁰¹ Aggravation of asthma by work was reported in 25.7% of these asthmatics.

Between 1997 and 1999, a voluntary register, Surveillance of Work-related and Occupational Respiratory Diseases in South Africa (SORDSA), recorded 324 cases of occupational asthma, a national incidence of 17.5 per million employed persons.¹⁰² Isocyanates (a component of automotive spray paints and other products), latex, flour and grain, and platinum salts were the most common agents. However, the above must be regarded as a minimum incidence as most cases are likely to be unreported.

The findings of workforce specific studies of asthma and respiratory allergy more broadly, using different methods are presented in Table 11.7. These reveal the existence of a serious problem in these industries.

A local study of prognosis of occupational asthma suffered from a low response rate, but among those cases followed up as many as 16.2% were no longer working.¹⁰³ Only 55% of workers had submitted claims for compensation. The poor functioning of the compensation system for occupational asthma¹⁰⁴ and for occupational disease in general¹⁰⁵ has been well documented.

Table 11.7. Occurrence of occupational asthma or respiratory allergy South African workforces

Workforce (N) (author, yr published)	Agent	Condition	Prevalence or incidence (%)
Platinum refinery (78) (Calverley 1995) ¹⁰⁶	Platinum salts	Platinum salt sensitivity	41 (24 month incidence)
Poultry workers (134) (Rees 1998) ¹⁰⁷	Feed, poultry matter (feathers, droppings, serum)	Asthma symptoms	12
Hospital workers (2 316) (Potter 2001) ¹⁰⁸	Latex	Latex allergy (ocular, respiratory and skin)	9.2
Vineyard workers (207) (Jeebhay 2002) ¹⁰⁹	Spider mite	Work-related spider mite respiratory allergy	7
Sea food processing (594)(Jeebhay 2003) ¹¹⁰	Fish products, fish parasite	Occupational asthma	3
Bakery employees (517) (Jeebhay 2005) ¹¹¹	Flour grains (wheat, rye) and alpha amylase	Occupational asthma	11
Grain mill (111) (Jeebhay 2000, 2005) ^{112, 113}	Grains, storage pests (mealworm)	Work-related wheeze	13

Guidelines for management of occupational lung disease

Guidelines for the diagnosis and management of occupational asthma have been published in South Africa, the most comprehensive being that in the Handbook of Practical Allergy.¹¹⁴ Guidelines for the diagnosis and compensation of a number of occupational lung diseases have been issued, e.g. the asbestos related diseases,¹¹⁵ and more recently in the form of "Circular Instructions" by the office of the Compensation Commissioner under the Compensation for Occupational Injuries and Diseases Act. These can be accessed on the Department of Labour website under Regulations and Notices under the above Act (<http://www.labour.gov.za/programmes/>). Guidelines currently available (either on line or on request) cover occupational asthma, irritant-induced asthma, work-aggravated asthma, byssinosis (airways disease associated with cotton dust), upper respiratory tract disorders, mesothelioma, lung cancer and tuberculosis in silica exposed workers.

6. POLICY AND RESEARCH

Primary prevention of disease

COPD has been called “the hidden epidemic” with the diagnosis going unrecognised or unrecorded by health-care providers.¹¹ This relative neglect may be partly as a result of the condition affecting primarily older people, and therefore being seen as a disease of “lifestyle”, i.e. subject to individual choice, and thus overshadowed by other health priorities competing for scarce health system resources. There may also be the perception, for example in Africa, that it is a disease of concern mainly to developed societies. In fact, most cases now occur in middle income and poor countries, especially given the huge burden in China and other Asian countries.^{11,12}

Enough is known about the causes of COPD to direct policy and research effort to finding the most effective preventive strategies to stem the enormous toll of morbidity, health service costs and loss of working days associated with the disease.^{116,117} Tobacco impact and control remain a research priority. Although much is known about the effects of tobacco, it seems that each country needs indigenous research to remind policy makers of the local burden. Thus, epidemiological monitoring of tobacco usage,^{84,118} morbidity and mortality¹¹⁹ remains important in South Africa. There is evidence that tobacco usage is falling among all but the highest income groups.¹²⁰ The increased cost of smoking is likely to be responsible for this pattern of change. Epidemiological monitoring needs to be supplemented by monitoring of policy and control activities. South Africa now has a decade's worth of serious tobacco control activities dating from the Tobacco Products Control Act of 1993, which requires critical reflection on its effectiveness. This is dealt with in more detail in Chapter 4 of this Report.

Complementary to upstream measures to reduce tobacco consumption are multidisciplinary approaches to understanding the determinants of smoking behaviour, for example, based on theories of behaviour change. One of the few such examples in South Africa is focused research into reducing the high smoking prevalence among low-income coloured women using public health antenatal facilities.¹²¹⁻¹²²

The role of post-tuberculous lung disease in contributing to the burden of chronic respiratory disease in South Africa is emerging.^{16,17,21} Research into tuberculosis and its control is a national priority and hardly needs emphasis. However, a better understanding of the pathophysiology of airflow limitation following tuberculosis is needed as there is relatively little work on the subject.¹²³ Such understanding may point the way to therapeutic strategies for control of symptoms in these patients.

The problem of indoor and localised outdoor air pollution as a result of polluting domestic fuels is complex and admits of no easy solution.^{76,124} The current programme to greatly expand electrification to households in all areas of the country is likely to produce a substantial health benefit. However, large-scale electrification is unlikely to be viable in rural areas. Here the emphasis needs to be on cleaner but affordable stoves, a substantial barrier in very poor communities.⁷⁶ South Africa's heavy reliance on coal for electricity generation also confers substantial external costs in ambient pollution,¹²⁴ which need to be taken into account in contemplating the benefits of electrification. Alternative domestic fuels, where affordable, are another solution. For example, paraffin (kerosene) is less polluting than coal,¹²⁵ although it is associated with increased risk of accidental child poisoning. Improvements to housing through relatively simple low-cost interventions, such as better thermal insulation and solar water heaters, are other approaches to reducing the volume of polluting fuels burned.¹²⁶

Other than the Vaal Triangle study,¹²⁷ there is a striking lack of large-scale epidemiological studies of the impact of ambient air pollution in South Africa. There is a need for properly funded studies of sufficient power to determine health effects in areas of high or increasing air pollution. In addition to traditional industrial pollution, the extent and impact of photochemical smog, resulting from the action of sunlight on oxides of nitrogen and hydrocarbons and which is becoming more common in heavily congested urban areas, need to be monitored.⁷⁹

There is now enough evidence for a general contribution of workplace “dust, gases, fumes and vapours”, as well as of specific agents, such as silica, coal, grain dust and welding, to the occurrence of COPD to include occupational health measures as a priority component of public health action to control COPD.^{100,128} Prevention of occupational respiratory disease is predicated on the enforcement of engineering and other well-established workplace controls required by the Occupational Health and Safety Act (No. 85 of 1996) and the Mine Health and Safety Act (No. 29 of 1996). However, there are a number of barriers to compliance, the most important being reluctance on the part of owners to incur the costs of appropriate technological controls. The risk assessment and monitoring of airborne hazards required by the law are absent or poorly carried out for preventive purposes in many South African workplaces, even in the mining industry.¹²⁹ The state's ability to enforce legislation is also weak, a situation attributable to skills shortage and fragmentation of enforcement

efforts across different government agencies.¹³⁰ Trade union pressure, historically an important force for improvement, has been attenuated by the effects of labour market restructuring and globalisation on unions' bargaining power in various sectors.¹³⁰

Protective statutory occupational exposure limits are another element of control of occupational respiratory hazards. Emerging evidence described above for the "occupational COPD effect" suggests that the legislative standard for so-called "nuisance dust" (i.e. low toxicity dusts without substance-specific occupational exposure limits) are insufficient to protect against COPD.¹²⁸ Also, much of the evidence on exposure-response relationships has come from cross-sectional studies, which suffer from well-known selection biases as well as difficulties in constructing historical exposures. Cohort studies, which could reduce some of these biases, are expensive and require the resources of a large industry. Although gold mining is one such industry, there are as yet no published cohort studies of black gold miners that could provide important evidence of exposure-response relationships for silicosis and tuberculosis and also better characterise the increasing risk of COPD among such miners.

With regard to asthma, there has been a burgeoning of research internationally over the past three decades, stimulated by the rising prevalence and burden of complications of asthma. There is now a large database of comparative asthma prevalences among children which have served to test various hypotheses about the population distribution of asthma. In South Africa, studies have almost all been confined to the Eastern Province (Transkei) and Cape Town. There is a real need for similar studies to be carried out elsewhere, particularly in the inland regions.

Asthma research has been rich in hypotheses, with the "hygiene hypothesis" in particular appealing to a wide audience because of its elegant biology and seeming ability to explain many of the unanswered questions about asthma trends. This hypothesis, which has undergone modification over time,¹⁸ has not been subject to systematic testing in South Africa, although some support for it can possibly be found in the studies reviewed above by Calvert⁴⁹ and, at least as regards childhood hayfever and eczema, by Obihara *et al.*⁴⁴

The state of knowledge of the causes of asthma is such that it lends itself less well to primary prevention approaches. The role of prolonged breastfeeding in prevention of allergic disease in childhood was recently tested retrospectively in a cross-sectional study in Cape Town and found to be associated with reduced allergic disease only in those children whose parents were not atopic.⁴⁴ This may have implications for reducing the risk of allergic asthma. However, there are two other areas where known preventive activities could make a substantial difference: passive smoking and occupational asthma.

The impact on asthma and respiratory illness in general of smoking by mothers in pregnancy and by parents who have children in the home, is now well characterised. The considerations above concerning tobacco control are thus relevant to asthma, with a particular educational focus required for parents of young children.¹³¹

Considering that between 10% and 15% of new adult asthma is occupational and that a larger proportion is aggravated by work, control of specific workplace exposures could make a significant difference to population incidence of asthma. Research which measures the burden and is able to characterise the asthmagenic risk of specific agents (see Table 11.7) serves to focus attention and prevention efforts. Most of these studies have been in food processing where workers are exposed to high molecular weight compounds. Apart from platinum salt asthma, asthma associated with exposure to low molecular weight chemical agents, such as isocyanates, sulphur dioxide, formaldehyde, chrome and other metals have not been the subject of significant epidemiological studies in South Africa. Reasons for this lack include the relatively low number of exposed workers in each of the relevant workforces and the absence or low predictive utility of immunological tests for these agents.

Secondary prevention of disease

Secondary prevention of disease includes early diagnosis and cost-effective management. The public health and clinical approaches to asthma have usually been distinguished from those with respect to COPD. For example, while measures aimed at COPD, such as control of tobacco and of domestic and workplace air pollution, are likely to have a positive impact on the prevalence of asthma as well, medical evaluation and therapy have been emphasised as central to public health approaches to asthma.¹³² However, with guidelines for both diseases emphasising control of risk factors, patient education, early diagnosis to prevent progression, as well as optimal drug therapy,^{1,30,66,67} the difference between COPD and asthma management in this regard may be diminishing.

International publications focusing on management of COPD and asthma in developing country settings are also becoming more common.^{12,133} However, a number of barriers to improved management of these diseases in low-resource settings have been cited. These barriers include insufficient skilled health-care personnel, inadequate training of practitioners in optimal management of these conditions, high cost and limited availability of medications, and failure to use or lack of basic diagnostic equipment. In the primary-care setting, asthma is under-treated, antibiotics over-prescribed and tuberculosis under-diagnosed.⁷² All of these factors are likely to contribute to the socio-economic and racial group differentials in morbidity and mortality of asthma and COPD in South Africa described above, as well as widening the gap between the private and public sector in quality of care.

A study by the South African Thoracic Society found that while pulmonology had grown as a discipline, pulmonologists were concentrated in Gauteng, Western Cape and KwaZulu-Natal, with a minority in the public sector.¹³⁴ The decline in clinical technology posts for lung function technologists in public sector facilities has also been noted.³³ There is thus a need for a national human resources plan for skilled personnel in pulmonology based on an estimate of future needs and a training and placement strategy to meet these needs. A plan for the deployment of pulmonology skills in referral and tertiary hospitals has in fact recently been developed.¹³⁵

The relatively high cost of medication is another barrier to optimal management of asthma and COPD, particularly in a resource strapped public sector struggling with many competing primary health-care goals.^{73,74} However, the cost-effectiveness of good control of asthma using maintenance corticosteroids has been shown in a developing country setting.¹³⁶ Also, innovative alternatives to expensive spacers for inhaled medication in young children have been developed in South Africa.¹³⁷ It is important to provide evidence to health service managers whose drug procurement policies are based purely on the cost of medication, that failure to take into account the "downstream" costs of treating complications of asthma will result in higher costs for the system as a whole,¹³⁸ even without taking into account impacts on work productivity and disruption of family life.

While consensus guidelines need to be widely publicised to medical practitioners, there is evidence that publication alone of such guidelines is insufficient to change practitioner diagnosis and treatment behaviour.¹³⁹ The CHAMP study described above found benefit in asthma outcomes in children from a single academic detailing visit to general practitioners, but ways of institutionalising such visits remain elusive. With regard to smoking cessation, there is evidence that simple advice by medical practitioners is able to produce some benefit in patients.¹⁴⁰ However, local medical practitioners in public sector facilities were found to be unaware of guidelines for structured smoking cessation counselling in pregnancy.¹²¹ Some were also pessimistic about their ability to influence such smoking behaviour given the pressures on antenatal care delivery and the barriers to quitting among their patients.¹²¹

A further barrier to improvement of the quality of care for asthma and COPD is the failure to use basic lung function testing in diagnosis and monitoring. A study of asthmatic children aged 6-8 years in Cape Town, found that only 46% of diagnosed asthmatics and 13% of undiagnosed asthmatics reported ever having used a peak flow meter.⁶⁵ There are no data on what proportion of primary-care facilities in South Africa has working spirometric equipment, but the Cape Town experience is that very few have such equipment, making it impossible to properly apply the guidelines for diagnosis and management of COPD.

Operations research is thus needed to identify barriers to the implementation of recommended COPD and asthma care at a primary-care level, particularly in the public sector, and ways to implement practice guidelines. The PALS study described above is important in that it takes into account the reality that primary care in South Africa will increasingly need to be supported by clinical nurse practitioners, and demonstrates that a syndromic approach to lung health using limited training of nurses, can produce benefit in patient management.⁷²

With regard to occupational asthma, secondary prevention in the form of early diagnosis improves prognosis if the patient is able to avoid further exposures.¹⁴¹ The ability of workers to avoid sensitising exposures is dependent on a legislative framework, including compensation, which allows for redeployment without threat to wage or job security and for retraining for a new occupation. Efforts currently underway to improve the efficiency of the compensation system for occupational disease will reduce some of the costs, particularly those related to delays in settling claims, currently borne by workers.¹⁴² However, with an unemployment rate of the order of 40% in South Africa,¹³⁰ diagnosis of an occupational disease will remain a serious threat to the patient's livelihood.

7. CONCLUSION

Much has been learned about COPD and asthma in South Africa in the decade since the last edition of this Report, stimulated by international efforts to standardise both the study of these conditions and their management. Although COPD is a preventable disease, we are destined to watch its inexorable increase in South Africa unless measures to prevent smoking and to control occupational exposures are pursued vigorously by the state and agencies involved in health promotion. Early detection of COPD through use of guidelines and spirometry should be the health service component contributing to prevention at an individual and population level.

In the case of asthma, we are still searching for the underlying causes of secular shifts in the condition and its severity. With its relatively rapid social and economic change, South Africa is well placed to investigate emerging hypotheses on the contribution to the population incidence of asthma of changing infection rates, immunisation rates, diet, family size and other correlates of development. However, the advances in management of the asthma will not reach the majority of the population unless ways can be found to ensure a sustained improvement in the basic quality of asthma care, particularly in public health services.

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