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Will More Doctors Increase or Decrease Death Rates?

An econometric analysis of Australian mortality statistics

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Abstract

There is now documented evidence that adverse events associated with medical interventions have a seriously deleterious affect on population health. However this literature does not indicate whether or not the net affect of an increase in the doctor supply is positive or negative. There are, additionally, other mechanisms by which an increase in the doctor supply could reduce the quality and length of life. This paper revisits two hypotheses concerning doctor induced ill health. Econometric results using Australian cross-sectional data are presented. They are consistent with the hypothesis that an increase in the doctor supply is associated with an increase in mortality.

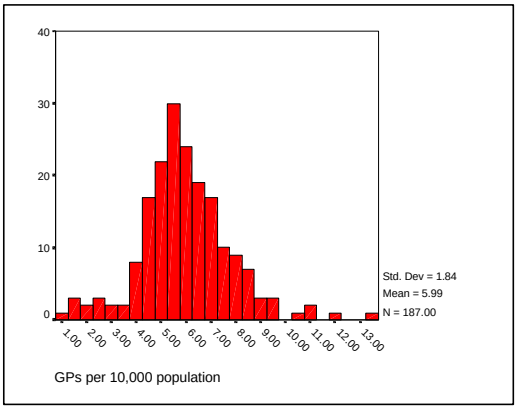
Will more doctors increase or decrease death rates? An econometric analysis of Australian mortality statistics

1 Introduction

Access to health services across Australia has always been uneven. Analysis of the data generated by the first full year of universal health insurance (1976) revealed a per capita use of GP and specialist services which varied by 47 to 58 percent between the States (excluding the Northern Territory) and by a *factor* of 3.1 and 15.7 between the 58 statistical divisions in the States (Richardson and Deeble 1982). There is little evidence to suggest that the subsequent increase in the doctor supply reduced inequalities[1]. Figures 1a, 1b below demonstrate that by 1994/95 large differences still existed in the per capita use of these services. *Prima facie* it might be expected that these differences would have an effect upon health status and Figures 2a and 2b reveal that Standardised Mortality Ratios (SMRs), indeed, vary significantly across the country.

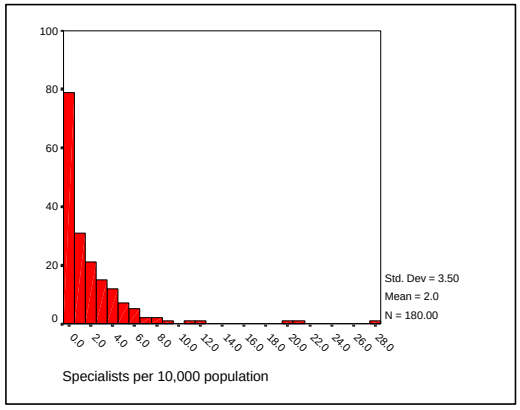
Figure 1 Differences in service use (1995)

1a GPs per 10,000 population



Source: HIC Data provided by DHAC

1b Specialists per 10,000 population



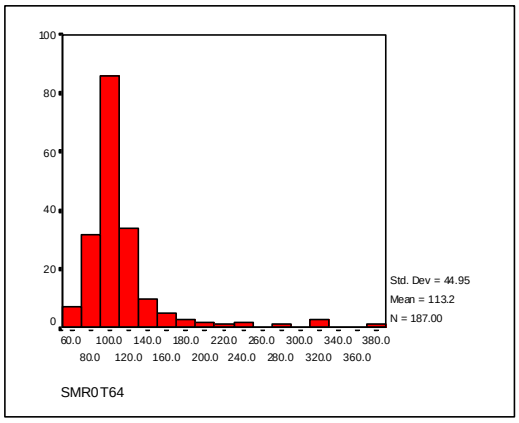
Source: HIC Data provided by DHAC

The purpose of the present paper is to investigate the hypothesis that these differences are causally related—that differences in access to medical services, as measured by the private doctor supply and the use of medical services, affects population health.

1 See Richardson et al 1991 Technical Paper 2 An evaluation of the effect of increasing doctor numbers and their geographical distribution, Centre for Health Program Evaluation, Monash University.
Richardson J 1999, 'The health care financing debate', in *Economics and Australian Health Policy*, eds G Mooney & RB Scotton, Allen and Unwin, Sydney.

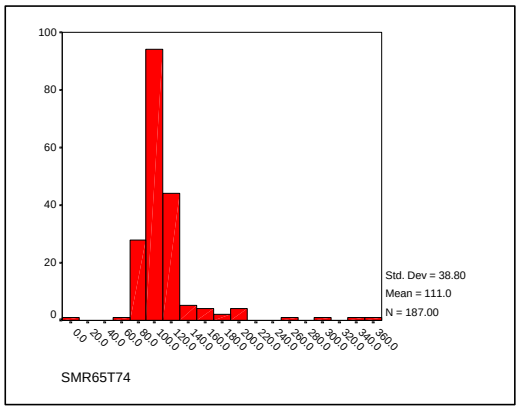
Figure 2 Standardised Mortality Ratios

2a SMR (0-64)



Source: Data file provided by ABS

2b SMR (65-74)



Source: Data file provided by ABS

It is, of course, difficult to isolate the effects of health services from the effects of the demographic and socio economic characteristics of a population[2]. Despite this, the task is important. The benefits obtained from medical care beyond some threshold level are surprisingly hard to demonstrate. Only a relatively small percentage of medical services have been rigorously tested and shown to be efficacious. The spectacular improvements in life expectancy in the last two decades are undoubtedly attributable, in part, to the introduction of effective therapies but also to improved nutrition and public health measures such as anti smoking education and legislation. Importantly, however, the introduction of a limited number of effective medical interventions does not necessarily require an expansion of the medical workforce—the opposite might even occur. If the new technologies are substitutes for earlier, labour intensive therapies, then the optimal medical workforce might contract.

A second reason for concern about the aggregate effect of marginal medical services is that studies of the aggregate relationship between medical services and mortality have not always revealed the expected relationship[3]. One of the last studies by Archie Cochrane (1978), the path breaking exponent of the randomised controlled trial, was a multi variate cross-national analysis of age specific mortality rates. Like a number of other studies the result was 'perverse': more doctors were associated with higher age specific mortality in all but one age cohort. The results have been criticised on statistical grounds. The true significance of the study may, however, be the public statement by an epidemiologist of Cochrane's stature that multi variate analysis sophisticated correlational evidence—is an appropriate methodology when there is no more powerful technique available. As a minimum, correlational evidence can support or contradict theories and, as a minimum, requires explanation.

Of particular relevance here, the first author found a statistically robust and consistent relationship between various measures of Australian mortality and the use of doctor services (Richardson and Richardson 1982). With the statistical technique used in this study, the

2 In particular, remote areas commonly rely upon public hospital and not private medical services and these are imperfectly measured by the bed supply and number of hospitals.

3 A number of the earlier studies are summarised in Richardson (1983).

direction of causation was from the doctor supply to mortality and not vice versa. The likely reason for this is discussed in Section 3 below.

The key results from this earlier study are reproduced in Table 1. Four dependent variables were used, namely, the infant mortality rate, still births and perinatal deaths per 1,000 live births and the standardised mortality ratio. Variation in each of these was 'explained' most satisfactorily with a regression equation including a quadratic term for the use of medical services, Q(GP), Q(SP) and Q(Doc). This implied a minimum death rate beyond which point deaths rose as the use of medical services increased. Table 1 reports the service use at the implied minimum.

Table 1 Services per capita at the Health Outcome Turning Point, 1976

		Q(Doc)	Q(GP)	Q(SP)
IM	OLS	5.1	3.7	1.4
	TSLS	5.1	3.7	1.5
STILL	OLS	5.7	3.8	1.6
	TSLS	5.1	3.8	1.7
PERI	OLS	5.1	3.7	1.5
	TSLS	5.4	3.7	1.5
Deaths	OLS	4.4	3.5	1.2
	TSLS	3.8	2.9	1.1
Statistical Division Average Use, 1976		5.4	3.95	1.59
Range		2.1 - 7.4	1.7 - 5.2	0.3 - 2.2

Source: Richardson and Richardson (1982)

Key	IM	Infant mortality/1,000 live births	OLS	Ordinary least square regression
	STILL	Stillbirths/1,000 live births	TSLS	Two stage least square regression
	PERI	Perinatal deaths/1,000 live births	Q(Doc)	Total doctor services/capita
	Deaths	Standardised mortality ratio	Q(GP)	GP services/capita
			Q(SP)	Specialist services/capita

To date there has been no satisfactory explanations for these results or for their consistency. (Each entry in Table 1 was obtained from a different regression equation. Despite this, the turning point for service use is, in most cases, identically equal to the sum of the GP and Specialist service use—consistency which would not normally be expected with imperfect data and statistical procedures.) A somewhat vague criticism of the study is that small area statistics suffer from the 'ecological fallacy' there may be something occurring within the geographical units which is submerged by aggregation. The criticism is more persuasive when a submerged element can be suggested which satisfactorily explains the aggregate results. However, to date, no such factor has been suggested. Despite this, the present study was motivated, in part, by the desirability of testing the results using a smaller geographical unit than the statistical division but also by the need to replicate the results with more recent and better quality data. The study below employs 1994/95 data from Australia's 187 Statistical Sub-Divisions (SSDs) in combination with ABS mortality and census data.

2 Can Doctors Increase Mortality?

A second reason for doubting the existence of the causal relationship implied in Table 1 (or more generally in any statistical association) might be that there is no sensible or persuasive reason for believing that causation is possible; that is, it may be argued that there is no plausible theory of causation. It is self-evidently true that patients die as a result of dangerous interventions and, further, there will always be a small number of deaths associated with even relatively safe procedures. However, the interesting and important question is whether or not *increasing* the doctor supply beyond some point results in greater harm than good. This idea is not new. Following the publication of his controversial book, 'Medical Nemesis', Ival Illich (1975a) popularised the idea that societies with an excessive number of doctors will suffer from social, cultural and physical iatrogenesis. His arguments were not universally embraced with Archie Cochrane representing one school of thought with the succinct summary 'Bullshit!' (Illich 1975b). At least Illich's term 'iatrogenesis' survived the interview! Despite Cochrane's damaging assessment there are at least three causal paths which might explain iatrogenesis and, indeed, Cochrane at el (1978) briefly discuss these.

2.1 Adverse Events

There is now a large literature on the adverse events associated with medical interventions. The most startling of these in Australia is the report of the 'Quality in Australian Health Care Study', published by Wilson et al (1995). 14,179 admission to 28 hospitals were individually perused to determine whether or not the admission was related to a medical error before or during the admission. Extrapolating the results to Australia, the authors estimated that 470,000 admissions occur annually because of medical mistakes. These, in turn, are associated with 18,000 deaths and 50,000 patients being permanently disabled to a greater or lesser extent. The results have been challenged as the 16.6 percent error rate is significantly above the 4.6 and 3.7 percent rates detected in two US studies (Wilson et al 1995 p468). A re-evaluation of the Australian data and a careful comparison with a Utah-Colorado study resulted in rates of 10.6 and 3.2 percent respectively (Thomas 2000). A 2001 New Zealand study which replicated the Australian methodology, obtained an adverse event rate of 12.9 percent (Davis 2001) while a recent Australian study of 1,125 patients found 'serious adverse effects' in 16.9 percent of the 1,125 patient records examined in a tertiary hospital in Melbourne (Bellomo et al 2002).

The significance of these studies is that they provide a direct and plausible causal linkage between the use of doctor services and mortality outcomes. Furthermore, the effects are of a sufficient order of magnitude that they would be observed in aggregate studies of the doctor-outcome relationship.

Indirect but persuasive evidence has also been obtained from the Health Insurance Experiment carried out at the Rand Corporation under the direction of Newhouse (Newhouse 1993). In one of the world's largest social experiments 7,706 persons were randomly assigned to health schemes with different levels of consumer copayments. Service use and health outcome were monitored for 5 years. The results were puzzling in one important respect. As expected, the demand for medical care fell when patient copayments rose. However, and contrary to expectation, the services discarded were not associated with trivial conditions. Rather, the

services which were dropped were unrelated to the need for medical care; patients stopped using services which should have had an important affect upon their health. Despite this, the experiment detected little if any effect on health outcomes (Lohr 1986). After considering a number of possible explanations, the authors concluded that the benefits from the additional services received by fully insured members of the experiment may have been off-set by the effects of medical errors.

2.2 The Dependency Hypothesis

A second possible causal link between the doctor supply and health outcome is closer to Illich's idea of social iatrogenesis. This is that with an increasing doctor supply the population will become increasingly dependent upon doctor services to maintain their health and neglect the ultimately more important lifestyle factors which are of greater importance for the prolongation of life. There are a number of reasons why this unintended and probably unconscious dependency might arise. First, the common experience of most young people will be that doctors can spectacularly alleviate or cure the problems they most commonly encounter. This may well lead to an incorrect assessment of the doctor's capacity to treat the more important risk factors later in life.

A second possible reason is that people experience a conflict akin to cognitive dissonance when they consciously or unconsciously plan their lifestyle as explained by Aronson (and quoted by Rice 1998):

> Cognitive dissonance is a state of tension that occurs when an individual simultaneously holds two cognitions (ideas, attitudes, beliefs, opinions) that are psychologically inconsistent… because (its) occurrence … is unpleasant, people are motivated to reduce it… to hold two ideas that contradict each other is to flirt with absurdity… (Rice, p76).

In the present context individuals may understand, intellectually, that certain lifestyle factors increase the risk of death. However this information is unwanted as it conflicts with the desire to live a relatively self-indulgent life. The resolution to this dilemma is to adopt an exaggerated confidence in the efficacy of medical care and its ability to off-set the harmful effects of self neglect.

It is difficult to obtain evidence to support or refute this hypothesis and, in particular, to demonstrate that the likelihood or extent of the exaggeration depends upon the present access to medical services. However an intriguing parallel issue has been studied by McCord (1978). Starting in 1939, 500 boys were randomly assigned to a control group or to counsellors who provided general assistance with life problems for 5 years. McCord reports the results of a follow up study. At interview, 30 years later, men in the intervention group provided a very positive assessment of the intervention, and typically commented on its importance for their personal development and capacity to cope with subsequent problems. Despite this, the objective evidence indicated that the program was associated with highly significant 'negative side effects as measured by criminal behaviour, death, disease, occupational status and job satisfaction' (p284). The authors suggest that the most likely explanation for the disappointing outcome was that the assistance provided by counsellors resulted in dependency and an inability to cope personally with life's problems. The dependency hypothesis is that, analogously, assistance with small order medical problems may lead to an attitude of dependency which encourages passivity and neglect of the lifestyle elements which determine risk factors and mortality.

2.3 The Substitution Hypothesis

The third theoretical pathway between an increasing doctor supply and mortality outcome is similar to the dependency hypothesis except that the substitution of doctors' services for a healthy lifestyle might be the result of rational, utility maximising behaviour. The hypothesis is summarised in Box 1. In this, the two key relationships are given by equations (1) and (2) for individual utility, U, and health status, H. Utility depends, *inter alia*, upon health status and lifestyle, L. Health depends, *inter alia*, upon the use of doctors, Doc, and the lifestyle adopted.

There are five behavioural assumptions in the model. These are that individual utility increases with health but decreases with lifestyle events adopted specifically for the purpose of increasing health (diet, additional exercise, exercise in excess of the time spent exercising for pleasure etc). Health rises with both the doctor supply and lifestyle. The fifth and pivotal assumption is that the second derivative of health with respect to lifestyle and doctor supply is negative. Restated, the beneficial effect of lifestyle on health (marginal benefit) declines with the doctor supply; that is, as doctors improve health there is less residual ill health which the individual may improve by changing their lifestyle.

Box 1 Lifestyle-Health Substitution in a One Time Period
Utility Maximising Model

$$U = U(\ldots H, L) \quad (1)$$

$$H = H(\ldots Doc, L) \quad (2)$$

Partial Effects

$$\frac{\partial U}{\partial H} > 0 ; \frac{\partial U}{\partial L} < 0$$

$$\frac{\partial H}{\partial Doc} > 0 ; \frac{\partial H}{\partial L} > 0$$

$$\frac{\partial^2 H}{\partial L \, \partial D(Doc)} = (\partial H / \partial L) / \partial Doc < 0$$

For each individual the doctor supply is exogenously determined

First order condition for maximum utility

$$\frac{\partial U}{\partial L} = \frac{\partial U}{\partial H} \cdot \frac{\partial H}{\partial L} \quad (3)$$

(Lifestyle is adjusted until (health benefits) equal lifestyle costs on the margin)

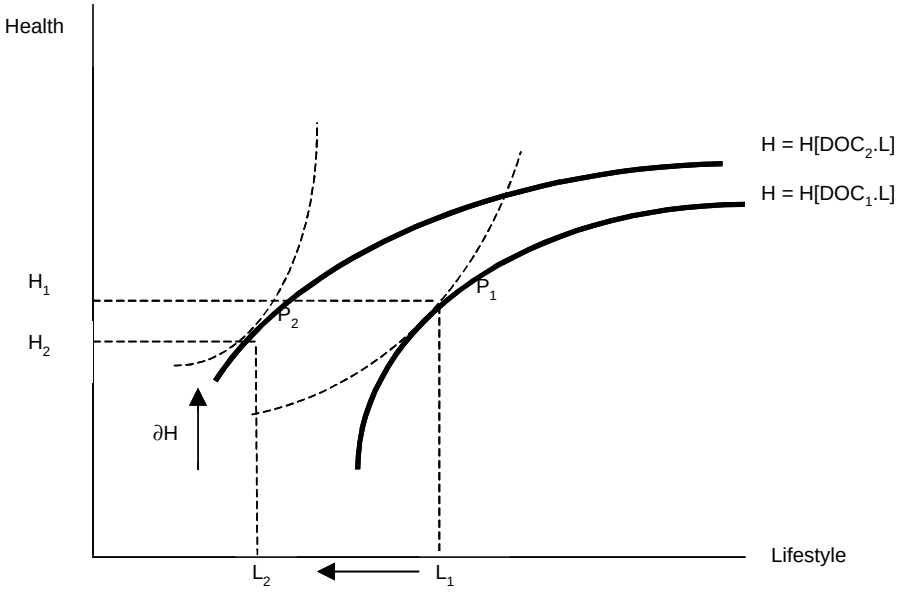
Differentiating with respect to the doctor supply (the supply increases)

$$\frac{dH}{dDoc} = \frac{dH}{d(Doc)} + \frac{dH}{dL} \cdot \frac{dL}{dDoc} \quad (4)$$

signs + + -

The effect of an autonomous increase in the doctor supply may be obtained by differentiating the first order condition(equation 3). This results in equation 4 which states that as Doc rises there is a direct positive effect upon health (dH/d(Doc)). However there is an indirect effect as the doctor supply affects lifestyle and lifestyle affects health. As noted, this effect is negative and so the sign of the overall effect of doctors upon health status is indeterminate (equation 4). This leaves open the possibility that the increase in the doctor supply will lead to an overall decrease in health because of the magnitude of this second, indirect, effect.

Figure 3 Doctor Lifestyle Substitution



The argument is illustrated in Figure 3. In this, the unbroken lines represent the health 'production function'. The two such functions depicted correspond with two levels of the doctor supply where $Doc_2 > Doc_1$. With both functions health rises with lifestyle. Critically, the gap between the two functions narrows: as the doctor supply rises there is less ill health amenable to improvement. In the limit both functions are asymptotic to full health. The broken positive sloping lines represent the 'indifference curves' combinations of lifestyle and health which give equal utility. The positive slope reflects the negative direct effect of lifestyle activities upon utility. Maximum utility is achieved at the point of tangency between the indifference curves and the health production function. As depicted, the higher utility is obtained at P_2 where both lifestyle and health are lower.

The intuition behind this model is relatively simple. The increase in the doctor supply changes the trade-off between lifestyle sacrifice and health. At the initial equilibrium level of lifestyle, L, the slope of the production function is reduced and a given reduction in health can be traded off against a larger reduction in lifestyle activities without loss of utility. If the reduction is sufficiently large then, as shown, the indirect reduction in health, sacrificed in exchange for lifestyle benefits, may outweigh the direct, marginal benefits from an increase in the doctor supply. The argument is intuitively more appealing in a two time period model in which future effects are discounted. It is easy to show that in such a model, the future adverse effects of an unhealthy lifestyle may be discounted and traded off against an increase in immediate undiscounted satisfaction and, particularly, when present ill health arising from a self indulgent lifestyle may be quickly and

effectively cured by medical intervention. In contrast with the dependency hypothesis this choice is 'rational' in the sense that it is based upon consistent behaviour and with full knowledge of the consequences of different choices.

3 Model, Data and Variables

The structure of the model underlying the econometric analysis is presented in Box 2. In this the supply and demand for medical services are in equilibrium. Supply is a function of the gross medical fee, the doctor supply, and other supply side variables, S. Demand is likewise a function of the usual demand side variables but also includes the doctor supply. That is, equation 3 incorporates the theory of supplier induced demand. (Without it, our estimated demand equations fail the test discussed later for omitted variable bias.)[4] Equation 4 for the doctor supply includes a set of variables (location) to measure the desirability of a residential location. A decrease in the value of these variables may be conceptualised as an increase in the subjective cost of living in an area and, as in standard economic theory, this reduces supply. Finally, standardised mortality rates are a function of the doctor supply or the use of medical services (Q) and exogenous variables (M). In equations 5 and 6 the relevant variable and the variable squared are both included to detect the possibility of a change in the impact upon the SMR beyond a particular value of the doctor supply/use of medical services.

Box 2 Hypothesised Model

$$Q_S = Q_D \quad \ldots (1)$$

$$Q_S = a_0 + a_1 \hat{fee} + a_2 D\hat{oc} + a_3 S + e_1 \quad \ldots (2)$$

$$Q_D = b_0 - b_1 \hat{fee} + b_2 D\hat{oc} + b_3 D + e_2 \quad \ldots (3)$$

$$Doc = c_0 + c_1 \hat{fee} + c_2 location + e_3 \quad \ldots (4)$$

$$SMR = d_0 + d_1 D\hat{oc} + d_2 (D\hat{oc})^2 + d_3 M + e_4 \quad \ldots (5)$$

OR

$$SMR = d_4 + d_5 \hat{Q} + d_6 \hat{Q}^2 + d_7 M + e_5 \quad \ldots (6)$$

From equations (1), (2) and (3) earlier

$$Doc = \left(\frac{1}{a_2 + b_2} \right) (b_0 - a_0) + -(b_1 + a_1) fee + (b_3 D - a_3 S) \quad \ldots (7)$$

Key See Table 1

S = exogenous supply side variables
D = exogenous demand side variables
M = exogenous mortality variables
Doc = doctor supply/10,000 (G or Specialist)
^ endogenous variable

The existence of a set of variables which reflect the desirability of a location but which are independent of demand or mortality is of pivotal importance in determining the direction of causation and, consequently, for determining the persuasiveness of the statistical argument. In the two stage least squares regression equations reported below the doctor supply is not measured by the observed supply in an SSD but by the supply predicted on the basis of the

[4] For a discussion of this and, particularly, the identification of supplier induced demand in such a system of equations see Richardson and Peacock (1999).

location and other variables in the doctor supply equation 4. Consequently if there is an element of reverse causation in the real market—if doctors do move into regions where they believe mortality to be higher—then this will not be reflected in the data used in stage 2 to measure supply (unless, in principle, it affected one of the 'other' variables in the supply equation in which case this variable would be independently included in the mortality equation). Consequently, there is a clear direction of causation: an autonomous improvement in the congeniality of a 'location' increases the doctor supply thereby improving access. This, in turn, will increase the use of medical services, either directly (supplier induced demand) or indirectly (via a reduction in fees and queuing times). Access and service use will impact upon the standardised mortality rates.

Data and Variables: The econometric analysis used data from 187 statistical sub-divisions in Australia (see Appendix). The variables used and their definitions are shown in Box 3. Three major data sources were employed. The first of these were two samples of the billing records for 1994/95 collected by the Health Insurance Commission. These cover 100 percent of private medical services in Australia.. The first of these was used to construct the utilisation statistics, QGP, QSP, QGP (Age Sex) and QSP (Age Sex). The second sample was used to construct full time equivalent GPs and Specialists in each statistical sub-division (Richardson and Peacock 1999). When doctors worked in two separate locations they were assigned to these in proportion to the workload. The third major data source was the 1991 Australian Census. This was used to construct the various demographic and socio economic variables for each SSD. In addition, information was obtained concerning mortality and the location of prestigious private schools as these are known to be located in SSDs which are congenial to doctors.

The mean value of the key variables are shown in Table 2 and selected correlations are given in Table 3. Further summary statistics are reproduced in the Appendix.

Table 2 Mean Values of Variables

Variable	Mean	Variable	Mean
INFSMR	107.09	Low Inc	0.18
SMR 0-64	113.24	Eco Res	0.97
SMR 65-74	111.03	ATSI (%)	3.90
SMR 75+	104.15	Urban Adv	0.97
GP	5.98	Schools	0.68
SP	2.03	Ed	3.98
QGP	4.96	HB/10,000	37.57
QSP	1.22	Hosp/10,000	0.99
QGP (Age Sex)	5.70		
QSP (Age Sex)	1.52		

Box 3 Variables used in the Statistical Analysis

Mortality Variables

SMR	Indirectly standardised Standardised Mortality Ratios (ratio of observed to expected deaths, National average=100)
INFSMR	SMR < 1 year old
SMR 0-64	SMR 0 - 64 years old
SMR 0-74	SMR 0 - 74 years old
SMR 65-74	SMR 65 - 74 years old
SMR 75+	SMR 75+ years old

General Practitioner Variables

GP	GP Supply = General Practitioners per 10,000 population
Fee GP	Gross Price = Fee charged per GP service
Q/GP	GP Workload = Number of GP services provided / Provider Count
QGP	GP Utilisation = GP services per capita
QGP (Age Sex)	Age/sex expected GP services per capita
Provider Count	If provider is full-time then PCOUNT = (1.00), If provider is part-time then PCOUNT = (Schedule Fee)/(total Schedule Fee) x FTE factor
BB GP	Percent of GP services Bilk Billed

Specialist Variables

SP	Specialist Supply = Specialists per 10,000 population
Fee SP	Gross Price = Fee charged per Specialist service
Q/SP	Specialist Workload = Number of Specialist services provided/ Provider Count
QSP	Specialist Utilisation = Specialist services per capita
QSP (Age Sex)	Age/sex expected Specialist services per capita
Provider Count	If provider is full-time then PCOUNT = (1.00), If provider is part-time then PCOUNT = (Schedule Fee)/(total Schedule Fee) x FTE factor
BB SP	Percent of Specialist services Bilk Billed

Socio-economic and Location Variables

ATSI	Percent Aboriginal and Torres Strait Islanders (1991 census)
Rural	Rural Dummy Variable (Urban = 0, Rural = 1)
Low Inc	Proportion Low income (<$345 pw) (1991 census)
Eco Res	SEIFA Index 3 – Economic Resources
Urban Adv	SEIFA Index 2 - Urban Advantage
Ed	Percent with Higher Degree (1991 census)
Pop Dens	Population Density (population per km^2) x 100
Schools	Private Schools (Number non-government schools: no funding/funding level 1) x 100
State Dummy Vars	NSW, VIC, QLD, SA, WA, TAS, NT

Hospital Variables

Hosp/10,000	Hospitals per 10,000 population
HB/10,000	Hospital Beds per 10,000 population
Med/School	Medical School Dummy Variable (Medical School present = 1, Not = 0)

Table 3 Selected Correlation Coefficients

3a Mortality

	INFSMR	SMR 0-64	SMR 65-74	SMR 75+
GP	-0.35**	-0.38**	-0.41**	-0.18*
SP	-0.11	-0.10*	-0.13*	-0.09
QGP	-0.41**	-0.52**	-0.49**	-0.19**
QSP	-0.40**	-0.50**	-0.49**	-0.24**
QGP (Age Sex)	0.37**	-0.41**	-0.48**	-0.16
ATSI	0.76**	0.91**	0.76**	0.16
Rural	0.18*	0.30**	0.24**	0.25**
Low Inc	0.09	0.20**	-0.01	0.05
Eco Res	-0.33**	-0.50**	-0.33**	-0.19*
Urban Adv	-0.34**	-0.49**	-0.41**	-0.20**
Ed	-0.87	-0.13**	-0.13*	-0.14*
HB/10,000	0.03*	0.14**	0.11*	0.08
Hosp/10,000	0.08*	0.25**	0.21**	0.16**

3b Doctor Supply

	GP	SP		GP	SP
GP	1.00	0.69**	Low Inc	-0.10	-0.23**
SP	0.69**	1.00	Eco Res	0.39**	0.33**
Fee GP	-0.20**	-0.58	ATSI	0.50**	-0.19**
Fee SP	0.28**	0.30**	Rural	-0.55**	-0.38**
BB GP	0.33**	0.20**	Urban Adv	0.47**	0.42**
BB SP	0.10	0.04	Schools	0.48**	0.58**
Q GP (Age Sex)	0.58**	0.34**	Ed	0.43**	0.60**
QSP (Age Sex)	0.52**	0.29**	HB/10,000	-0.17	0.43*
Q/GP	0.03	0.04	Hosp/10,000	-0.37**	-0.26**
Q/SP	0.33**	0.33**			

Key: significance level * = p<0.05 ** = p<0.01

4 Results

Results for the doctor supply—equation 4 in Box 2—are reported in Table 4 and results for mortality—equations 5 and 6—are given in Tables 5-7.

4.1 Doctor Supply

GP Supply: All of the regression results reported in Table 4 have very high explanatory power and most of the variables have the expected sign. There are more general practitioners in SSDs with an elderly population (QGP (Age Sex)). Likewise there are more GPs in low income SSDs where medical need is greater and bulk billing is the norm. The supply of GPs increases with the number of hospital beds in an SSD, which are a complementary product for GP services, especially in rural areas. In their pioneering work in this area Fuchs and Kramer (1972) suggested that doctors may be disinclined to work in areas where the unavoidable workload was too large. Consistent with this, the workload variable (Q/GP) enters the GP equations with a negative sign.

Table 4 Regression Results: The Supply of GPs and Specialists

	GPs per 10,000 population				Specialists per 10,000 population			
	OLS		2SLS		OLS		2SLS	
Independents	**4.1**	**4.2**	**4.3**	**4.4**	**4.5**	**4.6**	**4.7**	**4.8**
Fee	-0.19***	-0.15**	-0.56***	ns	0.04***	0.03***	0.06***	ns
QGP (Age Sex)	2.49***	1.72***	2.77***	ns	-	-	-	-
QSP (Age Sex)	-	-	-	-	4.75***	3.28**	5.27***	ns
Low Inc	-	14.44***	-	17.72**	-13.05**	ns	-12.66*	ns
Eco Res	ns	16.82***	ns	19.98**	ns	ns	ns	ns
Pop Dens	5.25***	6.19***	4.41**	7.27***	-	5.12**	-	5.85**
Ed	0.15**	0.15**	0.23**	ns	-	0.23**	-	0.35**
ATSI	-6.08***	-5.44***	-6.34***	-5.34***	ns	ns	ns	ns
Schools	ns	ns	ns	ns	0.49***	0.32***	0.45***	0.26**
Med School	ns	ns	ns	ns	0.94**	0.86**	ns	ns
Urban Adv	-9.65***	-15.10***	-12.16***	-16.06***	ns	ns	ns	ns
Rural	-0.81***	-0.83***	ns	ns	ns	ns	ns	ns
Q/GP	-3.78***	-4.19***	ns	-9.84**	-	-	-	-
Q/SP	-	-	-	-	1.11**	1.59***	ns	6.15*
Hosp/10,000	ns	-0.14*	ns	ns	-0.45***	-0.42***	-0.56***	ns
HB/10,000	ns	0.01***	ns	0.01**	0.03***	0.02***	0.03***	0.02**
QLD	0.85***	1.02***	1.22***	1.09***	-	ns	-	ns
R^2 Adj	***0.69***	***0.70***	***-***	***-***	***0.69***	***0.71***	***-***	***-***

Key: (***), (**), (*) = Significant at 1%, 5%, 10%

Table 5 Regression Results: SMR Age 0 - 64

	Dependent Variable: SMR 0-64								
	GP Models OLS					Specialist Models OLS			
Independents	**5.1**	**5.2**	**5.3**	**5.4**	**Independents**	**5.5**	**5.6**	**5.7**	**5.8**
GP	-15.06***	-15.98***			SP	-1.96*	ns		
GP^2	1.21***	1.36***			SP^2	0.48***	0.40***		
Q(GP)			-24.56***	-27.90***	QSP			ns	ns
Q/GP^2			2.28***	2.72***	$Q(SP)^2$			ns	ns
QGP (Age Sex)	21.16***		23.59***		QSP (Age Sex)	42.80***		40.40***	
HB/10,000	0.11**	0.10**	0.13***	0.13***	HB/10,000	0.07*	ns	0.12***	0.13***
Urban Adv	-139.69***	-71.94**	-130.01***	ns	Urban Adv	-160.40***	-99.63***	-151.18***	-113.13***
ATSI	4.61***	4.44***	4.42***	4.18***	ATSI	4.55***	4.32***	4.47***	4.27***
% Low Inc		95.88***		103.15***	% Low Inc		97.86***		85.28***
R^2 Adj	***0.86***	***0.86***	***0.85***	***0.85***	***R^2 Adj***	***0.88***	***0.88***	***0.87***	***0.87***
Turning Point	***6.22***	***5.86***	***5.39***	***5.13***	***Turning Point***	***2.04***	***-***	***-***	***-***
Test Statistic	*0.03*	*1.16*	*0.27*	*0.53*	*Test Statistic*	*0.00*	*0.50*	*0.01*	*0.33*

Key: (***), (**), (*) = Significant at 1%, 5%, 10%

Table 6 Regression Results: SMR Age 65 - 74

	Dependent Variable: SMR 65-74								
	GP Models OLS					Specialist Models OLS			
Independents	**6.1**	**6.2**	**6.3**	**6.4**	**Independents**	**6.5**	**6.6**	**6.7**	**6.8**
GP	-26.11***	-24.88***			SP	ns	ns		
GP^2	1.88***	1.73***			SP^2	ns	ns		
Q(GP)			-64.28***	-61.14***	QSP			-65.98***	-65.38***
Q/GP^2			5.70***	5.33***	$Q(SP)^2$			20.53***	18.51***
QGP (Age Sex)	-15.37*		-14.03*		QSP (Age Sex)	-32.16**		-27.99*	
HB/10,000	0.15**	0.17***	0.17**	0.17**	HB/10,000	0.17**	0.21***	0.15**	0.17***
Urban Adv	ns	-105.13***	ns	-97.41**	Urban Adv	ns	-119.30***	ns	-111.64**
ATSI	2.14***	2.25***	1.19***	1.33***	ATSI	2.42***	2..52***	1.65***	1.73***
% Low Inc		-92.35***		-79.28**	% Low Inc		-115.49***		-107.86***
R^2 Adj	***0.58***	***0.58***	***0.60***	***0.61***	***R^2 Adj***	***0.52***	***0.53***	***0.55***	***0.56***
Turning Point	***6.94***	***7.19***	***5.64***	***5.74***	***Turning Point***	***-***	***-***	***1.61***	***1.77***
Test Statistic	*10.22*	*6.45*	*11.59*	*8.39*	*Test Statistic*	*0.79*	*2.17*	*17.63*	*23.95*

Key: (***), (**), (*)(^{o}) = Significant at 1%, 5%, 10%

Variables measuring *'location'* are highly significant. Population density, the SEIFA index of Urban Advantage, the percent Aboriginal and Torres Strait Islanders (ATSI) and the rural dummy are all significant, as is Ed the variable measuring educational attainment. In contrast the variable 'schools', the number of prestigious private schools relative to population, is not independently significant. Finally, the variable 'fee' enters GP equations with a significant but perverse sign. This suggests that, contrary to expectation, the variable is not measuring the effect of fees upon supply but, possibly, the effect of an increase in supply upon fees. In principle, this might bias other parameter estimates. However, this possibility was tested by removing 'fee' from GP supply models, which did not result in significant changes to other coefficients.

Specialist Supply: In contrast with this latter result, there is a positive association between specialist fees and specialist supply. However this is not an unambiguous confirmation of a positively sloped supply curve. A detailed analysis of price formation in the medical market found evidence suggesting that the association is attributable to the spurious correlation of both fees and the specialist supply with the complexity of the specialist service; that is, super specialist services may be concentrated in particular locations where their higher fees drive up the average observed fee. Also contrasting with the results for GPs there is no consistent association between the specialist supply and workload. As expected, specialists concentrate in the high prestige suburbs indicated by the existence of a private school, and in areas with fewer low income residents, higher education levels and greater population density.

Two stage least squares supply equations were tested using a general test for misspecification (Godfrey, 1988). The test consists of a null hypothesis that the equation is correctly specified, and a general specification of the alternative hypothesis. Test statistics for equations 4.3 and 4.4 were 5.75 and 2.52 respectively, and for equations 4.7 and 4.8, 8.71 and 8.79 respectively. All equations were very well specified (χ^2_{150} (0.05) critical value = 179.58).

4.2 Mortality

Returning to the hypothesised model in Box 2, the equations of primary interest in this study are the mortality (SMR) equations (5) and (6). The model has a block recursive structure (Maddala, 1988), where Q, fee and DOC are endogenous variables, and estimation of equations (2) - (4) requires the use of instrumental variable techniques due to correlation in the error terms. However, the error terms in equations (5) and (6) are not correlated with the error terms in (2) – (4) because SMR does not appear in these equations. Equations (5) and (6) can therefore be estimated using Ordinary Least Squares. To test this, supply and demand equations were estimated including SMR variables. All SMR variables were not found to be significant, confirming the block recursive structure for the model.

SMR Age 0 - 64: An extremely large proportion of the variation in the SMR for the population cohort aged 0 to 64 is explained by each of the equations in Table 5, which explore various combinations of the explanatory variables. The results accord with expectations in the case of the age sex variable, hospital beds, Urban Advantage, Aboriginality and income. Both the GP supply and the use of GP services are significant in their quadratic form implying that past a minimum level the SMR rises. When the specialist supply replaces GPs, the quadratic form of the equation is only significant in equation 5.5. Replacing the doctor supply with service use per capita does not result in a significant quadratic fit for specialist services (equations 5.7, 5.8).

SMR Age 65 - 74: Similar results are obtained in the eight regression equations reported in Table 6 for the SMR between ages 65 and 74. In the GP equations the higher coefficients on both the GP supply and GP use variables implies a higher turning point than in the equations for SMR Age 0 to 64. In contrast with the specialist equations for SMR Age 0 to 64, specialist supply variables are not significant for SMR Ages 65 to 74. However, specialist use equations (equations 6.7, 6.8) have significant and large linear and quadratic terms, again implying a relatively high turning point.

SMR Age 75+: None of the regression equations explaining mortality for the population cohort aged 75 or above resulted in a significant coefficient on either the doctor supply or the use of doctor services.

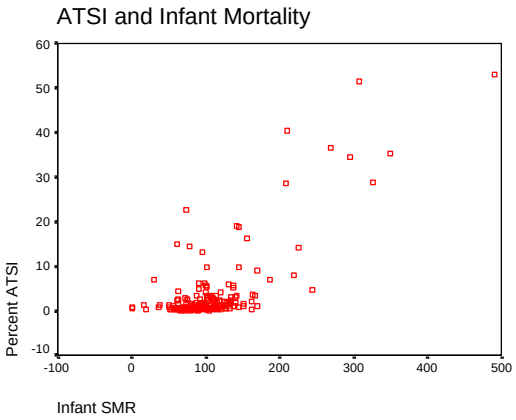
Infant Mortality: Between 42 - 49 percent of the variation in infant mortality was explained by the regression equations reported in Table 7. However this is almost entirely attributable to the extraordinary correlation between infant mortality and the percent of the population which are Aboriginal or Torres Strait Islanders (ATSI). This result is shown in Figure 4.

Table 7 Regression Results: Infant Mortality

Dependent Variable: Infant SMR					
	GP Models OLS			Specialist Models OLS	
Independents	**7.1**	**7.2**	**Independents**	**7.3**	**7.4**
GP	ns		SP	ns	
GP^2	ns		SP^2	ns	
QGP		ns	QSP		ns
QGP^2		ns	QSP^2		ns
QGP (Age-sex)	ns		QSP (Age-sex)	ns	
HB/10,000	ns	ns	HB/10,000	ns	ns
Urban Adv	ns	ns	Urban Adv	ns	ns
ATSI	4.61***	4.13***	ATSI	4.25***	4.23***
R^2 Adj	***0.48***	***0.49***	***R^2 Adj***	***0.44***	***0.42***
Test Statistic	*0.10*	*0.71*	*Test Statistic*	*0.42*	*0.48*

Key: (***), (**), (*) = Significant at 1%, 5%, 10%

Figure 4 Infant Mortality, Aboriginal and Torres Strait Islanders



The various turning points implied by the quadratic equations are summarised in Table 8 which also includes, for comparison, the mean value and range of the variables.

Mortality equations were tested for misspecification using a form of the RESET test (Horowitz, 1994). Test statistics are reported in tables 5-7. All SMR 0-64 and Infant SMR equations proved to be well specified ($F_{1,150}$ (0.05) critical value=3.904, $F_{1,150}$ (0.01) critical value=6.807). SMR65-74 equations proved to be less well specified, with signs of misspecification in five of the equations estimated.

Spurious Correlation: An unavoidable problem with statistical analyses of the type presented here is that the effects attributed to a pivotal variable—(doctor supply or service use in the present study) may be artefacts arising from their correlation with other variables. The criticism is only persuasive when the omitted variables are nominated. A further possibility is that the apparent effect of the variable is the result of its correlation with other variables in the study—the coefficient is an artefact of the interaction of different variables. A stringent test of this possibility is to omit the pivotal variable from a regression equation to determine whether or not the coefficient on the confounding variables changes. Further, the residual from such a regression may be used as the dependent variable in a second stage regression to determine whether or not the pivotal variable has additional explanatory power *after the effects of the potentially confounding variables have been eliminated.* Results of such an analysis are reported in Table 9. The results strongly refute the hypothesis of confounding in the GP regressions. The insignificant results for specialists indicate that confounding is possible. However an insignificant result indicates ambiguity, not disproof[5].

[5] The result indicates that the specialist supply is sufficiently correlated with confounding variables that its effect is fully attributed statistically to these variables in the stage 1 regression. The result does not discriminate between the hypotheses that The specialist supply does/does not have an independent effect.

Table 8 Doctor Supply (Doctors/10,000 population) with Minimum Mortality

		Minimum Mortality		Observed Supply	
	Eqn	SMR (0-64)	SMR (65-74)	Mean	Range
GP	1	6.22	6.94	5.98	1.13 - 13.50
	2	5.86	7.19		
SP	1	2.04	-	2.03	0.00 - 27.82
	2	-	-		

Doctor Utilisation (Services/capita) with Minimum Mortality

		Minimum Mortality		Observed Utilisation	
	Eqn	SMR (0-64)	SMR (65-74)	Mean	Range
QGP	1	5.39	5.64	4.96	1.23 - 8.41
	2	5.13	5.74		
QSP	1	-	1.61	1.22	0.13 - 2.77
	2	-	1.77		

Table 9 Regression Results: Analysis of Residuals (OLS)

	Residual GP		Residual SP	
Independents	**SMR (0-65)**	**SMR (65-74)**	**SMR (0-65)**	**SMR (65-74)**
GP	-5.61**	-10.79***		
GP^2	0.53***	0.70***		
SP			ns	ns
SP^2			ns	ns
	0.06	0.64		

Key: (***), (**) = Significant at 1%, 5%

5 Discussion

The data summarised in Table 8 represents striking support for the hypothesis that, after standardising for other relevant variables, an increase in the doctor supply is associated with increasing mortality. The results are both stable and highly significant in the statistical sense. Comparing the results with those in Table 1 suggests that while the turning point has risen the pattern has remained relatively unchanged over time. The insignificance of the relationship in the case of infant mortality may be attributed to the major advances that have been made in perinatal technologies and procedures.

The results raise three questions. First does an aggregate statistical analysis represent a satisfactory basis for asserting a causal relationship from the doctor supply to mortality outcome? Can causation be asserted when there is no variable which measures the transmission mechanism (adverse event or index of dependency)? Secondly, if such causation is accepted which of the alternative mechanisms discussed earlier is best supported by these results? Finally, what policy conclusions would be implied by the existence of such a causal relationship?

The problem of causation is common to most empirical work in the social sciences. Econometrics is a sub-discipline which deals, essentially, with highly sophisticated correlational evidence. However, the problem is not confined to the social sciences. Randomised controlled trials—widely regarded as the gold standard for establishing the causal relationships after a medical intervention—sets a conventional statistical threshold for the rejection of the null hypothesis and acceptance of an alternative, hypothesised, causal relationship. As noted in the introduction, the persuasiveness of the evidence commonly depends upon the persuasiveness of the theoretical relationships *tested* (not proved) by the statistical evidence. Randomised controlled trials are highly persuasive because the underlying theory is compelling: under controlled conditions and when only one variable is altered, any difference in outcomes can reasonably be attributed to this variable. More generally, correlational evidence per se is seldom challenged when the theory is self evident or persuasive. The same statistical evidence in a different problem-context may be less persuasive, but the reason for this is the theoretical plausibility not the 'validity of the statistics' per se. For a contentious hypothesis or theory the most persuasive statistical evidence is obtained from a discriminating test: a statistical result which supports one theory and *contradicts* the competitive hypothesis. This may be achieved if one theory implies a significant positive coefficient on a pivotal variable and the competitive theory implies a significant negative coefficient. Using this criterion the results presented here and their consistency with the earlier results support the hypothesis of a causal relationship. Importantly, the present results are not based upon *simple* correlational evidence. The first stage of the two stage least squares procedures employed here indicates that there is a large autonomous component in the doctor supply associated with the doctor's choice of location: doctors like living and working in professionally and personally congenial locations as indicated by variables which are not directly related to medical need or mortality. To the extent that this element of the doctor supply is associated with higher mortality the causation cannot run from mortality to the doctor supply. Other causal elements, such as the age-sex and the socio economic characteristics of populations are separately accounted for in the final regressions and there remains no obvious alternative causal path.

The statistical results shed some light upon the second question; viz, if causation is accepted then which of the three hypothesised causal paths is most consistent with the data? Adverse events—clinical iatrogenesis—is generally associated with in-hospital errors. More generally it is likely that lethal mistakes will be associated with the more complex and dangerous interventions carried out by specialists. In contrast, the dependency hypothesis arises from excessive confidence in the efficacy of medical care obtained through easy access to services which provide immediate and relatively spectacular cures for minor problems. That is, dependency is likely to arise because of access to the primary care provided by general practitioners. In its simplest form the third, substitution hypothesis, implies full information and, to the extent that it is specialist services which will be received in the case of fatal illnesses, it is the specialist supply that would trigger this causal mechanism. However it is unlikely that the local specialist supply in an SSD would be as influential as the availability of specialist services city or state wide (patients may cross SSD borders!). More realistically, patients do not have full information and the most plausible, non-clinical explanation of iatrogenic effects probably involves a combination of the substitution and dependency hypotheses: people 'rationally' trade health for an indulgent lifestyle but with an erroneous and exaggerated belief in the efficacy of modern medicine.

Recent research findings increase the plausibility of this dependency/substitution thesis. Following Fogel's path breaking research it is now known that nutrition has been the major

determinant of mortality in the last 200 years. More recently health has been closely linked to lifestyle. Kenkel (1995), for example, demonstrates the health effects of eating habits, smoking, and exercise. Sturm (2002) and Sturm and Wells (2001) demonstrate that obesity is now a more serious problem in the USA than smoking, heavy drinking and poverty. While it is not discussed by the authors, obesity may well be linked to the over-consumption of 'stress alleviating foods' and, consequently, to the effects of the workplace and lifestyle upon individual stress.

The possible policy implications of these results depend upon the causal path of the iatrogenic effects and also upon social values. This two way policy dependence is shown in Table 10. If the principle problem is clinical iatrogenesis then the solution is, in principle, straight forward. Intensive quality assurance programs should be reinforced with the objective of implementing a form of evidence based medicine which is conservative and minimises clinical interventions.

Table 10 Policy Implications of Iatrogenesis

Values / Cause	Libertarian	Paternalistic
Clinical	Information	QA, EBM Conservative Medicine
Rational Substitution	nil	Health Promotion Education
Misinformed Dependency	Information	as above + information

The implications of the substitution hypothesis are more contentious. In a libertarian world where only utility maximisation was the individual and social objective there would be little need for interventions other than the provision of accurate information. Subject to this caveat, the reason for an increase in utility would be unimportant. Doctors would be valued if they allowed people to increase their utility by living more self indulgently and any other consequences foreseen and accepted by patients would not be of social concern. It is, however, unlikely that this scenario describes social values—it is unlikely that a collectively financed national health scheme would be socially acceptable if its benefits were known to be a more self indulgent and shorter life. Rather, it is likely that the appropriate response would be a significant increase in the resources devoted to health promotion with the objective of encouraging a better understanding of the use and role of medical services and the need for individuals to take 'ownership' of their own health.

6 Conclusions

Systemic evidence is surprisingly consistent. It implies an association between mortality and an increase in the doctor supply which is not easily attributed to reverse causation or to a spurious correlation with some other attribute of the population. The cross-sectional evidence seems to be stable. The present results are largely consistent with those obtained from data 20 years ago. Ideally, further research is required using panel data. However until this is completed and the evidence presented here is contradicted, then the hypothesis that iatrogenic effects may more than off-set the direct beneficial effects of additional, and largely unregulated, medical services must be contemplated seriously. May be Ival Illich got it right!

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Appendix

Small Area Definitions

Small area data used in empirical analysis is based on the hierarchical structure of the Australian Standard Geographical Classification (Australian Bureau of Statistics, 1999). In non-census years the classification consists of Statistical Local Areas (SLAs), Statistical Sub-Divisions (SSDs), Statistical Divisions (SDs), and States/Territories. Under the hierarchical structure SLAs are aggregated to form SSDs, SSDs are aggregated to form SDs, and SDs aggregate into States and Territories. These spatial units cover all of Australia without gaps or overlaps. As at 1999, there were 1,331 SLAs, 194 SSDs, and 66 SDs covering mainland and offshore Australian States and Territories. SLAs and SSDs are based on defining regions that show social and economic homogeneity by identifiable links between inhabitants, and on local government boundaries. SDs also maintain this basis, but in addition the capital city of each State/Territory is defined as a single SD.

Descriptive Statistics – Mortality Variables

	N	Range	Minimum	Maximum	Mean	Std. Deviation
INFSMR	187	490.20	0.00	490.20	107.09	58.00
SMR (0-64)	187	325.51	61.32	386.83	113.24	44.95
SMR (0-74)	187	328.17	52.40	380.57	112.18	41.67
SMR (65-74)	187	359.63	0.00	359.63	111.03	38.80
SMR (75+)	187	229.54	46.11	275.65	104.15	19.66

Descriptive Statistics – General Practitioner Variables

	N	Range	Minimum	Maximum	Mean	Std. Deviation
GP	187	12.36	1.13	13.50	5.98	1.84
Fee GP	187	11.23	21.54	32.77	25.42	1.88
BBGP	187	86.41	10.56	96.97	68.30	16.69
Q/GP	187	60.47	62.01	12.24	79.79	7.90
QGP	187	7.18	1.23	8.41	4.96	1.24
QGP(Age sex)	187	1.32	5.00	6.32	5.68	0.28

Descriptive Statistics – Specialist Variables

	N	Range	Minimum	Maximum	Mean	Std. Deviation
SP	180	27.82	0.00	27.82	2.03	3.49
Fee SP	178	161.03	38.47	199.50	75.31	16.89
BBSP	187	43.85	8.52	52.37	28.18	9.74
Q/SP	135	62.50	15.96	78.46	44.84	9.02
QSP	187	2.64	0.13	2.77	1.22	0.47
QSP (Age Sex)	187	0.77	1.13	1.90	1.52	0.15

Descriptive Statistics – Socio-economic and Location Variables

	N	Range	Minimum	Maximum	Mean	Std. Deviation
ATSI	187	52.88	0.10	52.98	3.90	8.38
Rural	187	1.00	0.00	1.00	0.70	0.46
Low Inc	187	0.28	0.05	0.33	0.18	5.69
Eco Res	187	0.39	0.81	1.20	0.97	6.17
Urban Adv	184	0.37	0.83	1.21	0.97	5.32
Ed	187	14.65	1.28	15.93	3.98	2.72
Pop Dens	187	41.00	0.00	41.00	3.49	7.39
Schools	187	11.00	0.00	11.00	0.68	1.46

Descriptive Statistics – Hospital Variables

	N	Range	Minimum	Maximum	Mean	Std. Deviation
Hosp/10,000	187	8.40	0.00	8.40	0.99	1.28
HB/10,000	187	176.01	0.00	176.01	37.57	29.91
Med School	187	0.00	0.00	10.00	5.65	23.00