LETTERS TO THE EDITOR

Asthma morbidity and mortality in New Zealand

The occasional review by Dr Garrett *et al* (March 1995;**50**:303–11) concerning asthma mortality and morbidity in New Zealand illustrates the considerable confusion that can arise from selective use of time trend data when it is shaped to fit a preconceived hypothesis that is unsupported by other evidence.

Leaving aside the fenoterol or β agonist issues, the time trend data simply do not support a socioeconomic "cause" for the New Zealand epidemic. First, the epidemic commenced in 1976 whereas unemployment only began to increase significantly in 1978.[1] Secondly, while unemployment was 50 000 in 1980 at the peak of the epidemic, it subsequently rose to well over 200 000 during the time that asthma mortality fell to the lowest levels for 30 years.[2] Thirdly, during the period of the second New Zealand epidemic there were no epidemics of deaths from other causes, many of which are more strongly related to unemployment and social deprivation. Lastly, when New Zealand experienced the first epidemic of asthma mortality in the mid 1960s there was no unemployment. Thus, although social deprivation and poor access to health care clearly plays a part in some asthma deaths, the time trend data are strongly inconsistent with the hypothesis that there was a socioeconomic cause for the epidemic.

While the authors do not mention these anomalies, they do suggest 11 initiatives that might have helped to reduce mortality. However, many of these were available before or during the epidemic, including educational material, the asthma task force, after hours care, and efficient ambulance services. Other initiatives were only introduced after the epidemic had ended, including the national promotion and distribution of action plans and peak flow meters.[3] Remaining factors, such as multidisciplinary approaches and specialised hospital based asthma clinics, did not exist on a national basis and, where they did exist at a local level, failed to reach the asthmatics most at risk. Surprisingly, the same authors who in their review suggest that such clinics might have contributed to the decline in asthma mortality have themselves illustrated the failure of such clinics to reach their target population. Thus Garrett *et al*[4] have reported that almost 80% of this high risk group either did not attend or attended poorly. It is difficult to see how this sort of experience could have altered national asthma mortality rates or, indeed, how any of these initiatives could have abruptly ended the epidemic in 1989.

Garrett *et al* have illustrated the problems and pitfalls of using selective time trend data alone to support hypotheses. That some of the factors cited were involved either in increasing or in decreasing the risk of death from asthma in New Zealand (such as the increasing use of high dose inhaled steroids) is clearly likely; that the conglomeration presented in their review might explain the sudden onset and sudden end of the epidemic in 1976 and 1989, respectively, is not supported by the time trend data nor by any other evidence.

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1 Pearce J, Beasley R, Crane J, Burgess C, Jackson R. End of the New Zealand asthma mortality epidemic. *Lancet* 1995;**345**:41–4.
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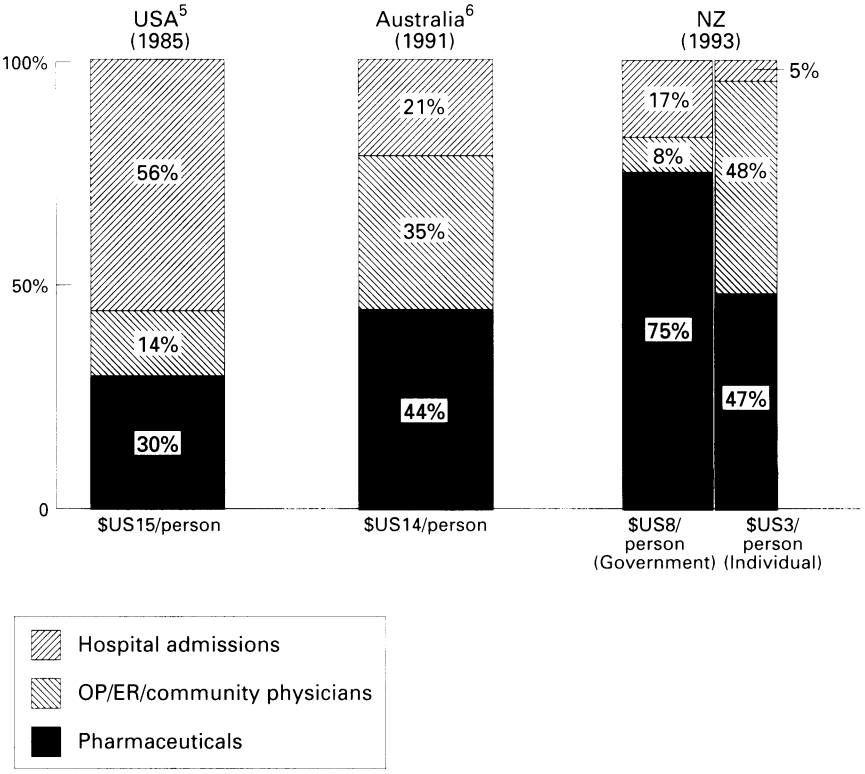
3 Crane J, Burgess C, Pearce N, Beasley R, Durham J. Adult asthma management: the patient takes the helm, health professionals chart the course. Department of Health Therapeutic Note 1991; vol 211.
4 McClellan VE, Garrett JE. Attendance failure at Middlemore Hospital asthma clinic. *NZ Med J* 1989;**102**:211–3.

AUTHORS' REPLY Dr Crane *et al* are correct in stating that time trend data do not support socioeconomic factors as the sole explanation for the New Zealand epidemic of asthma deaths. However, no single factor can be defined to explain the epidemic using time trends analysis and this was the rationale for writing the review. Our aim was to illustrate how a variety of factors (some unique to New Zealand) may have interacted to create the epidemic of asthma deaths, and how a different variety of factors have combined to reduce the mortality rate. Increasing poverty is likely to have been very important and in the 1990s remains an important risk factor for severe life threatening asthma. In a prospective study Kolbe *et al*[1] have shown that 35% of patients admitted with acute severe asthma come from a household in which the only income is a Social Security benefit; this compares with approximately 12% in the general population. There have been mortality trends in other conditions in New Zealand, that may be related to socioeconomic factors, which would



Direct cost of asthma management in a comparison of three countries using similar methods of analysis.

Cost benefit analysis of inhaled steroids (South Auckland, New Zealand; population 310 000)

Direct costs	*1988 ($)*	*1992 ($)*	*Difference*
Pharmacy:			
Inhaled steroids*	1 400 000	2 700 000	+1 300 000
Other medications*	2 100 000	1 800 000	−310 000
Hospital:			
Admissions (N × bed days)†	1 500 000	880 000	−620 000
ICU admissions (N × bed days)*	96 900	8 500	−88 400
ER attendances (N)†	411 200	187 000	−224 200
OP attendances (N)†	93 600	62 500	−31 100
Community:			
After hours attendances for acute attacks (N × govt subsidy)‡	42 000	29 400	−12 600
Total	5 643 700	5 667 400	+23 600

ICU = intensive care unit; ER = emergency room; OP = outpatients.
* IMS data.
† Middlemore Hospital/Princess Mary costs.
‡ After hours community care (Government subsidies).

support our hypothesis; specifically, a threefold increase in suicide rates in 15–24 year old men and a twofold increase in 15–24 year old women between 1970 and 1988.[2] Of interest, the most dramatic increase in suicides occurred between 1976 and 1977 in both 15–24 and 25–44 year old men – the first year of the asthma epidemic. Even in the UK, where there are no obvious financial barriers to health care, poverty has been shown to be associated with an increased risk of severe asthma[3] and such patients would be expected to have been further disadvantaged if they were confronted with escalating costs of primary health care such as those which have been imposed increasingly since the 1960s in New Zealand.

The direct costs of managing asthma in New Zealand are substantially higher for the individual patient than in any other developed country where such studies have been performed.[4,5] Using the same methods as Mellis *et al*[5] we found that up to 30% of the direct costs of managing asthma are borne by the patient in New Zealand (figure). Further, only a small proportion of the direct costs of managing asthma in New Zealand is spent on primary health care or specialist care, the majority being spent on medication. In the late 1970s, therefore, most of the financial support was for bronchodilator therapy (as opposed to inhaled steroids) and secondary health care with very little funding of primary health care. A multivariate linear regressional analysis of data collected in 1993/4 has shown that those on oral theophylline ($p = 0{\cdot}0006$) and with the highest serum salbutamol levels ($p = 0{\cdot}03$) had the most severe asthma at the time of attendance at two New Zealand emergency departments.[6] In the 1990s, therefore, some of the factors which contributed to the epidemic in the 1960s and 1970s are still apparent – namely, high unemployment, financial barriers to primary health care, and overreliance on bronchodilator therapy for the management of acute asthma (though acknowledging that this trend in behaviour may have improved). Such factors would be expected to have reduced the effectiveness of the various strategies developed to improve asthma care![7]

The evidence that inhaled corticosteroids reduce morbidity and thus mortality is overwhelming. In one prospective randomised study in a GP setting acute attacks were virtually abolished with the introduction of inhaled steroids.[8] In the most comprehensive randomised study of asthma education undertaken to date[9] we have shown significant improvement in knowledge and self management skills for those randomised to a community-based education centre, but no change in behaviour or hospital admission rates. Despite this, there was a 64% reduction in hospital admissions in the community targeted during the study, and one factor shown to have changed substantially was the sale of inhaled steroids (1·4 million to 2·7 million dollars) (table).

Asthma clinics had been established in most urban settings in New Zealand by 1985. Non-attendance rates for new references to our asthma clinics in Auckland are about 35% (not 80%).[10] Although we have not validated the efficacy of these multidisciplinary outpatient clinics directly, Allen *et al*[11] have. They had a similar non-attendance rate (38%) to us, despite which they were able to show a significantly lower readmission rate (OR 2·3) analysed on an intention to treat basis for those randomised to the clinic. Mayo *et al*[12] found a threefold reduction in readmission rate over three years in a group of patients randomised to intensive treatment compared with usual treatment within the outpatient setting, and this difference was largely due to the fourfold increase in use of inhaled corticosteroids in the intensive treatment group. This would explain why readmission rates began to drop in New Zealand in 1987 within two years of high dose inhaled steroids being registered for use by specialists only and two years before the debate on inhaled fenoterol began.

Whilst no study has yet shown benefit from changing from regular inhaled bronchodilator therapy to that of as required use only, there are many studies which show substantial benefit from the introduction of higher dose inhaled steroids. The major escalation in inhaled steroid sales in New Zealand therefore remains the most logical explanation for the reduction in both morbidity and mortality, particularly in view of the continued increase in sales of inhaled β agonists.

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In their review of asthma morbidity and mortality in New Zealand (March 1995;**50**:303–11) Dr Garrett and his colleagues argue that withdrawing fenoterol as a prescription medicine in New Zealand in late 1989 was only one among many factors that led to an overall improvement in morbidity and mortality from asthma in New Zealand. They cite no less than 11 initiatives which they consider to have been important contributors to the reductions in hospital admission and mortality rates which have occurred in New Zealand over the last 10 years, but exclude the withdrawal of fenoterol as one of them. Why?

We have no doubt that each of the initiatives discussed was necessary and important and contributed to the decline in mortality rates for some considerable time before 1989. However, in their evaluation of trends they do not account for the fact that first (fig 5) and total (fig 7) hospital admission rates remained obstinately unchanged until 1989, despite the fact that all of the measures outlined in their review were implemented on a nationwide basis well before that time.

The only new strategy adopted in 1989 was the withdrawal of fenoterol followed by recommendations to use β agonists as required rather than as regular treatment. This practice is now accepted in international guidelines for the management of asthma. The first of these steps was prompted by epidemiological data from Wellington,[1] and the second by the results of a study conducted in Dunedin which showed that control of asthma deteriorated when fenoterol was given regularly rather than as required to patients with mild to moderate asthma.[2] The results of these investigations were complementary. They indicated not only an increased risk of death from asthma among patients receiving inhaled fenoterol, but also that the underlying cause was likely to be an increase in baseline severity when inhaled fenoterol was being taken regularly. We have recently discussed the relationship between β agonist use and asthma morbidity and mortality in greater detail.[3]

It is therefore quite understandable that improvements in the delivery of and access to medical care – by whatever mechanisms – reduced asthma mortality before 1989, but the available data indicate that they had little impact on improving severity and hence morbidity, particularly first hospital admissions. Only when the major factor affecting severity was removed was there a subsequent reduction in morbidity.

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1 Crane J, Pearce NE, Flatt A, Beasley R. Prescribed fenoterol and death from asthma in New Zealand. *Lancet* 1989;i:917–22.
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AUTHORS' REPLY Drs Taylor and Wong are correct in stating that it is our contention that the dramatic decline in asthma morbidity and mortality in New Zealand was due to multiple factors and that the precise contribution of individual factors cannot be accurately estimated retrospectively. However, on the one hand they agree that the possible contribution of the withdrawal of fenoterol was acknowledged, but immediately make the accusation that we "exclude(d) the withdrawal of fenoterol as one of them".

In a retrospective cohort study we have also shown that fenoterol was associated with an increased risk of fatal or near-fatal asthma (crude RR = 2·1).[1] However, because of more accurate, complete and detailed information on asthma severity we were better able to control for confounding by severity. After controlling for multiple risk factors, fenoterol was no longer associated with an increased