1 Modelling the aggregate impact of medical technology on expenditure

This appendix outlines the methodology, data and results of the quantitative analysis undertaken by the Commission to estimate the impact of advances in medical technology on healthcare expenditure over the last decade. The Commission has adopted two approaches to quantifying the impact of advances in technology: the residual approach and the direct approach. The methods and results for both approaches are detailed in sections 1.1 and 1.2 respectively. Results are summarised and analysed in chapter 3.

The data series and sources used for both approaches are summarised in table 1.9 at the end of this paper.

1.1 The residual approach

The residual approach quantifies the impact of non-technology determinants on healthcare expenditure. The component of expenditure not explained by these variables (the residual) is attributable largely to changes in technology. Table 3.1 summarises previous residual studies, including the other determinants of healthcare expenditure controlled for and the magnitude of the estimated residual.

Methodology

The Commission has calculated the residual using average annual growth rates for real healthcare expenditure and each of its determinants. A continuous compound rate is assumed (Chiang 1984):

$$V_t = V_0 e^{gt} \Rightarrow g = \frac{1}{t} \ln \frac{V_t}{V_0}$$

where V_t = the value of the variable in the final year

V_0 = the initial value of the variable

g = the average annual growth rate

t = the number of years of growth (length of the period in years -1).

The growth rates of the various factors sum to give the growth rate in real health expenditure in the following fashion:

$$G = \sum_{i=1}^{n} \varepsilon_i g_i + R = \varepsilon_1 g_1 + \varepsilon_2 g_2 + ... + \varepsilon_n g_n + R$$

where G = the average annual growth rate of real healthcare expenditure

ε_i = the elasticity (or responsiveness) of real healthcare expenditure with respect to factor i

g_i = the average annual growth rate of factor i

R = the average annual growth rate of the residual.

The percentage point contribution of the residual to growth is calculated by subtracting the impact of all the determinants (their growth rates multiplied by their elasticities) from the growth rate in real healthcare expenditure. The residual can also be expressed as a percentage of the average annual growth in health expenditure, that is: $\frac{R}{G} \times 100$.

Determinants of healthcare expenditure and their elasticities

The Commission has considered the following potential drivers of real healthcare expenditure growth:

- population growth;
- ageing and gender shifts;
- health sector inflation in excess of economywide inflation;
- real income (GDP) growth; and
- the proportion of the population with private insurance.

The discussion below provides more information on the relationships between these variables and real healthcare expenditure, in particular, the elasticities the Commission has assumed in its calculations. Information on the data series used is presented in table 1.9 at the end of this appendix.

Population growth

All else given, an increase in the size of the population is likely to increase healthcare expenditure. The standard assumption is that there is a one-on-one pass through from population growth to healthcare expenditure or, in other words, an elasticity of one. That is, the flow of extra people into the population receives the same amount of healthcare services per person as the stock of people already there.

Age and gender shifts

Changes in the age and gender profile of the economy's population are likely to affect healthcare expenditure. An increase in those demographic groups that receive relatively large amounts of healthcare is likely to increase healthcare expenditure.

The impact of population ageing on healthcare expenditure is quantified using the same methodology as PC (2005) (box 1.1). An alternative method, taking into account changes in the crude death rate, was also explored by the Commission (box 1.1). Estimates of the residual and the ageing effect using this alternative method are presented in table 1.3.

Box 1.1 **The past impact of demographic change on health expenditure**

Consistent with a range of other studies (such as COA 2002), PC (2005) calculated that demographic change was likely to have increased health expenditure by between 0.4 per cent and 0.6 per cent per annum over the last 30 years. This was based on the assumption that, for all components of health expenditure, the age effect could be calculated from the age-profile of expenditure and the changes in population structure.

However, the projections of *future* hospital expenditure in PC (2005) incorporated health costs in the last year of life, as well as ongoing age-related health expenditure. The Commission has now extended this approach to recalculate the *past* impact of ageing.

As discussed in a forthcoming PC technical paper, incorporating hospital costs associated with the end of life results in a significantly lower past demographic impact on health expenditure than using the traditional approach — 0.2 to 0.25 per cent a year between 1970 and 2003, or about half the previous result. The reason for the lower result is that, although the population has been ageing for some decades, the crude death rate has been falling (from around 9 to 6.6 deaths per 1000 between 1970 and 2003). As such, the component of health expenditure related to the cost of dying has been declining, partially offsetting rises from the changing age structure.

However, as discussed in PC (2005), it should be noted that the crude death rate has reached its projected minimum and will begin to rise as the 'baby boomers' reach the end of their lives. By 2045, it is projected to be between 10 and 11 deaths per 1000 people. This is one reason why demographic change is expected to have a more significant impact on hospital expenditure in the future than it has had in the past.

Excess health inflation

Inflation in the healthcare sector is frequently greater than that in the general economy, although it depends on the time period chosen. The difference between the growth in prices of healthcare compared to other goods and services in the economy is expressed as 'excess' health inflation. There are two potential mechanisms through which excess health inflation can affect healthcare expenditure: inflation and substitution.

The faster growth in prices in the healthcare sector will tend to increase healthcare expenditure relative to spending in the rest of the economy. Failure to allow for this (that is, only controlling for general inflation) might overestimate the growth in real expenditure and hence the residual. In contrast, excess health inflation will make people switch from healthcare to other products as the latter become relatively cheaper. The effect of this substitution will be to reduce healthcare expenditure. However, this effect is likely to be small because of people's limited ability to substitute other goods and services in the economy for healthcare.

Some studies (Mushkin and Landefeld 1979; Wanless 2001) ignore this potential substitution effect and assume a one-on-one pass through from excess health inflation to nominal health expenditure (an elasticity of one). Other studies (Cutler 1995; Fuchs 1972) assume an elasticity of one for the inflationary effect and -0.2 for the substitution effect, giving an overall elasticity of 0.8. Other studies ignore the impact of excess health inflation altogether (table 3.1).

Because of the strong relationship between changes in the health price index and changes in technology, controlling for the excess health inflation variable is problematic. The only comprehensive health price index available is an implicit price deflator, calculated based on a fixed basket of medical wages and intermediate input costs (AIHW 2004). Price indices based on fixed bundles of inputs do not control for improvements in quality arising from technological change (Berndt et al. 2000; Wanless 2001). At an extreme, some US studies have suggested that quality improvements account for all of the excess health inflation (Cutler et al. 1996).

To the extent that the effects of advances in technology are captured in the health price index this may constitute 'double counting'. The Commission presents estimates of the residual controlling for excess health inflation for the purpose of comparability with other studies. However, these estimates should be used with caution.

Income

As discussed in chapter 3, although it is generally agreed that there is a positive relationship between income and healthcare expenditure, there is unresolved controversy about whether healthcare is a 'normal' or a 'superior' good. In particular, macro studies (using time-series or international cross-sectional data) tend to produce higher income elasticity estimates than micro level studies (chapter 3).

Getzen (2000) has suggested that these different estimates from micro and macro data can be reconciled by the possibility that insurance causes a separation between the budget constraints of the group and individuals. If the group is large enough, the availability of insurance makes the budget constraint non-binding at the individual level but binding at the group level. Consequently, the demand for healthcare is more likely to be more responsive to changes in income of the group than it is to that of the individual.

Getzen (2000) suggests that it is important when looking at income elasticity to specify the unit of analysis (micro or macro) and then use an estimate from a

matching source. This would imply that a higher income elasticity is appropriate for the Commission's aggregate analysis. However, these macro estimates might overestimate the impact of income because of endogenous technological change (Newhouse 1992; Ringel et al. 2002). This issue is explored further in chapter 3, particularly box 3.2.

For these reasons, the Commission has estimated an upper and lower bound for the residual based on a plausible range of elasticities.

The Commission's lower bound residual estimate is based on an elasticity of one, consistent with a number of other aggregate studies (Fuchs 1972; Mushkin and Landefeld 1979; Newhouse 1992; PC 2005). This implies that the growth in aggregate healthcare expenditure increases by as much as aggregate income growth (Di Matteo 2005).

The Commission's upper bound residual estimate is based on the assumption of an elasticity of 0.2. This elasticity was estimated in the RAND health insurance experiment, which randomly assigned individuals to different health insurance plans and recorded their healthcare expenditure (Manning et al. 1987). This estimate has been adopted in a number of other residual studies (Cutler 1995; Oxley and MacFarlan 1994).

An estimate based on an elasticity of 0.6 is presented as the Commission's preferred estimate. The elasticity is the midpoint between the macro level estimates, not controlling for technological change, and the micro level estimates based on individual decision making.

Private health insurance coverage

An increase in the proportion of the population covered by private insurance (or the fraction of each individual's medical expenses covered) is likely to increase health expenditure. On the demand side, this occurs because the insured seek to use medical services even when the benefits are small because they bear only a small fraction of the total cost. Similarly, doctors may be more willing to prescribe expensive treatments for patients who do not have to bear the full cost (Gerdtham and Jönsson 2000).

In the context of universal free access to public hospitals in the Australian healthcare system, an increase in insurance coverage may manifest in an increase in the use of:

- ancillary health services, such as optical and dental, which are covered by private health insurance but are not widely publicly funded;

- elective surgery (due to shorter waiting times for private patients); and
- more expensive technologies for a given intervention — the use of drug eluting stents rather than bare metal stents, for example.

The previous legislative arrangements, requiring health insurers to fund prostheses with 'no gap', provided further incentives for doctors to provide their patients with the best technology available, regardless of price (chapter 2).

On the supply side, it can be more costly to treat a private patient than a public one because of the higher costs of medical devices in the private system. BUPA Australia (sub. 28) provides details of the price mark-up for devices in the private system, showing that prices can be more than 50 per cent higher in the private sector (chapter 4).

There is very little information available about the elasticity of healthcare expenditure with respect to private health insurance. Mushkin and Landefeld (1979) use regression analysis to estimate the elasticity of health spending with respect to third-party payments, assuming different values for the income elasticity. Under the assumption of an income elasticity of one, they estimate the elasticity with respect to third-party payments to be 0.54.

The Commission's modelling of the impact of technology using the direct approach (section 1.2) produces an elasticity estimate for private health insurance coverage of 0.37. In the absence of any other Australian estimates, this elasticity is used by the Commission in calculating the residual.

Results

The estimates of the technology residual are presented in table 1.2. Due to concerns about measurement of the excess health inflation variables and the elasticity for the private health insurance variable, estimates are also presented excluding these variables from the analysis, both individually and jointly.

Table 1.1 **The technology residual, 1992-93 to 2002-03[a]**

	Controlling for all determinants[b]	*Controlling for the impact of excess health inflation*[c]	*Controlling for the impact of health insurance*[d]	***Preferred estimates***[e]
Percentage points of annual growth attributable to the residual [f]				
Lower bound[g]	-0.2	0.1	0.6	**0.9**
Mid-range[h]	0.8	1.2	1.6	**1.9**
Upper bound[i]	2.0	2.3	2.6	**2.9**
Per cent of average annual growth attributable to the residual				
Lower bound	—	2.5	10.5	**16.9**
Mid-range	15.6	22.0	30.0	**36.4**
Upper bound	37.9	44.3	49.4	**55.8**

a The residual is calculated from real health expenditure data (table 1.9). **b** Controlling for the effects of population growth, age and gender, GDP, excess health inflation and private health insurance coverage. **c** The impact of excess health inflation is controlled for, but not private health insurance coverage. **d** The impact of private health insurance coverage is controlled for, but not excess health inflation. **e** The Commission's preferred estimates were obtained without controlling for the impact of excess health inflation or private health insurance coverage. These are the estimates presented in chapter 3. **f** Represents the percentage point contribution of the residual to the average annual growth in real health expenditure of 5.3 per cent. **g** The lower bound is based on the assumption of an income elasticity for healthcare of one. **h** The mid-range estimate is based on the assumption of an income elasticity for healthcare of 0.6. **i** The upper bound is based on the assumption of an income elasticity for healthcare of 0.2.

Source: Commission estimates.

The Commission's preferred estimates are discussed in more detail in chapter 3.

Table 1.2 presents the contribution of each determinant of healthcare expenditure to the average annual growth in real health expenditure, based on the Commission's preferred estimates (excluding excess healthcare inflation and private health insurance coverage). Estimates using the alternative method for controlling for the impact of ageing are presented in table 1.3.

Table 1.2 **Determinants of real health expenditure growth, 1992-93 to 2002-03**

Standard age adjustment

	Lower bound	*Mid-range*	*Upper bound*
Percentage points of annual growth attributable to each determinant[a]			
Population growth	1.2	1.2	1.2
Age adjustment	0.6	0.6	0.6
Income growth	2.6	1.5	0.5
Residual	0.9	1.9	2.9
Total[b]	**5.3**	**5.3**	**5.3**
Per cent of average annual growth attributable to each determinant			
Population growth	22	22	22
Age adjustment	12	12	12
Income growth	49	29	10
Residual	17	36	56
Total[b]	**100**	**100**	**100**

[a] Represents the percentage point contribution of each determinant to the annual growth in real health expenditure of 5.3 per cent. [b] Totals may not sum due to rounding.

Source: Commission estimates.

Table 1.3 **Determinants of real health expenditure growth, 1992-93 to 2002-03**

Age adjustment controlling for crude death rates

	Lower bound	*Mid-range*	*Upper bound*
Percentage points of annual growth attributable to each determinant[a]			
Population growth	1.2	1.2	1.2
Age adjustment	0.3	0.3	0.3
Income growth	2.6	1.5	0.5
Residual	1.3	2.3	3.3
Total[b]	**5.3**	**5.3**	**5.3**
Per cent of average annual growth attributable to each determinant			
Population growth	22	22	22
Age adjustment	5	5	5
Income growth	49	29	10
Residual	24	43	63
Total[b]	**100**	**100**	**100**

[a] Represents the percentage point contribution of each determinant to the annual growth in real health expenditure of 5.3 per cent. [b] Totals may not sum due to rounding.

Source: Commission estimates.

The mid-range estimates from table 1.2 are discussed in chapter 3.

Table 1.4 presents the contribution of each determinant of healthcare expenditure across different periods, based on an income elasticity estimate of 0.6. For each year, the average growth rates are calculated between that year and 2002-03. For

example, the average annual growth rate reported in the table for 1995-96 is the average annual growth rate in real health expenditure over the period 1995-96 to 2002-03.

Table 1.4 **Determinants of healthcare expenditure over time[a,b]**

	1992-93	*1993-94*	*1994-95*	*1995-96*	*1996-97*	*1997-98*	*1998-99*	*1999-00*	*2000-01*	*2001-02*
Percentage points of annual growth attributable to each determinant[c]										
Population growth	1.2	1.2	1.2	1.2	1.2	1.2	1.2	1.2	1.2	1.2
Age	0.6	0.6	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.7
Income growth	1.5	1.5	1.5	1.4	1.4	1.3	1.0	0.8	1.1	0.7
Residual	1.9	2.0	2.1	2.2	2.2	2.4	2.3	2.5	2.1	2.6
Total[d]	**5.3**	**5.3**	**5.4**	**5.5**	**5.4**	**5.5**	**5.2**	**5.3**	**5.0**	**5.2**
Per cent of average annual growth attributable to each determinant										
Population growth	22	22	22	21	22	22	24	23	24	23
Age	12	12	12	12	12	12	13	13	14	13
Income growth	29	28	27	26	26	23	19	16	21	13
Residual	36	37	39	40	40	43	44	48	42	51
Total[d]	**100**	**100**	**100**	**100**	**100**	**100**	**100**	**100**	**100**	**100**

a The growth rates presented in the table are average annual growth rates calculated between the year shown and 2002-03. **b** The growth rate presented for 2001-02, is based on a single year of growth (2001-02 to 2002-03), and hence is more subject to statistical fluctuations than the growth rates calculated by averaging over longer periods. **c** Represents the percentage point contribution of each determinant to the annual growth in real health expenditure of 5.3 per cent. **d** Totals may not sum due to rounding.

Source: Commission estimates.

Table 1.4 demonstrates that the average annual growth in health expenditure has been reasonably stable across the ten-year period, varying between 5 and 5.5 per cent.

The percentage point contribution of ageing and population growth to the growth in real healthcare expenditure has also remained reasonably stable over the period. For income growth, on the other hand, the percentage point contribution was lower in the late 1990s and early 2000s, compared to the early 1990s. With the contribution of income falling, the proportion of the annual growth rate attributable to the technology residual increased steadily over time, from 36 per cent across the ten-year period to 51 per cent over the period 2001-02 to 2002-03.

Decomposing the residual further to control for the impact of private health insurance coverage, it becomes evident that some of the additional growth in health expenditure in later years may be explained by higher annual growth in the proportion of the population with private health insurance. Indeed, after controlling for private health insurance coverage, the estimated residual is reasonably stable

over the 1990s, only increasing between 2000-01 and 2002-03 (this is sensitive to the choice of elasticities however).

1.2 The direct approach

The direct approach for estimating the impact of technological advances on healthcare expenditure is based on specifying a proxy for technological change. For reasons discussed in chapter 3, US health research and development (R&D) spending is the proxy used. The Commission's modelling largely follows the approach of Okunade and Murthy (2002). This section summarises the Commission's methodology and results. More detailed results are available upon request (where indicated).

Determinants of healthcare expenditure

For the direct approach, real per capita health expenditure is used as the starting health expenditure series. It is preferable to adjust for population growth at the outset when using an econometric approach, to avoid losing degrees of freedom in estimating what is essentially an accepted relationship between population growth and real health expenditure. (A one-to-one pass through from population growth to healthcare expenditure has been assumed in all previous studies.) However, the final results are presented in terms of real health expenditure (not adjusted for population growth) to allow for comparison with those from the residual approach.

For this approach, the Commission considered all the potential drivers of healthcare expenditure taken into account in the residual approach (other than population growth). The rationale for considering these variables, and the direction and magnitude of their expected relationship with healthcare expenditure, are outlined in section 1.1. The key differences in the variables used in the direct approach are:

- population growth is not controlled for because the starting health expenditure series is real *per capita* health expenditure;
- income is measured by real per capita GDP;
- ageing is measured by the proportion of the population older than 65, whereas for the residual approach it is controlled for using more complex adjustments based on the age and gender profile (section 1.1); and
- the impact of technology is quantified directly through the R&D proxy.

The model is estimated over the period 1971-72 to 2002-03. All series are specified in natural logarithms (logs) so that the elasticities are estimated by the final model. More information on the data series used are presented in table 1.9 at the end of this paper.

Econometric methodology

The direct approach estimates the impact of technology on expenditure by quantifying the relationship between health expenditure and its hypothesised determinants (including the technology proxy) *over time*. As with any time-series econometric analysis, it is first necessary to establish the properties of each series (including testing for stationarity and structural breaks) before choosing the appropriate modelling technique.

Unit root testing

Unit root tests are used to determine whether a series is stationary. It is important to establish the stationarity of a series prior to carrying out econometric analysis in order to avoid the possibility of a spurious regression (Granger and Newbold 1974).[1]

Some of the earlier papers using econometric techniques to estimate the relationship between health expenditure and its determinants obtained spurious results by failing to recognise the non-stationarity of their data (Hansen and King 1996) (chapter 3).

The Commission has used three alternative unit root tests to establish the order of integration of each series: [2]

- Augmented Dickey Fuller (ADF) Test (Dickey and Fuller 1979);
- Phillips-Perron (PP) Test (Phillips and Perron 1988); and
- Dickey-Fuller Test with generalised least squares detrending (DF-GLS) (Elliott et al. 1996).

1 Ordinary least squares regression of two *independent* but non-stationary series will often indicate a significant relationship between these series (certainly more frequently than indicated by the nominal significance level).

2 The order of integration of a series refers to the number of times it must be differenced before it is rendered stationary. So, for example a series integrated of order 1 (I(1)), is non-stationary in levels but stationary in first differences.

Structural break testing

The unit root tests are based on the assumption that there is no structural change in the series. When there are structural breaks in the series, the unit root tests are biased towards not finding a unit root (Enders 1995). For example, Jewell et al. (2003) show that once structural breaks are accounted for, health expenditure and GDP data previously demonstrated by Gerdtham and Löthgren (2000) to be non-stationary, are indeed stationary.

The Commission has used the Zivot-Andrews Test (Zivot and Andrews 1992) to test for a unit root in the presence of a deterministic structural break in each series considered. The Zivot-Andrews Test has the advantage of not requiring the *a priori* specification of the timing of the structural break (Olekalns and Cashin 2000).

Shifts in the mean, trend, and both the mean and the trend were all tested using the Zivot-Andrews framework.

Cointegration testing

As explained above, spurious results can arise from regression analysis in the presence of non-stationary variables. However, if at least one cointegrating relationship can be established between the variables then regression analysis involving these variables is potentially meaningful. Essentially cointegrating relationships are stable long-term relationships between variables (box 1.2).

Box 1.2 Cointegrating relationships

A cointegrating relationship exists between I(1) (non-stationary) variables, if there is some linear combination of these variables that is stationary. This essentially implies that there is a tendency for these variables to move together over time — deviations in their path do not persist in the long run.

Without knowledge of cointegration, the only valid (non-spurious) method for regressing I(1) variables is to difference the variables in the linear regression model. This limits the interpretability of the estimated coefficients. Cointegration makes regression between I(1) variables both statistically valid and interpretable.

Source: Wooldridge (2003).

The Commission has used the Johansen cointegration test to determine the number of cointegrating relationships between the variables (Johansen 1988).

The Johansen procedure is sensitive to the model specification (Maddala and Kim 1998). The choice of lag length and deterministic components is discussed in the results section below.

Vector error correction models

If a cointegrating relationship is found to exist between a set of series, then this relationship can be represented in the form of a dynamic vector error correction model (VECM) (Engle and Granger 1987). It is useful to represent the model in this form because both the short- and long-run dynamics of the system become evident.

Results

Unit root testing

The ADF, PP and the DF-GLS tests were run for each series in both levels and differences to establish the order of integration. The test statistics and the relevant critical values are presented in table 1.5.

Table 1.5 **Unit root test results[a,b]**

Variable	*ADF* [c]	*PP* [d]	*DF-GLS* [c]
Levels			
Health expenditure	0.76 (7)	-2.02 (0)	-2.85 (1)
GDP	-2.30 (1)	-1.79 (3)	-2.45 (1)
% population 65+	0.13 (3)	0.53 (4)	-1.32 (1)
% population with private health insurance	-1.95 (1)	-1.33 (3)	-2.35 (1)
US R&D	-0.93 (1)	-0.49 (2)	-1.47 (1)
First differences			
Δ Health expenditure	-5.04 (6)	-3.54 (1)	-3.59 (0)
Δ GDP	-3.72 (6)	-6.51 (14)	-4.59 (0)
Δ% population 65+	-4.52 (2)	-3.55 (7)	-3.32 (2)
Δ % population with private health insurance	-4.49 (1)	-4.87 (13)	-4.47 (1)
Δ US R&D	-3.51 (0)	-3.52 (1)	-3.63 (0)
5% critical value[e]	**-3.59**	**-3.59**	**-3.19**

[a] For the period 1971-72 to 2002-03. [b] All variables are in natural logs. Lag lengths are in parentheses. All regressions include a constant and a linear time trend. [c] Lag lengths for ADF and DF-GLS chosen based on Akaike Information Criterion. [d] Truncation lags for PP chosen based on Newey-West criterion. [e] 5 per cent critical value for ADF and PP tests based on a sample size of 20, from MacKinnon (1996). 5 per cent critical value for the DF-GLS test is interpolated from the values simulated in Elliott et al. (1996).

Source: Commission estimates.

The unit root tests suggest that most of the series are I(1), that is, non-stationary in levels, but stationary in first differences. While the results from the ADF and/or PP

test are borderline towards not rejecting the null hypothesis of a unit root in first differences of the health expenditure, percentage of the population greater than 65, and US health R&D series, the DF-GLS tests suggest that all the series are I(1), at the 5 per cent level. Given the superior power of the DF-GLS test in small samples (Elliott et al. 1996), it is assumed that all the series are I(1). This is supported by visual inspection of the series.

The excess health inflation series was also tested over the time period available (1974-75 to 2002-03) and was found to be I(1) under all three tests.

Structural break testing

All possible break points are tested in each series between 1975-76 and 1998-99 (table 1.6).

Table 1.6 **Zivot-Andrews structural break test[a]**

Variable	*Shift in mean*	*Shift in trend*	*Shift in mean and trend*
Health expenditure	-3.74	-3.76	-3.81
GDP	-3.89	-2.35	-3.54
% population 65+	-1.62	-0.77	-1.55
% population with private health insurance	-1.93	-4.12	-4.13
US R&D	-2.28	-2.71	-2.24
5% critical value[b]	**-4.80**	**-4.42**	**-5.08**

a All variables are in natural logs. All break points in the series are tested for between 1975-76 and 1998-99. (An integer range of 0.13–0.87 of the full sample.) **b** 5 per cent critical values are from Zivot and Andrews (1992).

Source: Commission estimates.

The Zivot Andrews tests suggest that all the series in table 1.6 are non stationary over the period considered. That is, there is no evidence of a deterministic structural change in the mean and/or trend around which the series are stationary. However, the excess health inflation series was also tested (over a shorter time period commensurate with the shorter series available) and was found to be stationary around a deterministic shift in the trend (in 1991), consistent with visual inspection of the series. The health price index is excluded from the cointegration analysis both because it is stationary and because of the limitations in the measurement of the series outlined in section 1.1.

Cointegration tests

General model

The Johansen cointegration test was performed on all the non-stationary variables (health expenditure, GDP, percentage of the population with private insurance, proportion of the population greater than 65 and US health R&D) to determine the number of cointegrating relationships between the variables.

Given that all five variables appear to have a constant and a trend, the model needs to allow for a linear deterministic trend in the data. The estimates reported are for a model with a linear trend in the data (in levels) but only intercepts in the cointegrating equations. Nonetheless, the model is shown to be robust to alternative specifications of the deterministic components. (Results from alternative specifications are available upon request.) It is estimated that there are two cointegrating equations between the five variables tested.[3]

Normalising one of the cointegrating equations on the log of health expenditure in the VECM, gives the first set of normalised cointegrating coefficients reported in table 1.7. As the model is specified in logs, the coefficients reported are the estimated elasticity of real health expenditure per capita with respect to each of the variables.

The magnitudes and signs of the coefficients in the model are in line with expectations. The coefficient on the ageing variable, however, is insignificant and the likelihood ratio exclusion tests support the exclusion of this variable. The finding of an insignificant ageing effect is consistent with a number of previous econometric analyses of health expenditure (Culyer 1990; Gerdtham et al. 1992; Jönsson and Eckerlund 2003). Possible explanations for why ageing is not found to be significant in this model are detailed in chapter 3.

The preferred model

Applying the Johansen cointegration test to the model excluding ageing indicates the presence of at least one cointegrating relationship between real health expenditure, GDP, percentage of population with private health insurance and US health R&D.[4] Again, the specification chosen has a linear trend in the data (in levels) but only intercepts in the cointegrating equations. Using lag exclusion tests,

3 Maximum eigenvalue and trace tests with a lag order of two were used.

4 The trace test indicates one cointegrating relationship while the maximum eigenvalue test indicates two.

a lag length of two was found to be optimal. The number of cointegrating equations and the normalised cointegration vector were found to be robust to alternative specifications of the deterministic elements, although not to the lag length. (Results are available upon request.)

Table 1.7 **VECM coefficients, 1971-72 to 2002-03[a]**

Normalised on health expenditure

	Health expenditure	*GDP*	*% population 65+*	*% population with private health insurance*	*US health R&D*
Model including all series					
Cointegrating coefficient[b, c]	1.00	1.28**	0.18	0.32**	0.34**
		(0.29)	(0.26)	(0.08)	(0.08)
Adjustment coefficient[c]	-0.42**	0.13**	-0.02	0.14	-0.03
	(0.08)	(0.06)	(0.01)	(0.34)	(0.13)
Best fitting model[d]					
Cointegrating coefficient[b, c]	1.00	1.74**		0.37**	0.25*
		(0.33)		(0.09)	(0.10)
Adjustment coefficient[c]	-0.41**	0.14*		0.09	-0.10
	(0.08)	(0.05)		(0.30)	(0.14)

[a] All variables are in natural logs. The vector is normalised on health expenditure (the coefficient of health expenditure is equal to one). [b] Cointegrating coefficients (except for health expenditure) are reported as they would appear on the right-hand side of the cointegrating equation. [c] Standard errors are reported in parentheses below the estimates. ** Denotes significance at the 5 per cent level. * Denotes significance at the 10 per cent level. [d] The best fitting model is the one that excludes the percentage of the population greater than 65.

Source: Commission estimates.

The normalised coefficients for the VECM model excluding the ageing variable are presented in table 1.7. Likelihood ratio exclusion tests confirm that the coefficients on health expenditure, GDP and the percentage of the population with private health insurance are all significantly different from zero at the 5 per cent level of significance. The coefficient on US health R&D is significant at the 10 per cent significance level (table 1.8).

The income elasticity of 1.7 is at the upper end of most empirical estimates. A likelihood ratio exclusion test on the coefficient rejects the null hypothesis that this coefficient is equal to one. That is, there is statistically significant evidence that healthcare is a superior good. This is consistent with the results of Okunade and Murthy (2002), who estimate an elasticity of 1.6 on GDP in their model which also includes US health expenditure and US health R&D. Other studies which have found income elasticities for health expenditure greater than one include Gerdtham (1992) and Newhouse (1977).

The estimated elasticities for private health insurance and US health R&D are both positive and significant, but are significantly less than one, as would be expected. The elasticity estimated for private health insurance coverage of 0.37, is close to that estimated by Mushkin and Landefeld (1979) (section 1.1). Similarly, the elasticity estimate for technological change of 0.25 is not significantly different from the Okunade and Murthy (2002) estimate of 0.32, using US health expenditure data.

Multiplying the growth in US health R&D over the last decade with the elasticity estimated for technological change (0.25), provides an estimate of the impact of technological change on health expenditure over the last ten years. This will only be accurate to the extent that the average relationship between technological change and health expenditure over the decade is the same as that over the 32-year estimation period.

The model implies that technological change has contributed 1.9 percentage points to the average annual growth in healthcare expenditure of 5.3 per cent over the last decade (1992-93 to 2002-03). This equates to 36 per cent of the average annual growth in real healthcare expenditure over the period. This estimate of the contribution of technological change to health expenditure falls within a wide range of statistically plausible values, from 0.4 percentage points at the lower bound to 3.4 percentage points at the upper bound (based on the 95 per cent confidence interval for the coefficient on the R&D variable). These results are discussed in more detail in section 3.3.

Other results

The adjustment coefficients for the preferred model, presented in table 1.7, are significant for health expenditure and GDP, suggesting these variables are not weakly exogenous. Tests of each of the series for weak exogeneity supports this conclusion, with only the percentage of the population with private health insurance and US health R&D found to be weakly exogenous at the 5 per cent level of significance. The test statistics and relevant critical values are presented in table 1.8.

Table 1.8 **Exclusion and weak exogeneity tests[a]**

Tests	*Health expenditure*	*GDP*	*Private insurance*	*US health R&D*	***5% Critical value***
Exclusion	37.35	12.05	13.82	3.63	**3.84**
Weak exogeneity	25.20	8.95	0.14	0.69	**3.84**

a All variables are in natural logs.

Source: Commission estimates.

In the context of the VECM, the concept of weak exogeneity pertains to the short run forecastability in the system. So, to say that private health insurance and US health R&D are weakly exogenous implies that lags of these variables provide useful information in predicting health expenditure. The finding that technology is weakly exogenous (and health expenditure is not) is consistent with Okunade and Murthy (2002). They argue that this lends support for the Fuchs (2000) hypothesis:

> … the assumption of exogenous technology … may be reasonable for a good deal of economic analysis. (cited in Okunade and Murthy 2002, p. 154)

The adjustment coefficient on US health R&D is estimated to be -0.10, although not significant at the 5 per cent level. This suggests that the system responds relatively slowly to changes in technology. This slow speed is unsurprising given that R&D spending will be a leading indicator of technological innovation and that new innovations take time to diffuse.

Summing up

The Commission's modelling suggests that health expenditure, GDP, the percentage of the population covered by private health insurance, and health technology (as proxied by US health R&D) exhibit a stable long-term relationship. Applying the elasticity estimates in the model, technology is estimated to have contributed 36 per cent of the average annual growth in real healthcare expenditure over the decade. The level of US health R&D spending is estimated to be a predictor of future health expenditure, albeit with a long lead time. Thus, the model provides empirical support for the proposition that technological innovation is a significant driver of health expenditure over time.

Table 1.9 **Summary of data sources**

Residual and direct approaches

Data series	*Source*	*Description*
Real health expenditure	AIHW (2004); ABS (2004b)	Nominal health expenditure data from the AIHW (includes public and private spending on health goods and services, health-related services and health-related investment). The data are deflated using the ABS GDP Implicit Price Deflator (IPD) (base year = 2002-03).
Population and ageing	ABS (2004a)	The proportion of the population greater than 65 is calculated by summing the number of people in each of the over 65 age groups and dividing by the total population. The calculation of age and gender shifts for the residual approach are explained in section 1.1.
Real GDP	ABS (2004b)	The GDP data are deflated using the ABS GDP IPD (base year = 2002-03).
Excess health inflation	AIHW (2004); ABS (2004b)	The AIHW total health price index (HPI) is an IPD constructed by the AIHW. AIHW (2004) contains data on the HPI from 1992-93 to 2002-03. Previous data are sourced from AIHW (1995–2003), and are spliced together using successive rebasing. The excess health inflation series is calculated as the ratio of the HPI to the GDP IPD series. For the residual approach, the growth rate in excess health inflation is calculated by subtracting the growth rate in the HPI from the growth rate in the GDP IPD.
Private health insurance coverage	PHIAC (2005)	Private health insurance coverage data are based on the proportion of Australians covered by a private health insurance policy. The 1971-72 to 1975-76 data are estimates based on multiplying data on the number of insurance policy contributors by the average number of people generally covered by each policy (PHIAC, Canberra, pers. comm., 13 January 2005).
Real US health R&D	OECD (2005)	Health R&D data are sourced in real US dollars (base year 2000) from the OECD. The data have been rebased to 2003. The data are in calendar years so do not correspond directly with the other variables. Each calendar year is matched to the financial year that it overlaps with from 1 January to 30 June. For example, the 2001 R&D data are treated as 2000-01 data.

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2 Estimating individual technology expenditure impacts

This appendix outlines the methodology, data and results of the analysis undertaken by the Commission to estimate the health systemwide expenditure impact of some of the key advances in medical technology over the last decade. The technologies considered include three drugs (statins, selective serotonin reuptake inhibitors (SSRIs) and long-acting beta-2 agonists) and a procedure and device combination used for cataract surgery (the phacoemulsification (phaco) technique and foldable intraocular lenses (IOLs)).

The results presented are not intended to be definitive estimates of the expenditure impacts of these technologies. Rather, they are designed to indicate whether, overall, these technologies are likely to have been cost increasing or decreasing, once their impacts on the volume of treatment and the cost savings they generate elsewhere in the health system are taken into account.

2.1 Methodology

In order to assess the net expenditure impact of a technology, actual health expenditure needs to be compared with the hypothesised health expenditure if the technology were not introduced (the no technological change scenario). This requires assumptions about the costs and patterns of treatment under this no change scenario. In each case, it is assumed that if the technology were not introduced, the previous mix of treatments would have persisted. That is, it is assumed that no alternative technology would have become available.

For each of the technologies considered, their net expenditure impact is estimated at a point in time. This involves comparing their unit cost, volume of treatment and cost savings relative to the previous technology. By considering the expenditure impacts of the technology at a single point in time, some of the lifetime expenditure impacts — the impacts on technology on longevity and consequent health expenditure, for example — may not be fully captured in the analysis.

The net expenditure impact of a technology which substitutes for another can be summarised by the following equation:

$$Exp = [v_p(p_t - p_p) + p_t(v_t - v_p)] - [v_p(s_t - s_p) + s_t(v_t - v_p)]$$

$$= v_t(p_t - s_t) - v_p(p_p - s_p)$$

Where:

Exp is the net expenditure impact of the new technology in a given year;

v_t is the volume of patients treated using the new technology in the year;

v_p is the hypothesised volume of patients that would have been treated using the previous technology in the year;

p_t is the per patient cost of the new technology in the year;

p_p is the per patient cost of the previous technology in the year;

s_t is the per patient offsetting cost savings from the new technology in the year; and

s_p is the hypothesised per patient offsetting cost savings from the previous technology in the year.

Estimating the volume of patients that would have been treated using the previous technology is particularly difficult because it is necessary to control for changes in the prevalence of disease. In each case, it is assumed that the use of the previous technology would have grown in line with disease prevalence. Thus, any growth in the use of new technologies above changes in disease prevalence, can be attributed to the perceived benefits of these technologies.

Sections 2.2 to 2.5 provide details of the data, assumptions and estimated expenditure impacts for each of the four technologies. For each of the technologies the expenditure analysis is for the year 2000-01, the most recent year for which comprehensive data are available.

2.2 Statins

Statins are a class of cholesterol-lowering drugs used to treat cardiovascular diseases such as coronary heart disease (CHD) and stroke. Appendix F provides details about the use of, and expenditure on, statins, in addition to information about

the benefits and cost effectiveness of these drugs. Prior to the introduction of statins, the main cholesterol-lowering drugs used were bile acid sequestrants, nicotinic acid and fibrates (Medicines Australia sub. 30). These drugs are used as the comparator technology for the purposes of this case study.

Unit cost impacts

The average cost of a prescription of statins is approximately the same as the average cost of a prescription of one of the previous cholesterol-lowering drugs (Medicines Australia sub. 30). For example, both *atorvastatin* (the most commonly prescribed statin) and fibrates, cost the Australian Government approximately $40 for a month's supply (at the defined daily dose) through the Pharmaceutical Benefits Scheme (PBS) in 2000-01. Therefore, whether a patient uses statins or one of the other cholesterol-lowering drugs does not change the total expenditure on cholesterol-lowering drugs.

Volume impacts

It is likely that the introduction of statins led to an overall increase in the number of patients treated with cholesterol-lowering drugs. This is because statins are both more effective and more tolerable than the previous drugs (Tobert 2003). The volume of statin prescriptions has grown strongly, increasing by more than 400 per cent between 1993-94 and 2000-01 (HIC 2005). This growth does not appear to be generated by patients switching from the previous cholesterol-lowering drugs to statins. Indeed, the number of prescriptions for cholesterol-lowering drugs other than statins, increased slightly above the rate of population growth in the corresponding period.

It is necessary to identify the extent to which the growth in statin prescriptions is generated by the availability of statins *per se*, rather than increases in the prevalence of high cholesterol or a more general willingness to prescribe medications for this purpose. It is also useful to distinguish between the volume of statins prescribed for primary (preventing a CHD event before it occurs) and secondary prevention (preventing a recurrence of a CHD event) of cardiovascular disease.

Secondary prevention

Use of cholesterol-lowering drugs for secondary prevention depends on the prevalence of CHD in Australia. There was no significant change in the prevalence of CHD between 1989-90 and 1995 (AIHW 2002). However, between 1995 and

2001 there was a 19.4 per cent decline in the proportion of people reporting heart, stroke and vascular diseases as a long-term condition (AIHW 2004).[1]

The fall in the reported prevalence of CHD over the decade was driven by a fall in the incidence of the disease (which decreased by 25 per cent between 1993-94 and 2000-01) which more than offset the declining mortality rates (AIHW 2004).

Primary prevention

High blood cholesterol levels are a key qualifying criterion for patients without existing CHD to receive subsidised access to cholesterol-lowering drugs through the PBS (box F.1). The minimum total blood cholesterol levels for subsidised access is 5.5 mmol/L. It is estimated that in 1999-00, over 50 per cent of the Australian population had blood cholesterol levels exceeding 5.5 mmol/L (AIHW 2002). However, the prevalence of high blood cholesterol levels did not significantly change between 1980 and 1999-00 (AIHW 2002).

Other qualifying criteria for subsidised access (in conjunction with high blood cholesterol) include: diabetes; hypertension; and hypercholesterolaemia (box F.1). There was a significant decline in the proportion of the population with hypertension (high blood pressure) between 1980 and 1999-00. This decline was apparent among both those receiving treatment for high blood pressure and those not (AIHW 2002). There are no national data on the trends in prevalence of diabetes (AIHW 2002).

Overall volume impact

The evidence presented above suggests that epidemiological prevalence (in terms of indications for either primary or secondary prevention) has not been responsible for the growth in statin prescriptions over the decade. Further, there have been no significant advances in diagnostic technologies, or apparent shifts in community attitudes toward heart disease that could explain the strong growth in the use of cholesterol-lowering drugs. For these reasons, growth in the volume of statin prescriptions above the rate of population growth is attributed to the introduction of statins. That is, it is assumed that the almost 400 per cent per capita growth in statin prescriptions between 1993-94 and 2000-01 can be attributed to the broadening of the treatment population after the introduction of statins.

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1 The prevalence data reported are from the ABS National Health Surveys in 1989-90, 1995 and 2001. These data reflect self-reported information on the prevalence of CHD and may be influenced by changing perceptions of the disease over time (AIHW 2004).

Overall, this implies that of the almost 11.4 million scripts for statins in 2000-01, 9.1 million were filled by patients who would not have taken cholesterol-lowering drugs in the absence of statins. The other 2.3 million scripts were filled by patients that would otherwise have taken one of the previous cholesterol-lowering drugs. These assumptions imply expenditure on cholesterol-lowering pharmaceuticals was $465 million higher in 2000-01 than it would have been if statins were not introduced.

Offsetting cost savings

As outlined in appendix F, it is difficult to attribute causality between statin use and cost savings elsewhere in the health system. The Australian Long-Term Intervention with Pravastatin in Ischaemic Disease trial studied the impact of statin use (compared to a placebo) on cardiovascular events and mortality in a group of more than 9000 patients with a history of myocardial infarction or unstable angina (LIPID Study Group 1998). An economic evaluation of the trial data, showed that after an average follow-up of six years, hospital costs and other long-term medical costs were lower in the statin group by $1385 and $360 respectively (Medicines Australia sub. 30).

Assuming these costs were distributed evenly across years, this amounts to offsetting cost savings of approximately $290 per year. The Commission has adopted this figure as a measure of the per person cost savings from statin use. However, in the context of this analysis, this is clearly a generous assumption as the estimates from the trial are based on:

- statin use for secondary prevention — statins are also prescribed in Australia for primary prevention and for this group offsetting cost savings are likely to be lower because of the lower probability of a cardiovascular event (Thomson and Temple 2004); and
- the effects of statin use compared to a placebo — this will overstate the benefits for the estimated 190 000 people taking statins that would otherwise be taking one of the older types of cholesterol-lowering drugs.

The assumption of annual per patient cost savings of $290 translates to total offsetting cost savings of $275 million in 2000-01.

Net expenditure impacts

Based on the assumptions outlined above, the following estimates are derived for the net expenditure impact of statins in 2000-01:

- of the almost 950 000 people taking statins, almost 750 000 take them because of their improved effectiveness and tolerability relative to other cholesterol-lowering drugs. This equates to about $465 million of additional expenditure on cholesterol-lowering drugs;
- offsetting cost savings from statins of $275 million are achieved through a reduction in hospital and other medical costs; and
- the net expenditure impact of statins is estimated to be $190 million.

2.3 SSRIs

SSRIs are a class of antidepressants commonly used to treat depression (Hegarty et al. 2003). Appendix G provides details about the use of, and expenditure on, SSRIs, in addition to information about the benefits and cost effectiveness of these drugs. SSRIs first because available in Australia in 1990. Prior to this, tricyclic antidepressants (TCAs) were the most commonly used medication for depression. These drugs are used as the comparator technology for the purposes of this case study.

Unit cost impacts

SSRIs are significantly more expensive on average than TCAs. For example, in 2000-01, the average cost to the Australian Government for a prescription of SSRIs was $31, compared to $5.10 for a TCA prescription.

Volume impacts

Antidepressant use in Australia has grown strongly over the last decade (McManus et al. 2000). Some likely drivers of this growth are:

- *Increased prevalence of depression.* Between 1989-90 and 2001, there was a more than 1000 per cent increase in the number of people per 1000 of the population reporting a long-term depressive condition (ABS 1989-90, 1995, 2001).[2] It is likely that most of this growth is driven by an increase in the awareness of depression in the community, rather than increased epidemiological prevalence (Holmwood et al. 2001).

[2] The 2001 survey did not distinguish between depression and other affective disorders. The figure has been adjusted downward, assuming that depression accounts for 81 per cent of affective disorders, consistent with ABS (1997).

- *Greater willingness to provide treatment*. There is evidence that general practitioners' recognition of psychological disorders, including depression, and their willingness to prescribe medication to treat these disorders, have increased over time (Naismith et al. 2001). For example, patient encounters in general practice involving depression increased from 2.1 per cent in 1990-91 to 3.5 per cent in 1998-99. Further, the number of antidepressant prescriptions per 100 consultations also increased over this period (Bridges-Webb et al. 1992 cited in McManus et al. 2000).
- *The introduction of SSRIs*. SSRIs are widely regarded to be superior to the TCAs in terms of tolerability and toxicity. The lower toxicity has increased practitioners' willingness to prescribe medication for depressive disorders because they are less concerned about the risk of overdose (Mant et al. 2004). The reduction in side effects has also allowed an expansion of the treatment population. For example, because SSRIs have lower rates of sedation, doctors are more willing to prescribe them for working patients (Hall et al. 2003). Similarly, because these drugs have fewer serious interactions with other medications, the risks of prescribing them for older patients have been reduced (Mackay et al. 1999).

In estimating the impact of the introduction of SSRIs on growth in antidepressant use, growth in the use of SSRIs *above* the increase in the prevalence of depression is attributed to the characteristics of the SSRIs themselves. Of this increase, some is assumed to be the result of switching from TCAs to SSRIs (based on the decline in TCA prescriptions after the introduction of SSRIs), while the remainder is attributed to a broadening of the treatment population because of the improved tolerability and lower toxicity of the SSRIs.

These assumptions imply that, the additional 5.1 million SSRI prescriptions in 2000-01 compared with 1993-94, can be attributed to:

- an increase in the prevalence of depression — 3.9 millions scripts (approximately 327 000 patients);
- a broadening of the treatment population because of the introduction of the SSRIs — 960 000 scripts (approximately 156 000 patients); and
- switching from TCAs — 748 000 scripts (approximately 121 000 patients).

However, it is possible that the improved awareness of depression in the community and the increased willingness of doctors to diagnose the disease has to some degree been driven by the introduction of SSRIs. That is, it is possible that practitioners are more willing to diagnose the disease because they know there is a (relatively) tolerable treatment available. To the extent that this is the case, the impact of SSRIs on antidepressant use will be underestimated in this analysis. That said, even if we were to attribute *all* the growth in antidepressant prescriptions to the introduction of

SSRIs, these drugs would still be estimated to be expenditure reducing to the extent that they have diffused to patients with severe depression.

Offsetting cost savings

It is difficult to link use of SSRIs directly with cost savings elsewhere in the health system. However, a number of studies have attempted to quantify the cost savings associated with SSRIs compared with TCAs — for example, Sclar et al. (1994), and Skaer et al. (1995). Most of these studies have found that overall health system costs are the same or lower for patients using SSRIs. This reflects the fact that the higher cost of SSRIs is more than offset by the additional cost savings they generate.

The offsetting cost savings from SSRIs arise mainly from reduced physician visits and hospital admissions. Reduction in physician visits may occur because the dosing regimes for SSRIs are simpler than for TCAs, thus reducing the visits necessary for dose adjustment (Panzarino and Nash 2001). There is evidence that patients receiving SSRIs are more likely to maintain their treatment for the 6-month period recommended for patients with major depression (McManus et al. 2004).

SSRI use may be associated with lower hospital expenditure relative to TCAs because these drugs have lower incidence of treatment-related adverse events and may reduce hospital stays following overdose (appendix G). To the extent that the introduction of SSRIs has allowed additional patients to receive treatment with antidepressants — for example, working patients that could not take TCAs because of the sedative effects — this may have contributed to better management of the symptoms of depression, and thus lower rates of acute intervention.

In a US study comparing direct depression-related health expenditure amongst patients in a health maintenance organisation, Skaer et al. (1995) find that TCA use is associated with an average of two additional physician visits and one additional day in hospital each year, relative to SSRIs.[3] These estimates are based on patient expenditure for those diagnosed with major depression by a primary care physician or psychiatrist.

Assuming the same reductions in health services apply for patients with major depression in an Australian context, this would imply annual cost savings from SSRI use compared to TCA use of approximately $570 per patient, over $500 of which is due to reduced hospitalisations. As this is less than the average annual cost

[3] Skaer et al. (1995) also consider reductions in laboratory tests, psychiatric hospitalisations and visits to psychiatrists.

of SSRI medication, this implies that this medication may have been cost saving to the extent that it diffused to patients with major depressive disorders.

There is some evidence that reductions in hospitalisations have been generated by the introduction of SSRIs, with separations for affective disorders (of which depression is the most common), falling by almost 50 per cent between 1996-97 and 2000-01, despite strong increases in the reported rate of prevalence of depression over this period.

However, it is not necessarily the case in Australia that all users of SSRIs have major depression. Despite the fact that the PBS restrictions state that SSRIs are for treatment of major depressive disorders (rather than milder depression), national survey data suggest that up to 80 per cent of patients managed for depression in general practice have chronic mild depression (McManus et al. 2003). Given that these patients are less likely to attempt suicide by overdose on antidepressants, average hospital cost savings produced by SSRIs are likely to be lower for this group of patients. Thus, to the extent that SSRIs have diffused to patients with mild depression, the estimates of offsetting cost savings will be overestimated.

Net expenditure impacts

Based on the assumptions outlined above, the following estimates are derived for the net expenditure impact of SSRIs in 2000-01:

- of the almost 905 000 people taking SSRIs, 627 000 are taking them because of the expanded prevalence of depression, 121 000 have switched from TCAs, and 156 000 are additional patients receiving antidepressants because of the improved tolerability and lower toxicity of SSRIs;
- SSRIs have increased expenditure on antidepressants by $149 million, of which $30 million is due to the expanded treatment population and, the remainder due to the higher cost of the SSRIs;
- for patients with a major depressive disorder, offsetting cost savings from SSRIs are estimated to be $570 per patient, which is more than the average per patient cost of SSRIs over a year, implying that SSRIs have reduced expenditure for these patients; and
- the expenditure impact of SSRIs for the treatment of mild depression is uncertain.

2.4 Phaco technique for cataract surgery

Phaco is the technique used for the surgical removal of cataracts in Australia. Generally the phaco technique is combined with the implantation of a foldable IOL. Appendix M provides details about the use of, and expenditure on cataract surgery, in addition to information about the benefits and cost effectiveness of phaco and foldable IOLs. Prior to the adoption of these technologies in the early 1990s, cataract surgeries were performed using the extracapsular cataract extraction (ECCE) technique. This technique is used as the comparator technology for the purposes of this case study.

The net expenditure impact is estimated for surgery performed on those aged 65 and older because this is the age range for which the most relevant offsetting cost data are available. Procedures for patients aged over 65 represent over 85 per cent of all lens procedures in hospitals, and thus expenditure trends in this group are likely to be representative of overall expenditure trends.

Unit cost impacts

The phaco technique has decreased the cost of cataract surgery in Australia. Assuming that the cost of ECCE grew at the average rate of health inflation, then the average costs of inpatient care for cataract surgery would have been $4577 in 2000-01 compared to an average cost of $3792 using the phaco technique.[4] The major savings from the new technology are shorter operations and reduced length of hospital stay (Asimakis et al. 1996).

Volume impacts

Cataract surgeries in the over 65 age group increased by 73 per cent over the period 1993-94 to 2000-01, reaching over 111 000 in 2000-01 (AIHW 2005). As cataracts are strongly associated with ageing, the ageing of the population is likely to be a key factor behind this growth. Improvements offered by the phaco technique relative to ECCE have also contributed to the increase in the number of surgeries, by reducing the visual acuity threshold for surgical intervention (appendix M).

In order to analyse the impact of the introduction of the phaco technique on the volume of treatment, the impact of ageing and population growth are controlled for. It is assumed that the age distribution of cataract surgery would be the same in

4 Commission estimates based on data from Eye Research Australia (2004). Using DoHA (2005) average cost figures for lens procedures (weighted average cost of $3569 in 2000-01) does not materially change the analysis.

2000-01 if ECCE were still used to perform surgery. However, to the extent that the availability of the phaco technique has allowed treatment of older patients (and thus changed the age distribution of treatment) this may underestimate the net expenditure impact of this technique.

Under this assumption, growth in the number of older people in the population can explain almost 20 000 of the additional cataract surgeries performed in 2000-01 compared with 1993-94 (or about 42 per cent of the total increase). The remaining 27 000 additional surgeries are attributed to the introduction of the phaco technique and foldable IOLs.

Offsetting cost savings

It is difficult to link cataract surgery directly with cost savings elsewhere in the health system. However, a number of studies have shown that vision impairment is associated with a higher probability of falls and hip fractures (Ivers et al. 2003; Klein et al. 1998). A UK study (Scuffham et al. 2003), has attempted to quantify the costs (by age group) from falls attributable to visual impairment, including: emergency room attendances; hospital admissions; GP consultations and long-term institutional care. The results imply average additional annual healthcare spending attributable to visual impairment of $58 for those aged 65–74 and $353 for those aged 75 and older (all estimates converted to 2002 AUD).

These figures can be applied to estimate the offsetting cost savings from additional cataract surgeries in Australia (based on the age distribution of these surgeries) by making the following assumptions:

- vision impaired people in Australia have a similar probability and severity of falls as vision impaired people in the United Kingdom;
- treatment patterns and costs for falls are similar in Australia and in the United Kingdom; and
- people with cataract impaired vision have a similar probability and severity of falls as vision impaired people more generally.

Applying these estimates to the 27 000 additional surgeries attributed to the improvements in technology, produces an estimate of overall cost savings of $6.8 million. This assumes that there are no offsetting cost savings from using the phaco technique relative to ECCE. However, to the extent the phaco technique produces superior visual acuity, this will understate the total offsetting cost savings from the technology. That said, even assuming that the full cost savings applied to *all* patients undergoing surgery using the phaco technique (regardless of whether they would have otherwise undergone an ECCE), the resulting cost savings estimate

of $11.7 million is not enough to outweigh the large increase in expenditure this technology has produced.

Net expenditure impacts

Based on the assumptions outlined above, the following estimates are derived for the net expenditure impact of the phaco technique and IOLs in 2000-01:

- of the almost 47 000 additional people aged 65 or over undergoing cataract surgery in 2000-01 (compared to 1993-94), 20 000 can be attributed to growth in the aged population, while 27 000 can be attributed to the improved surgical technique;
- the lower unit cost of the phaco technique generates $15.5 million in savings in operating on patients who would otherwise have undergone the more expensive ECCE procedure. However, the total cost of cataract surgery is $87.5 million higher because of increases in the number of surgeries attributable to the new technique;
- the additional surgeries produce estimated cost savings of $6.8 million elsewhere in the health system (including nursing homes); and
- the net expenditure impact of the phaco technique and IOLs is estimated to be $80.7 million.

2.5 Long-acting beta-2 agonists

Over the last decade, there have been a number of advances in medication for the prevention and long-term control of the symptoms of asthma. This section analyses the expenditure impact of one of these advances — the use of long-acting beta-2 agonists, such as *salmeterol*, to manage the symptoms of asthma. The long-acting agonists are used by patients who experience asthma symptoms despite treatment with corticosteroids (preventer medications). In particular, because their effects last for about 12 hours, they are used for the treatment of nocturnal asthma symptoms and exercise-induced asthma (National Asthma Council of Australia 2002).

Long-acting agonists appear to have been adopted as a complement to the combination of medications used to manage asthma in patients with moderate to severe asthma: short-acting beta-2 agonists to relieve acute asthma symptoms, and corticosteroids.

The evidence suggests that the long-acting agonists have been used in conjunction with existing doses of oral or inhaled corticosteroids. Some studies have indicated that equivalent symptom control can be achieved with lower doses of

corticosteroids by combining treatment with the long-acting agonists in well-controlled patients (Wilding et al. 1997). However, with prevalence of the disease unchanged,[5] the slight increase in inhaled corticosteroid prescriptions (in terms of defined daily doses (DDDs) per 1000 per day) since 1996 suggests that the long-term agonists are primarily being prescribed to achieve improved asthma control in symptomatic patients without reducing the dose of corticosteroids (ACAM 2005). This is most likely a result of the PBS guidelines which restrict PBS subsidies for long-acting agonists to patients with 'frequent episodes of asthma who are receiving treatment with oral corticosteroids or maximal doses of inhaled corticosteroids' (HIC 2005).

Combination therapy, involving an inhaled formulation of long-acting beta-2 agonists and inhaled corticosteroid, became available through the PBS in 2000-01.

The long-acting beta-2 agonists are also considered a complement to existing reliever medications (short-acting agonists such as Ventolin) which are the first line treatment for the relief of acute symptoms (National Asthma Council of Australia 2002). Prescriptions for reliever medications (in terms of DDDs per 1000 per day) were stable between 1996 and 2001 (ACAM 2005).

Treatment with corticosteroids and reliever medications only, is the comparator technology for the purposes of this case study.

Direct expenditure impact

The direct expenditure impact of the long-acting agonists is the difference in expenditure on asthma medications in 2000-01 compared with what it would have been if these medications were not introduced. As these medications have been introduced as a complement to existing drug treatments they have increased overall asthma drug expenditure. It is assumed that expenditure on corticosteroids and short-acting agonists would be the same regardless of whether the long-acting agonists were introduced. The cost to the Australian Government of corticosteroids and short-acting agonists through the PBS was $51 million[6] and $63 million respectively in 2000-01 (HIC 2005).

5 Prevalence of asthma was largely unchanged between 1994-95 (when the long-acting agonists were first reimbursed through the PBS) and 2000-01, with 11.6 per cent of the population reporting asthma as a long-term condition in 2000-01 compared to 11.3 per cent in 1994-95 (ABS 1995, 2001).

6 This excludes the cost of corticosteroids as part of combination therapies with long-acting beta-2 agonists.

The cost to the Australian Government of long-acting agonists through the PBS was $29 million in 2000-01. This is a conservative estimate of the cost to the PBS of these drugs because it excludes the cost of these drugs in combination therapies which were listed for the first time in this year.

Offsetting cost savings

Long-acting beta-agonists, in combination with inhaled corticosteroids, help control asthma symptoms for patients with moderate to severe asthma. There is evidence that people with poorly controlled asthma — that is, those that take above the maximum recommended doses of reliever medications — are more likely to attend emergency departments or be admitted to hospital with exacerbations of asthma (Anis et al. 2001; Nestor et al. 1998).

There was a 39 per cent decrease in hospital separations for asthma- and bronchitis-related conditions between 1995-96 and 2000-01 (DoHA 2005). However, the expenditure impact of this reduction was partially offset by the increase in the average cost of treatment. Making the generous assumption that all of the reduction in hospital expenditure between 1995-96 and 2000-01 can be attributed to the introduction of the long-acting agonists, then the offsetting cost savings from this technology are approximately $18 million.

Net expenditure impacts

Based on the assumptions outlined above, the following estimates are derived for the net expenditure impact of long-acting beta-2 agonists in 2000-01:

- there were 973 000 prescriptions for the long-acting agonists in 2000-01, generating $29 million of Australian Government PBS spending. This estimate is conservative because it excludes the long-acting agonists component of the combination therapies and relates only to Australian Government expenditure;
- there was not any reduction in spending on corticosteroids or reliever medications as a result of the introduction of the long-acting agonists;
- the maximum offsetting cost savings in hospitals from reductions in emergency department attendances and hospitalisations is an estimated $18 million; and
- the lower bound of the net expenditure impact of the long-acting beta-2 agonists was $11 million in 2000-01.

2.6 Summing up

Of the four advances in technology analysed, three (statins, phaco and long-acting beta-2 agonists) were estimated to be expenditure increasing and one (SSRIs) was estimated to reduce expenditure for certain patient groups. Although some of the expenditure-increasing technologies were about the same or lower unit cost than the treatment they superseded (statins and phaco), the consequent broadening of the treatment population led to an increase in overall health expenditure.

The estimates reported are dependent on the assumptions made regarding the impact of the technology on unit costs, volumes of treatment and offsetting cost savings, and thus they provide a rough guide to the expenditure impacts of these four key advances in medical technology. To consider the *overall* impact of these technologies would require a detailed analysis of the benefits these technologies have generated, relative to their comparators.

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3 Future Expenditure Impacts of Medical Technology

Terms of reference (c) ask the Commission to 'as far as practicable, identify the likely impact of advances in medical technology on healthcare expenditure over the next five to ten years, and identify the areas of significant potential growth'.

Chapter 11 outlines potential advances in medical technology and summarises the Commission's work in estimating the expenditure impacts of some likely future technologies. This technical paper sets out in detail the assumptions and methodology used to make quantitative estimates of the expenditure impacts of some likely future technologies to treat cardiovascular disease, cancer, diabetes and neurological disease.

The estimates presented here should be considered experimental and should not be interpreted as a definitive forecast of the impacts of the selected future advances in medical technology. Rather, they are only illustrative of possible impacts of future advances. The analysis also highlights the inherent difficulties in estimating future expenditure impacts of medical technology. Limitations of the analysis include:

- lack of data;
- lack of direct comparability between disease prevalence, incidence and cost data; and
- the need to make general assumptions about unit costs, volumes, offsetting cost savings and health inflation.

A 'technology-specific' rather than aggregate or 'residual' approach has been used to estimate future expenditure impacts. According to Mohr et al. (2001) the key advantages of the technology-specific approach are that:

- it treats technologies at an identifiable, descriptive level, rather than as a residual; and
- analysis can be tailored to accommodate unique aspects of medical advances, such as their impact on quality of life.

In estimating net future expenditure impacts, it is assumed that the selected advances in medical technology will occur by 2015-16 (ten years into the future).

To assist in determining whether a new technology is expenditure increasing or decreasing, a comparison is made between estimates of:

- net expenditure in 2015-16 using a new technology for treatment of a condition within a disease category; and
- net expenditure in 2015-16 using a current technology for treatment of a condition within a disease category.

Estimates are derived for the following four advances in medical technology:

- insulin sensitisation drugs for prevention of type 2 diabetes mellitus (DM);
- implantable atrial defibrillators (IADs) for control of atrial fibrillation (AF) and stroke prevention;
- robotic-assisted surgery for prostate cancer; and
- a vaccine for treatment of established Alzheimer's Disease (AD).

These technologies have been selected from a US study by Goldman et al. (2004) aimed at predicting future advances in medical technology in the United States. The selected technologies encompass a broad range of medical interventions — pharmaceuticals, devices, surgical procedures and vaccines — across different disease groups. Other reasons for selecting these technologies include:

- the technology is currently in advanced clinical trials or is already widely used overseas and therefore has a reasonably high likelihood of being introduced widely in Australia over the next ten years; and
- the technology is considered at least a partial substitute for an existing technology, allowing for comparison of estimated net expenditure impacts of current and future technologies.

Throughout the analysis, projections of net expenditure impacts are derived by estimating volume and unit cost impacts, and any offsetting cost savings that are forecast to arise from the technology.

To convert expenditure estimates to 2015-16 dollars, a health inflation rate of 2.7 per cent per annum is assumed. This rate is based on the average health inflation rate between 1992-93 and 2002-03 and is derived from the total health price index (an implicit price deflator) calculated by the Australian Institute of Health and Welfare (AIHW 2004a).

Table 3.1 summarises the qualitative estimated future net expenditure impacts of selected advances in medical technologies for four major disease categories. To varying degrees, all four selected technologies are projected to be expenditure increasing, an outcome generally related to their high unit costs and potentially wide

application. This outcome is consistent with Mohr et al. (2001) and Goldman et al. (2004) who both found that new medical advances they examined would have an upward impact on healthcare expenditure.

Given these findings, the key question which arises is whether the benefits of a new technology outweigh the increased costs. All four selected technologies have the potential to deliver large benefits by reducing morbidity and mortality.

Table 3.1 **Estimated net expenditure impacts of selected advances in medical technology**

Technology	*Disease category*	*Per patient costs*			*Volume*	*Net expenditure impact*
		Unit cost	Substitute/ complement/ add on	Costs elsewhere		
Insulin sensitisation drugs for prevention of type 2 DM	Diabetes	↑	Partial substitute	↓	↑	↑
IADs for control of AF and stroke prevention	Cardiovascular disease	↑	Substitute/ add on	↓	↑	↑
Robotic-assisted surgery for prostate cancer	Cancer	↑	Substitute	↓	↑	↑
Vaccine for treatment of established Alzheimer's disease	Neurological disease	↑	Substitute	na	↑	↑

na Not available.

3.1 Insulin sensitisation drugs for the prevention of type 2 DM

Insulin sensitisation drugs for prevention of type 2 DM among the obese population have been identified by Goldman et al. (2004) as a likely future advance in medical technology. Key benefits from this advance could include a reduction in morbidity

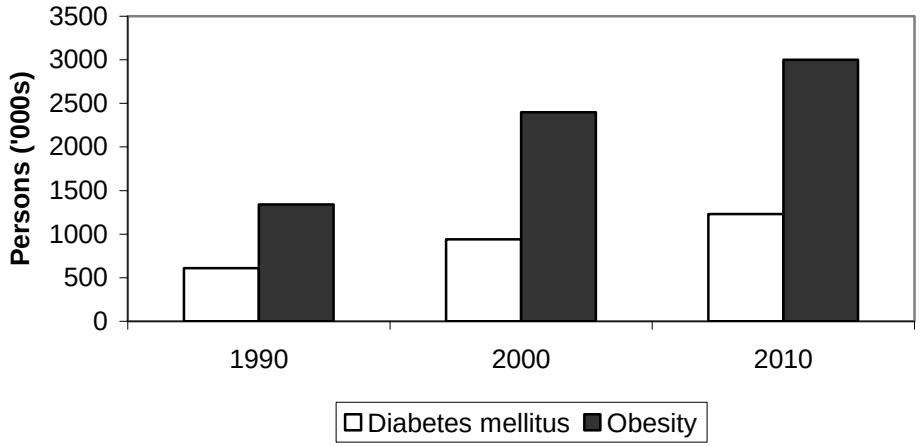
and mortality associated with type 2 DM. Offsetting cost savings could also be achieved through a reduction in healthcare expenditure for treatment of this condition.

Type 2 DM is a condition characterised by reduced levels of insulin and an inability of the body to respond to insulin properly (insulin resistance) (AIHW 2002). The condition is estimated to account for 85 to 90 per cent of all diabetes cases in Australia (ABS 1997).

A key risk factor for type 2 DM is excess weight (AIHW 2002). The Australian Diabetes, Obesity and Lifestyle Study found that people with type 2 DM were more likely to be overweight and obese than the general population (Dunstan et al. 2001). The prevalence of obesity in Australia has also increased significantly over time — between 1989-90 and 2001, the prevalence of obesity increased from 9 to 16 per cent in men and from 10 to 17 per cent in women. The study also showed that for every person in the sample who had known DM, there was another in whom it had not yet been diagnosed. This suggests that the prevalence of DM in Australia may be up to twice the number of currently diagnosed cases.

This trend is expected to continue — one estimate from Dixon and Waters (2003) predicted that approximately 17 per cent of Australians over 18 will be obese by 2020. The prevalence of type 2 DM is also expected to increase from approximately 5 per cent in 2001 to 6 per cent of the population in 2010 (figure 3.1).

Figure 3.1 **Estimates and projections of DM and obesity prevalence for Australia, 1990 to 2010[a]**



a Estimate of DM prevalence includes both type 1 and type 2 DM.

Data sources: Dixon and Waters (2003); Dunstan et al. (2001); Marks et al. (2001).

Current treatments for type 2 DM include improvements in activity levels and diet, and the use of oral blood glucose-lowering drugs such as the biguanides, the sulphonimades and the alpha glucosidase inhibitors. In 2001-02, approximately $70 million was spent in Australia on over four million prescriptions for oral blood glucose lowering drugs (Dixon 2005). In 2003, two new drugs from the thiazolidinedione (also known as the 'glitazone') class of drugs, *rosiglitazone* and *pioglitazone*, were listed on the Pharmaceutical Benefits Scheme (PBS)/Repatriation Pharmaceutical Benefits Scheme (RPBS) for the treatment of type 2 DM. These drugs are used in combination with other blood glucose-lowering drugs and:

> … reduce insulin resistance by enhancing the sensitivity of peripheral tissues and the liver to insulin … (Bryant et al. 2003, p. 647)

Recent research has examined the use of a thiazolidinedione as a preventative treatment for type 2 DM (Goldman et al. 2004). For example, the US Diabetes Prevention Program trial reported that *troglitazone* reduced the development of diabetes by 75 per cent compared to a placebo. However, the use of *troglitazone* had to be discontinued after reports that it had caused liver toxicity (Diabetes Prevention Program Research Group 2005).

At this point in time, there is limited data from human trials to suggest that prolonged treatment with a thiazolidinedione can prevent the development of type 2 DM in high-risk persons (Goldman et al. 2004). However, for the purposes of illustrating likely future expenditure impacts from treatment of type 2 DM, it has

been assumed that one drug from the thiazolidinedione class is widely prescribed in Australia by 2015-16 with the aim of preventing type 2 DM among the obese population. This assumption is broadly consistent with the assumption made by Goldman et al. (2004), who predict a median likelihood of this breakthrough occurring within 10 years in the United States of 50 per cent, and a median likelihood of this breakthrough occurring within 20 years of 65 per cent.

In addition, current clinical trials such as the Diabetes Reduction Approaches with ramipril and rosiglitazone Medications (DREAM) trial are exploring the ability of intensive therapy with *rosilglitazone* in preventing diabetes, raising the prospect of a significant discovery in this area (GlaxoSmithKline 2000).

Estimated net expenditure impacts of a type 2 DM prevention drug

This section describes the assumptions used to derive an estimate of future net expenditure impacts from the use of a thiazolidinedione for prevention of type 2 DM among the obese population.

Volume impacts

A type 2 DM prevention drug is likely to result in an overall increase in the number of persons taking diabetes drugs. This effect is caused by the drugs being widely available for prevention — as well as control — of type 2 DM. Consistent with Goldman et al. (2004), it is assumed that 30 per cent of the obese population will be targeted for treatment with a type 2 DM prevention drug in 2015-16. Using predictions of obesity prevalence from Dixon (2005), it is estimated that approximately 900 000 Australians would be eligible for this drug if it were to be listed on the PBS.

Unit cost impacts

The unit cost of a type 2 DM prevention drug is likely to be significantly higher than current oral blood glucose-lowering drugs. For example, the 2004-05 price of the thiazolidinediones listed on the PBS — *rosiglitazone* (approximately $62 for a minimum monthly supply) and *pioglitazone* (approximately $66 for a minimum monthly supply)[1] — significantly exceed the prescription cost of older and more commonly prescribed type 2 DM drug treatments such as *metformin*

[1] These prices refer to the dispensed price for maximum quantity, which includes the ingredients, the container and the dispensing fee of the pharmacist (DoHA 2005a).

(approximately $15 for a minimum monthly supply) (Diabetes Australia and the Royal Australian College of General Practitioners 2004; DoHA 2005a).

Following Goldman et al. (2004) the 2004-05 minimum monthly cost of a prescription for *rosiglitazone* is used as a basis for estimating unit costs of a type 2 DM prevention drug in 2015-16. This results in a unit cost estimate of approximately $80 per prescription per month or approximately $990 per year. However, this is likely to be an overestimate of the cost of a type 2 DM prevention drug, as it is possible that generic versions of a type 2 DM drug could become available around 2015-16 (Baker Heart Research Institute, Melbourne, pers. comm., 22 August 2005).

Offsetting cost savings

A type 2 DM prevention drug is likely to reduce the prevalence of DM amongst the obese population. Following Goldman et al. (2004) it is assumed that a type 2 DM prevention drug results in a 50 per cent reduction in type 2 DM among obese persons. Assuming also that in the absence of the drug, 10 per cent or 300 000 obese Australians would develop type 2 DM, a type 2 DM drug would see approximately 150 000 persons avoid the condition in 2015-16.

A reduction in DM prevalence is likely to deliver cost savings in both direct costs (for example, hospital admissions and nursing homes) and indirect costs (for example, lost income and lost years of life). Dixon (2005) estimated that in 2000-01, average health expenditure on diabetes in Australia was $1469 per self reported case of diabetes. Converting this figure to 2015-16 dollars and multiplying it by the estimated reduction in prevalence of type 2 DM (150 000 persons) results in a cost saving of approximately $220 million per annum. However, this is likely to be an underestimate of offsetting cost savings, as a type 2 DM prevention drug could also be expected to reduce the rate of DM complications (Baker Heart Research Institute, Melbourne, pers. comm., 22 August 2005).

Summarising potential net expenditure impacts of a type 2 DM prevention drug

Using the assumptions outlined above regarding volume, unit costs, offsetting cost savings and health inflation, the following estimates are derived regarding the net expenditure impacts of a type 2 DM drug in 2015-16:

- a type 2 DM prevention drug available to approximately 900 000 obese Australians would cost approximately $990 per person per year. This would result in an increase in health expenditure of approximately $890 million;

- offsetting cost savings of approximately $220 million would be achieved from a reduction in the prevalence of diabetes among the obese population; and
- the net expenditure impact of a type 2 DM prevention drug is therefore estimated at approximately $670 million in 2015-16 dollars or $30 per capita. This finding of a significant net increase in expenditure is consistent with Goldman et al. (2004), who estimate the net increase in total expenditure in the United States of a DM prevention drug at approximately US$4 billion or US$13 per capita in 2015-16. This differential in per capita cost could be partly caused by the higher unit cost of the glitazone drugs in Australia compared to the United States.

Comparison of estimated net expenditure impacts: type 2 DM prevention drug and current oral blood glucose-lowering drugs

To further illustrate estimated net expenditure impacts of a type 2 DM prevention drug, it is useful to compare with the case where the drug is not widely prescribed and current technology continues to be used to manage type 2 DM.

In this case, the current technology is oral blood glucose lowering drugs for obese persons who develop type 2 DM. Assuming that 10 per cent of obese Australians will suffer from type 2 DM and that the average cost of a prescription for these drugs grows in line with projected health inflation, the gross expenditure impact of continuing with current treatment is estimated at approximately $80 million per annum in 2015-16 dollars.

This impact is likely to be partly offset by a reduction in the cost of complications associated with type 2 DM. Clarke et al. (2005) estimated that treatment of overweight diabetic patients with *metformin* in the United Kingdom reduced the cost of DM complications by approximately £2800 (A$5700) over a ten-year period. Assuming these cost savings were distributed evenly over ten years, this amounts to a cost saving of approximately £280 (A$570) per year. The assumption of annual per patient cost savings of $570 translates to total offsetting cost savings of approximately $230 million in 2015-16. Accordingly, continuing with existing treatment could be expected to deliver overall cost savings of approximately $150 million in 2015-16 dollars.[2]

The analysis presented above suggests that a type 2 DM prevention drug would be likely to increase health expenditure in Australia significantly due to:

[2] Clarke et al. (2005) also found that *metformin* therapy for overweight diabetic patients was cost saving compared with conventional glucose control. This was because the increased cost of *metformin* therapy was less than the reduction in the cost of complications.

- a significant expansion in the number of persons eligible for oral blood glucose-lowering drugs; and
- the higher unit cost of this drug compared to other blood glucose-lowering drugs.

The analysis assumes that a type 2 DM prevention drug would be considered a cost-effective intervention compared with other treatments such as lifestyle modification programs and surgery for the severely obese.

3.2 IADs for control of AF and stroke prevention

IADs are a promising new treatment for AF, a heart condition where the pathway of the electrical stimulation to the atria is abnormal. The condition causes the muscle fibres in the atria to twitch, rather than beat normally, and the ventricles to beat irregularly, resulting in an irregular pulse (Victorian Government 2005). AF is associated with increased hospitalisations, higher mortality, heart failure, acute myocardial infarction and quality of life impairment (Santini and Ricci 2003). It is also associated with an increased risk of stroke (Hankey 2001).

In Australia, AF is estimated to affect around 5 per cent of persons over the age of 65 years (Hankey 2001). In 2002-03, AF and flutter was the principal diagnosis for over 36 000 Australian public and private hospital separations (AIHW 2005b).

Currently, the primary therapies used for treatment of AF are antiarrhythmic drugs and external defibrillation in hospital. In addition, anticoagulant drugs such as *warfarin* are prescribed to sufferers of AF with the aim of preventing stroke. However, antiarrhythmic drugs have been known to fail and are subject to safety concerns (Swygman et al. 2002). Furthermore, repeat external defibrillation treatment is expensive for hospital systems, inconvenient and unreliable for patients (Swygman et al. 2002). As such, IADs are currently being trialled as alternative treatment for AF.

IADs are not a substitute for implantable cardioverter defibrillators (ICDs). ICDs are used to treat ventricular arrhythmias, whereas IADs are used to treat atrial arrhythmias.[3] However, clinical trials have occurred in recent years with both stand alone IADs and with IADs combined with ICDs in dual chamber ventricular systems (Daoud 2002; Santini and Ricci 2003).[4] To date, these trials have

3 Unlike ventricular fibrillation, AF is not usually associated with sudden death.

4 Dual chamber ventricular systems assist to overcome the risk of an IAD triggering ventricular arrhythmias (Goldman et al. 2004).

demonstrated the efficacy of both these devices in preventing, detecting and terminating AF (Swygman et al. 2002). For example, the results of clinical trials conducted by Ricci et al. (2002) showed that the dual defibrillator detected nearly 100 per cent of atrial arrhythmias and had a favourable impact on hospitalisations and quality of life (Santini and Ricci 2003).

However, there remain a number of safety and tolerability concerns over IADs. For example, patients are:

> … usually conscious when AF occurs and thus may experience significant pain and discomfort with each shock. (Goldman et al. 2004, p. 158)

Further research aimed at minimising pain associated with IADs is expected in the future. For the purposes of illustrating future expenditure impacts of treatment of cardiovascular disease, it has been assumed that IADs are widely available to Australian sufferers of AF by 2015-16 with the aim of managing AF and reducing the incidence of stroke. This assumption is broadly consistent with Goldman et al. (2004), who predict a median likelihood of this breakthrough occurring within 10 years in the United States of 50 per cent, and a median likelihood of this breakthrough occurring within 20 years in the United States of 50 per cent.

Estimated net expenditure impacts of IADs

This section describes the assumptions used to derive an estimate of future net expenditure impacts from the widespread introduction of IADs for the treatment of AF.

Volume impacts

IADs are considered a partial substitute for anticoagulant drugs used to reduce the risk of stroke in sufferers of AF (Goldman et al. 2004). On the other hand, IADs are unlikely to substitute for antiarrhythmic drugs. Rather, they are likely to be used in combination with these drugs in a 'hybrid therapeutic approach' (Santini and Ricci 2003, p. 232), although it has not yet been established that this combination can be effective (Baker Heart Research Institute, Melbourne, pers. comm., 22 August 2005).

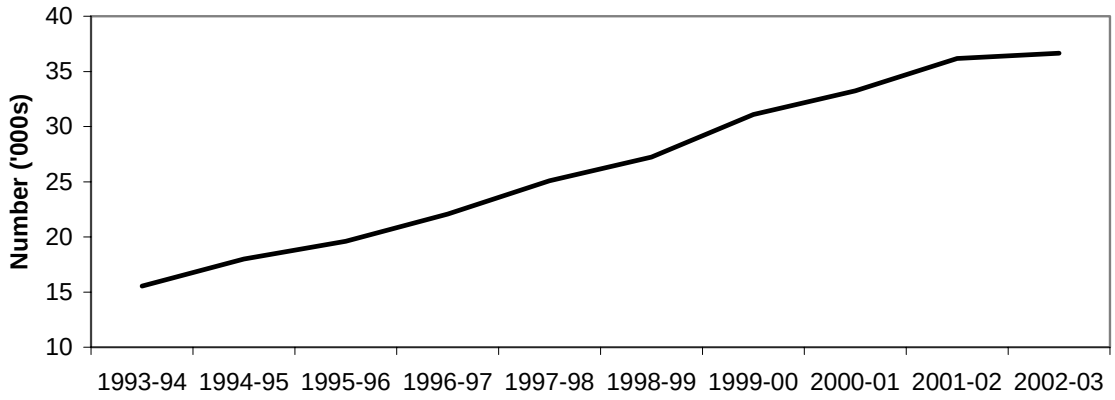
Potentially all patients with a diagnosis of atrial fibrillation or flutter could be eligible for access to an IAD in 2015-16 (Goldman et al. 2004). However, patients with chronic AF show enlarged atria and other features which may mean that they will never respond to defibrillation. Furthermore, advances in competing technologies such as surgical ablation procedures and antiarrhythmic drugs may

limit IAD utilisation (Baker Heart Research Institute, Melbourne, pers. comm., 22 August 2005).

In this analysis, incidence data for AF is used to estimate volume impacts. Incidence data simplifies the analysis, as it avoids the need to estimate a diffusion rate for IADs. A diffusion rate would be required if prevalence data were used, given that there would be a group previously diagnosed with AF prior to 2015-16 who would also potentially be eligible for IADs.

Assuming that the incidence of AF grows at 10 per cent per annum between 2002-03 and 2015-16 (a rate equivalent to the average growth rate for AF diagnoses between 1993-94 and 2002-03), approximately 126 000 persons would be potentially eligible for an IAD in 2015-16 (figure 3.2).

Figure 3.2 **AF and flutter diagnoses, 1993-94 to 2002-03**



Data source: AIHW (2005b).

Unit cost impacts

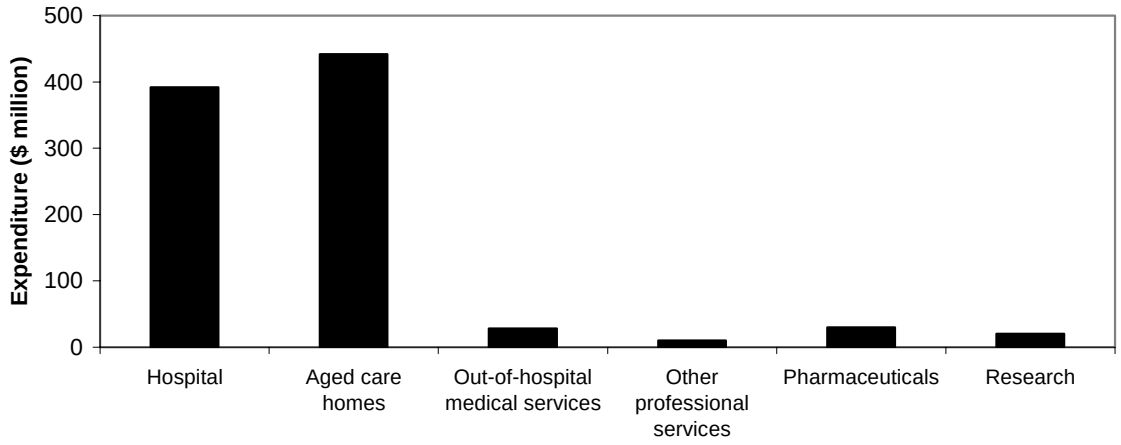
Consistent with Goldman et al. (2004) it is assumed that an IAD device will have a unit cost similar to the cost of an ICD device. In 2002-03, the weighted average cost of an ICD across the Australian public and private hospital sector was approximately $35 000. Assuming health inflation of 2.7 per cent per annum, this cost converts to approximately $50 000 in 2015-16 dollars. However, this may be an overestimate of the cost of IADs as cost per device is likely to fall as more models become available (Baker Heart Research Institute, Melbourne, pers. comm., 22 August 2005).

Offsetting cost savings

According to Goldman et al. (2004), IADs may deliver offsetting cost savings in other areas of the health system by reducing the number of strokes. Goldman et al. (2004) estimate that if IADs were available to 100 per cent of the eligible population, then they could reduce the incidence of stroke by 50 per cent (equivalent to the percentage of strokes that are attributable to AF) and reduce hospitalisations due to recurrent AF by 50 per cent.

Estimates of the costs of stroke provide a starting point for estimating offsetting cost savings from a reduction in the incidence of stroke.[5] In 2000-01, there were approximately 40 000 hospitalisations in Australia where stroke was the principal diagnosis at an estimated cost of $392 million (AIHW 2004b). In addition, a further $530 million was spent in other areas of the health system treating strokes, including $442 million on aged care homes (figure 3.3).

Figure 3.3 **Health system expenditure on strokes, 2000-01**



Data source: AIHW (2004b).

By reducing the incidence of stroke, IADs are estimated to have the potential to deliver offsetting cost savings of approximately $690 million in 2015-16 dollars.

5 See Mathers et al. (1998) for a discussion of the difficulties in interpreting estimates of the cost of disease.

Summarising potential net expenditure impacts of IADs

Using the assumptions outlined above regarding volume, unit cost and offsetting cost savings, the following estimates are derived regarding the net expenditure impacts of IADs for the treatment of AF in 2015-16:

- IADs are supplied to approximately 126 000 persons at a cost of approximately $50 000 per person. This results in an increase in health expenditure of approximately $6 billion;
- offsetting cost savings of approximately $265 million are achieved from a corresponding 20 per cent reduction in strokes; and
- the net expenditure impacts of IADs are therefore estimated at approximately $2.2 billion per annum in 2015-16 dollars.

Comparison of estimated net expenditure impacts in 2015-16: IADs and warfarin

To further illustrate estimated net expenditure impacts of IADs, it is useful to compare with the case where IADs are not widely available and anticoagulant drugs remain a key treatment in preventing stroke in AF sufferers.

Warfarin is an anticoagulant commonly prescribed to Australian patients with AF to reduce the risk of stroke (Ninio 2000). In 2003-04, there were approximately 1.7 million PBS prescriptions recorded for *warfarin* at a cost to the Australian Government of approximately $9 million or $5 per prescription (HIC 2005b). Monitoring of the effects of *warfarin* therapy through laboratory testing — approximately 20 times a year (NHMRC 1997) — is also required.[6] The Medicare Benefits Schedule currently lists the fee for a prothrombin time test (the test used to monitor anticoagulation therapy) at approximately $14 (DoHA 2003).

Assuming that prescriptions for *warfarin* continue to grow at 11 per cent per annum (a rate equivalent to the average growth rate for *warfarin* prescriptions between 1993-94 and 2003-04) and that the cost of a *warfarin* prescription and monitoring grows in line with health inflation, the expenditure impact of current *warfarin* treatment continuing is approximately $50 million per annum in 2015-16 dollars.

Warfarin therapy is also considered to deliver offsetting cost savings by reducing the number of strokes. However, there would appear to be limited estimates of these cost savings. The National Health and Medical Research Council (NHMRC 1997) used a study by Gage et al. (1995) to estimate that *warfarin* prevented 520 strokes,

6 More frequent monitoring is normally undertaken in the first few weeks after a patient starts anticoagulant therapy.

saving approximately $25 million per year. This suggests that continued use of *warfarin* would deliver offsetting cost savings of approximately $35 million.

It should be noted that over the next ten years, *warfarin* may itself be replaced by new anticoagulant drugs such as direct thrombin inhibitors (Eikelboom and Hankey 2004). While these drugs are likely to be more expensive than *warfarin*, they will negate the need for regular blood tests to monitor anticoagulation activity (Baker Heart Research Institute, Melbourne, pers. comm., 22 August 2005; Eikelboom and Hankey 2004).

The analysis presented above shows that IADs would be likely to increase health expenditure in Australia significantly, largely due to the high unit cost of IADs and the large eligible population. This analysis also implicitly assumes acceptable cost-effectiveness outcomes for IADs compared to other treatments for AF. However, the high potential costs of such an intervention may make widespread use problematic. It may also be the case that IADs are themselves replaced over the next 20 years by catheter-based ablation techniques aimed at stopping the initiation or maintenance of AF (Goldman et al. 2004).

3.3 Robotic-assisted surgery for prostate cancer

Robotic-assisted surgery represents the latest advancement in surgical treatment of prostate cancer (chapter 11). A number of factors suggest that this type of surgery will become widely available over the next ten years in Australia:

- the technology has recently become available in two private hospitals in Australia (chapter 11);
- the Australian Safety and Efficacy Register of New Interventional Procedures – Surgical (ASERNIP-S 2004) conducted a 'technology overview' of a robotic system in 2004;
- the Medical Services Advisory Committee is currently undertaking a review of minimally invasive robotic surgery for radical prostatectomy (MSAC 2005);
- the Victorian Department of Human Services (sub. 24) stated that several Victorian public hospitals have indicated a desire to install a robotic system; and
- in the United States, approximately 10 per cent of radical prostatectomy procedures are robot-assisted (Binder et al. 2004).

Among Australian men, prostate cancer is the most common cancer diagnosed (over 11 000 cases diagnosed in 2001) and the second leading cause of cancer death (AIHW and AACR 2004). In 2002-03, there were over 25 500 public and private hospital separations recorded for prostatectomy procedures, including

approximately 3000 for radical prostatectomy (AIHW 2005a). Radical prostatectomy is one option, along with radical radiotherapy and watchful waiting, for treatment of localised prostate cancer.

Robotic prostatectomy (RP) is considered a substitute for open radical prostatectomy and laparoscopic radical prostatectomy (El-Hakim and Tewari 2004). Open radical prostatectomy is the most common prostatectomy technique performed in Australia and involves the removal of the whole of the prostate gland through a cut in the abdomen (Andrology Australia 2003). Laparoscopic radical prostatectomy is a less invasive procedure which aims to reduce abdominal wall morbidity and improve operative precision by providing better visualisation of the surgery site (ASERNIP-S 2003).[7] RP essentially involves performing laparoscopic surgery with the aid of robotic technology (El-Hakim and Tewari 2004).

RP is considered to have the potential to enhance surgeons' technical abilities and thus facilitate more precise removal of the cancer and better preservation of sexual function and urinary control (El-Hakim and Tewari 2004). Another key benefit is shorter hospital stays (ASERNIP-S 2004). Other general benefits of robotic-assisted surgery are outlined in box 11.3.

A number of overseas studies have recently compared the efficacy and safety of RP with open radical prostatectomy and laparoscopic radical prostatectomy procedures (El-Hakim and Tewari 2004). While ASERNIP-S (2004) found that there was insufficient evidence to determine the safety or efficacy of RP compared to open radical prostatectomy and laparoscopic prostatectomy, a recent review of RP by El-Hakim and Tewari (2004) found that:

> … short-term clinical and pathological results are comparable to those with open and laparoscopic prostatectomy. (p. 1)

For the purposes of illustrating future expenditure impacts of treatment of prostate cancer, it has been assumed that RP is the preferred surgical technique for Australian men undergoing a radical prostatectomy in 2015-16.

Estimated net expenditure impacts of a robotic-assisted surgery for prostate cancer

This section describes the assumptions used to derive an estimate of future net expenditure impacts from the use of robotic-assisted surgery for prostate cancer in 2015-16.

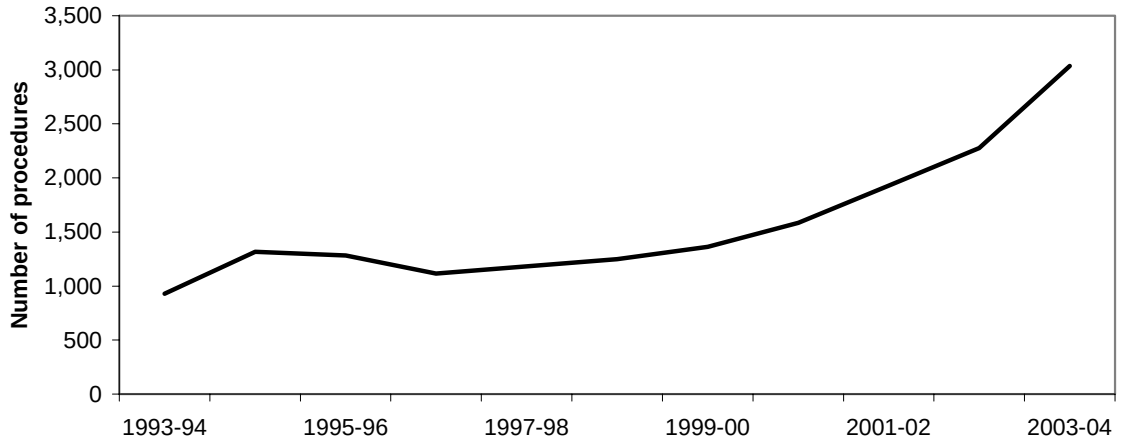
[7] ASERNIP-S (2003) found that the availability of laparoscopic radical prostatectomy was not widespread, with the technique being only available at specialised urological units.

Volume impacts

As discussed above, RP is a substitute for open and laparoscopic radical prostatectomies (El-Hakim and Tewari 2004). In addition, RP may reduce side effects of radical prostatectomies and therefore positively influence the decision of men to undertake the surgery. Accordingly, it is assumed that RP accounts for an additional 5 per cent per annum average increase in the volume of radical prostatectomy procedures.

Data from the Health Insurance Commission (HIC 2005b) suggest that annual growth in radical prostatectomy procedures in Australia averaged 13 per cent between 1993-94 and 2003-04 (figure 3.4). Taking into account the assumed extra 5 per cent growth in the procedure from a switch to RP, implies there would be approximately 22 000 procedures performed in 2015-16.

Figure 3.4 **Radical prostatectomy procedures, 1993-94 to 2003-04**



Data source: HIC (2005a).

This projected increase in the number of radical prostatectomy procedures is broadly consistent with:

- a projected 22 per cent increase in the burden of disease from prostate cancer in Victoria between 1996 and 2016 (VDHS 1999); and
- a six-fold increase in the proportion of men undergoing radical prostatectomy in Western Australia between 1982 and 2001 (Hall et al. 2004).

Unit cost impacts

Wykypiel et al. (2004) estimated the unit capital cost of a RP surgical system at approximately US$1 million. Ongoing service and maintenance costs are also

significant, as most instruments on the robotic system can be used for only ten procedures. Morgan et al. (2005) estimated the cost of a service contract for a robotic system at approximately US$100 000 per annum. Other important costs of the system are operating room time and training costs, although these are expected to fall over time, resulting in a significant reduction in the associated cost of the technology. RP also incurs other normal hospital intraoperative and postoperative costs such as equipment, the surgeon's professional fee, hospital room and intravenous fluids/medications (Lotan et al. 2004).

A cost comparison of conventional open radical prostatectomy and RP by Lotan et al. (2004) found that RP was US$1726 or approximately 25 per cent more expensive than conventional surgery. The large difference resulted from the high initial purchase cost of the robot and the high cost of equipment per case.

In this analysis, the unit cost of an open radical prostatectomy is derived from the weighted average cost of Australian Refined Diagnostic Related Group (AR-DRG) L03A in both the public and private sectors in 2002-03 (DoHA 2005b).[8] This cost is calculated at approximately $13 000 or approximately $18 000 in 2015-16 dollars. Following Lotan et al. (2004) it has been assumed that RP is 25 per cent more expensive than open radical prostatectomy implying a cost per procedure of RP of approximately $23 000 in 2015-16 dollars.

Offsetting cost savings

Offsetting cost savings from RP are likely to occur through a reduction in hospital stay. A US study by Guru et al. (2004) found that average hospital stay for RP patients was 1.7 days, compared to 2.4 days for open prostatectomy patients. This contributed to a 50 per cent reduction in ward care costs for RP patients. In Australia, the direct ward care costs for AR-DRG L03A in the public sector in 2002-03 were approximately $2000 (DoHA 2005b), which suggests a cost saving of approximately $1000 in ward care costs from switching to RP. Assuming 22 000 radical prostatectomy procedures would be performed in 2015-16, a switch to RP could result in a potential saving of approximately $30 million in 2015-16 dollars.

RP may also deliver offsetting cost savings by reducing complications associated with radical prostatectomy such as incontinence and loss of sexual function. However, these savings are difficult to quantify. To date, there would appear to be no studies estimating savings that may arise to the patient and society from lower

8 AR-DRG L03 includes radical prostatectomy, kidney, ureter and bladder procedures for cancers (DoHA 1998).

levels of complications. As such, these cost savings are not quantified in this analysis.

In addition, regardless of whether it is carried out by conventional means or with robotic-assistance, radical prostatectomy is likely to also deliver cost savings by avoiding the costs of treatment of advanced stage prostate cancer.[9] In Australia, advanced-stage prostate cancer is commonly treated through anti-androgen drugs (Boyer 1996). In 2004-05, there were approximately 102 000 PBS/RPBS prescriptions recorded for anti-androgen drugs used to treat advanced prostate cancer (HIC 2005b). These cost the PBS/RPBS approximately $260 per prescription. This suggests that 22 000 RP procedures would save at least $10 million in 2015-16 dollars.[10]

Summarising potential net expenditure impacts of RP

Using the assumptions outlined above regarding volume, unit cost and offsetting cost savings, the following estimates are derived regarding the net expenditure impact of RP for treatment of prostate cancer in 2015-16:

- approximately 22 000 radical prostatectomies are performed using RP at a cost of approximately $23 000 per procedure. This results in an increase in health expenditure of approximately $500 million;
- offsetting cost savings of at least $40 million are achieved from a reduction in ward care costs and avoidance of treatment costs of advanced prostate cancer; and
- the net expenditure impact of RP is therefore estimated at approximately $460 million.

Comparison of estimated net expenditure impacts in 2015-16: RP and open radical prostatectomy surgery

To further illustrate estimated net expenditure impacts of RP, it is useful to compare with the case where RP is not widely used and conventional open surgery remains the preferred method for radical prostatectomy.

9 When diagnosed at an advanced stage, or the cancer has progressed after diagnosis and initial treatment, it is generally accepted that radical prostatectomy is not an option for treatment for prostate cancer (Chamberlain et al. 1997).

10 Assumes one prescription avoided each year for every patient who undergoes radical prostatectomy.

As outlined above, the unit costs of conventional open radical prostatectomy are estimated at approximately $18 000 in 2015-16 dollars. Assuming an annual growth rate of procedures of 13 per cent, then there would be approximately 13 000 radical prostatectomy procedures performed in 2015-16, resulting in a gross expenditure impact of approximately $240 million. In addition, as noted above, radical prostatectomy is likely to deliver cost savings by avoiding treatment of advanced prostate cancer. These offsetting cost savings are estimated to be at least $5 million in 2015-16 dollars.

In summary, RP would be likely increase health expenditure in Australia, although to a significantly smaller degree than a type 2 DM prevention drug or IADs. However, it may also be the case that key drivers of cost for RP will decrease in future, for example, the cost of the robot may decrease and operating room times may decrease as surgical teams become more familiar with the technology. Other benefits such as improvement in postoperative quality of life and a faster return to work may also make RP a cost-effective option (Morgan et al. 2005).

3.4 Vaccine for treatment of established AD

Goldman et al. (2004) identify a vaccine for established AD as a likely future breakthrough in the treatment of neurological diseases. AD is the progressive loss of memory and cognitive function and is characterised pathologically by the development of plaques of beta-amyloid (Aβ) in brain cells (Goldman et al. 2004). AD is the most common cause of dementia disorders (comprising 50 to 70 per cent of cases) and is currently incurable (Access Economics 2003).

In Australia, AD is estimated to affect approximately 100 000 persons (Access Economics 2004). The AIHW (2004b) has estimated that health system expenditure on the treatment and prevention of AD and other dementias cost approximately $2.2 billion in 2000-01, with aged care homes accounting for 86 per cent or $1.9 billion of this expenditure.

While AD is currently incurable, a number of treatment and management strategies have been developed over the past 20 years. These include improving the quality of care for persons suffering from dementia and addressing lifestyle factors that may contribute to onset of the disease (Access Economics 2003). Current anti-dementia pharmacotherapies approved for use in Australia to improve cognitive function are the cholinesterase inhibitors (*donepezil*, *galantamine*, *rivastigmine* and *tacrine*. These drugs act to increase and prolong acetylcholine levels and have been shown to enhance cognitive functioning (Bryant et al. 2003).

In addition to cholinesterase inhibitors, a number of other drugs are currently being trialled for treatment of AD (Goldman et al. 2004). Some of these new drugs are based on the amyloid hypothesis, which is that:

> … Aβ deposits in the brains of patients with AD are responsible for the disease. (Goldman et al. 2004, p. 179)

One of the most promising streams of research in this area has been the development of a 'vaccine' to clear amyloid once it has been deposited. The likely effect of such a vaccine would not be to cure AD, but to decrease the rate of progression by between 20 and 50 per cent (Goldman et al. 2004). Broytman and Malter (2004) report that trials of the vaccine on animals have demonstrated positive results such as:

- markedly reduced Aβ deposition;
- preservation of normal neurone architecture; and
- improved performance in memory and spatial learning tasks.

Following extensive safety studies of the vaccine on several animal species such as mice, rabbits, guinea pigs and monkeys, a human trial of the AD vaccine commenced in 2001 (Broytman and Malter 2004; Schenk et al. 2002). The vaccine successfully completed Phase I trials, however Phase II trials were terminated early after 5 per cent of patients given the vaccine developed aseptic meningoencephalitis (inflammation of the brain) (Broytman and Malter 2004). This prompted renewed criticism of the vaccine approach from sceptics such as Robinson et al. (2004), who have argued that Aβ deposits may be a proactive response of the brain to the presence of disease (Bradbury 2003).

Despite unforeseen side effects, the vaccine trial has also reported some positive results, including a significant reduction of the cognitive decline in patients who received the vaccine (Hock et al. 2003; Schenk et al. 2002). Knowledge gained from the human trial is currently being used in research with the aim of refining the vaccine to avoid side effects (Broytman and Malter 2004; Gelinas et al. 2004). For example, Bowers et al. (2005) recently tested a vaccine on mice aimed at clearing existing amyloid plaque deposits and preventing the formation of new deposits without adverse side effects such as inflammation. By including a tetanus toxin, the authors recently reported that side effects disappeared (Doherty 2004). According to Doherty (2004), this development offers promise that:

> … a level of control over the immune response to an AD vaccine can be attained. (Doherty 2004, p.731)

To illustrate the potential effects of future technology on treatment of neurological diseases, it has been assumed that human trials into an AD vaccine continue and

that the vaccine is demonstrated to be successful in slowing the progression of AD by 20 per cent by 2015-16 (Goldman et al. 2004). Based on a current rate of AD progression from diagnosis to death of about 10 years, this breakthrough could slow the progression of AD by approximately 2 years.

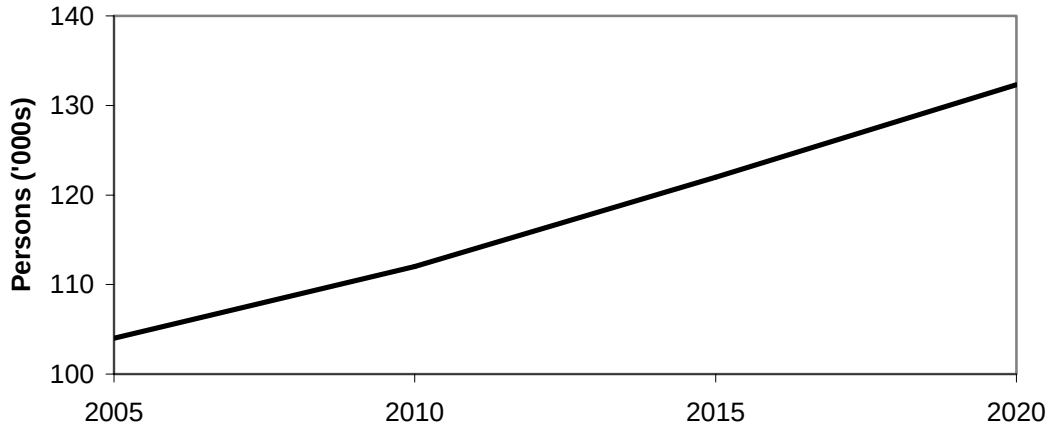
Estimated net expenditure impacts of a vaccine for established AD

This section describes the assumptions used to derive an estimate of the net expenditure impacts of an AD vaccine in 2015-16.

Volume impacts

Potentially all patients with a diagnosis of AD could be eligible for an AD vaccine (Goldman et al. 2004). Access Economics (2004) estimated that in the absence of a treatment that delays the onset of AD, the prevalence of AD in Australia will be approximately 120 000 persons by 2015 (figure 3.5**)**.

Figure 3.5 **Estimated prevalence of AD, 2005 to 2020**



Data source: Access Economics (2004).

Unit cost impacts

Goldman et al. (2004) estimate the unit cost of an AD vaccine at approximately US$3000. Assuming a health inflation rate of 2.7 per cent and converting to Australian dollars, the unit cost of an AD vaccine is estimated at approximately A$5500 in 2015-16 dollars.

Offsetting cost savings

By delaying the progression of AD, an AD vaccine could potentially deliver cost savings in aged care expenditure. However, it is extremely difficult to estimate aged care savings from an AD vaccine, as there are likely to be two opposing effects on aged care expenditure:

- firstly, expenditure may decrease, as persons with AD delay their entry into aged care facilities. For example, a person may enter an aged care facility seven years into the progression of the disease instead of five; and
- secondly, expenditure may increase, as once persons with AD enter aged care facilities, they stay longer due to slower progression of the disease. For example, once a person enters an aged care facility they may stay up to six or seven years instead of five.

Given the difficulties in estimating these two opposing effects, offsetting cost savings are not estimated in this analysis.

Summarising potential net expenditure impacts of a vaccine for treatment of established AD

The assumptions outlined above regarding volume and unit cost suggest that in 2015-16 an AD vaccine could be available to approximately 120 000 persons at a unit cost of approximately $5500. This results in an increase in health expenditure of approximately $330 million, although this does not take into account any possible offsetting cost savings of an AD vaccine.

Comparison of net expenditure impacts – AD vaccine and cholinesterase inhibitors

To further illustrate estimated net expenditure impacts of an AD vaccine, it is useful to compare with the case where the vaccine does not prove successful and cholinesterase inhibitors remain the primary pharmacologic treatment for AD.

In 2003-04, there were approximately 220 000 PBS prescriptions for cholinesterase inhibitors at a cost to the Australian Government of approximately $34 million or $170 per prescription (HIC 2005b). Current PBS guidelines for obtaining access to subsidised cholinesterase inhibitors are complex, and as such only around 18 500 or 20 per cent of Australians diagnosed with AD were estimated to be using cholinesterase inhibitors in 2002 (Access Economics 2004).

Assuming that 20 per cent of those diagnosed with AD are eligible for PBS subsidy of cholinesterase inhibitors in 2015-16 and that the average cost of a prescription for these drugs grows in line with projected health inflation, the net expenditure impact

of continuing with current treatment is estimated to be at least $65 million in 2015-16 dollars.

The analysis presented above suggests that an AD vaccine would be likely to increase health expenditure in Australia. However, it may be that the vaccine will have a high probability of acceptance in Australia if it can demonstrate benefits over cholinesterase inhibitors such as improvements in quality of life.

Summing up

Based on the assumptions outlined in this technical paper, all four advances in medical technology examined would appear likely to increase health expenditure in the future. Because the results depend heavily on assumptions regarding volumes, unit costs, offsetting cost savings and health inflation, they serve only as illustrations of possible expenditure impacts of future technological advances. Importantly, these technologies also have the potential to deliver significant benefits, which have not been evaluated in this expenditure analysis.

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4 Distributional Data

4.1 Separation rates by age and sex [a]

Table 4.1 **Catheterisation**

Year	*Sex*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1993-94	Men	1.40	1.42	1.43
	Women	0.60	0.61	0.61
1994-95	Men	1.41	1.43	1.44
	Women	0.59	0.60	0.60
1995-96	Men	1.40	1.41	1.42
	Women	0.61	0.61	0.62
1996-97	Men	1.39	1.40	1.42
	Women	0.61	0.62	0.63
1997-98	Men	1.40	1.41	1.42
	Women	0.61	0.62	0.63
1998-99	Men	1.38	1.39	1.41
	Women	0.63	0.64	0.64
1999-00	Men	1.39	1.40	1.41
	Women	0.62	0.63	0.64
2000-01	Men	1.38	1.39	1.40
	Women	0.63	0.64	0.65
2001-02	Men	1.37	1.38	1.39
	Women	0.64	0.65	0.66
2002-03	Men	1.36	1.38	1.39
	Women	0.65	0.65	0.66
2003-04	Men	1.36	1.37	1.38
	Women	0.65	0.66	0.67

a Age standardised separation rate is based on separation rate per 1000 people. Confidence limits set at 95 per cent.

Source: AIHW unpublished data.

Table 4.2 **Angiography**

Year	*Sex*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1993-94	Men	1.40	1.42	1.43
	Women	0.59	0.60	0.61
1994-95	Men	1.41	1.43	1.44
	Women	0.59	0.60	0.60
1995-96	Men	1.40	1.41	1.42
	Women	0.61	0.61	0.62
1996-97	Men	1.39	1.41	1.42
	Women	0.61	0.62	0.63
1997-98	Men	1.40	1.41	1.42
	Women	0.61	0.62	0.63
1998-99	Men	1.39	1.40	1.41
	Women	0.62	0.63	0.64
1999-00	Men	1.39	1.40	1.42
	Women	0.62	0.63	0.64
2000-01	Men	1.38	1.39	1.40
	Women	0.63	0.64	0.65
2001-02	Men	1.37	1.38	1.39
	Women	0.64	0.65	0.66
2002-03	Men	1.37	1.38	1.39
	Women	0.65	0.65	0.66
2003-04	Men	1.36	1.37	1.38
	Women	0.65	0.66	0.67

Source: AIHW unpublished data.

Table 4.3 **Angioplasty without stent**

Year	*Sex*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1993-94	Men	1.50	1.54	1.57
	Women	0.46	0.48	0.50
1994-95	Men	1.51	1.54	1.57
	Women	0.45	0.47	0.49
1995-96	Men	1.50	1.53	1.56
	Women	0.47	0.49	0.51
1996-97	Men	1.48	1.52	1.56
	Women	0.48	0.50	0.52
1997-98	Men	1.51	1.55	1.59
	Women	0.46	0.48	0.50
1998-99	Men	1.46	1.52	1.57
	Women	0.48	0.51	0.54
1999-00	Men	1.47	1.53	1.58
	Women	0.47	0.50	0.53
2000-01	Men	1.50	1.56	1.62
	Women	0.45	0.48	0.52
2001-02	Men	1.48	1.54	1.60
	Women	0.47	0.50	0.53
2002-03	Men	1.45	1.52	1.59
	Women	0.48	0.51	0.55
2003-04	Men	1.44	1.52	1.59
	Women	0.48	0.52	0.56

Source: AIHW unpublished data.

Table 4.4 **Angioplasty with stent**

Year	*Sex*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1993-94	Men	-2.31	2.41	7.13
	Women		0.00	
1994-95	Men	1.51	1.61	1.71
	Women	0.36	0.41	0.45
1995-96	Men	1.51	1.57	1.62
	Women	0.43	0.46	0.49
1996-97	Men	1.52	1.56	1.59
	Women	0.45	0.47	0.49
1997-98	Men	1.52	1.55	1.58
	Women	0.46	0.47	0.49
1998-99	Men	1.51	1.54	1.57
	Women	0.47	0.49	0.50
1999-00	Men	1.52	1.55	1.57
	Women	0.47	0.49	0.50
2000-01	Men	1.51	1.54	1.56
	Women	0.48	0.49	0.51
2001-02	Men	1.52	1.54	1.56
	Women	0.48	0.49	0.51
2002-03	Men	1.52	1.54	1.57
	Women	0.48	0.49	0.50
2003-04	Men	1.52	1.55	1.57
	Women	0.48	0.49	0.50

Source: AIHW unpublished data.

Table 4.5 **Coronary artery bypass graft**

Year	*Sex*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1993-94	Men	1.58	1.61	1.63
	Women	0.42	0.44	0.45
1994-95	Men	1.58	1.61	1.64
	Women	0.42	0.43	0.45
1995-96	Men	1.58	1.60	1.63
	Women	0.43	0.44	0.46
1996-97	Men	1.58	1.60	1.63
	Women	0.43	0.45	0.46
1997-98	Men	1.57	1.60	1.63
	Women	0.43	0.45	0.46
1998-99	Men	1.57	1.60	1.63
	Women	0.44	0.45	0.47
1999-00	Men	1.58	1.60	1.63
	Women	0.43	0.45	0.46
2000-01	Men	1.58	1.61	1.63
	Women	0.43	0.45	0.46
2001-02	Men	1.57	1.60	1.63
	Women	0.43	0.45	0.46
2002-03	Men	1.58	1.61	1.64
	Women	0.43	0.44	0.46
2003-04	Men	1.59	1.62	1.65
	Women	0.42	0.43	0.45

Source: AIHW unpublished data.

Table 4.6 **Implantable Cardiac Defibrillator**

Year	*Sex*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1993-94	Men	1.36	1.65	1.93
	Women	0.28	0.42	0.55
1994-95	Men	1.43	1.70	1.97
	Women	0.24	0.36	0.48
1995-96	Men	1.33	1.55	1.77
	Women	0.39	0.51	0.63
1996-97	Men	1.53	1.73	1.93
	Women	0.26	0.35	0.43
1997-98	Men	1.54	1.69	1.85
	Women	0.31	0.38	0.45
1998-99	Men	1.49	1.63	1.77
	Women	0.36	0.43	0.50
1999-00	Men	1.56	1.69	1.81
	Women	0.32	0.38	0.44
2000-01	Men	1.54	1.67	1.80
	Women	0.35	0.41	0.47
2001-02	Men	1.53	1.65	1.77
	Women	0.37	0.42	0.48
2002-03	Men	1.63	1.73	1.83
	Women	0.30	0.34	0.38
2003-04	Men	1.63	1.72	1.81
	Women	0.31	0.35	0.39

Source: AIHW unpublished data.

Table 4.7 **Lens insertion and or removal**

Year	*Sex*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1993-94	Men	0.95	0.96	0.97
	Women	1.02	1.03	1.04
1994-95	Men	0.94	0.95	0.96
	Women	1.03	1.04	1.05
1995-96	Men	0.94	0.95	0.96
	Women	1.03	1.04	1.05
1996-97	Men	0.93	0.94	0.95
	Women	1.04	1.05	1.06
1997-98	Men	0.93	0.94	0.95
	Women	1.04	1.05	1.06
1998-99	Men	0.94	0.95	0.96
	Women	1.04	1.05	1.05
1999-00	Men	0.95	0.96	0.97
	Women	1.03	1.04	1.05
2000-01	Men	0.94	0.95	0.96
	Women	1.04	1.05	1.05
2001-02	Men	0.93	0.94	0.95
	Women	1.05	1.06	1.07
2002-03	Men	0.92	0.93	0.94
	Women	1.06	1.06	1.07
2003-04	Men	0.94	0.94	0.95
	Women	1.05	1.06	1.06

Source: AIHW unpublished data.

4.2 Separation rates by remoteness area

Table 4.8 **Catheterisation**

Year	*Remoteness area*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1996-97	Major City	1.00	1.01	1.02
	Regional	0.69	0.70	0.71
	Remote	0.44	0.48	0.52
1997-98	Major City	1.07	1.08	1.09
	Regional	0.79	0.80	0.81
	Remote	0.49	0.53	0.57
1998-99	Major City	1.08	1.09	1.10
	Regional	0.82	0.83	0.84
	Remote	0.60	0.64	0.68
1999-00	Major City	1.05	1.06	1.07
	Regional	0.84	0.85	0.86
	Remote	0.57	0.60	0.64
2000-01	Major City	1.02	1.02	1.03
	Regional	0.92	0.93	0.94
	Remote	0.88	0.93	0.97
2001-02	Major City	1.00	1.01	1.02
	Regional	0.93	0.94	0.95
	Remote	0.85	0.90	0.94
2002-03	Major City	0.98	0.98	0.99
	Regional	0.96	0.97	0.98
	Remote	0.96	1.00	1.05
2003-04	Major City	0.98	0.99	0.99
	Regional	0.96	0.97	0.98
	Remote	0.87	0.91	0.95

Source: AIHW unpublished data.

Table 4.9 **Angiography**

Year	*Remoteness area*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1996-97	Major City	1.00	1.01	1.02
	Regional	0.68	0.69	0.70
	Remote	0.44	0.48	0.52
1997-98	Major City	1.08	1.09	1.10
	Regional	0.77	0.78	0.80
	Remote	0.49	0.54	0.58
1998-99	Major City	1.08	1.09	1.10
	Regional	0.82	0.83	0.84
	Remote	0.61	0.65	0.69
1999-00	Major City	1.05	1.06	1.07
	Regional	0.84	0.85	0.86
	Remote	0.57	0.61	0.64
2000-01	Major City	1.02	1.02	1.03
	Regional	0.92	0.93	0.94
	Remote	0.88	0.93	0.97
2001-02	Major City	1.00	1.01	1.02
	Regional	0.93	0.94	0.95
	Remote	0.85	0.90	0.94
2002-03	Major City	0.97	0.98	0.99
	Regional	0.96	0.97	0.98
	Remote	0.96	1.00	1.05
2003-04	Major City	0.98	0.99	1.00
	Regional	0.96	0.97	0.98
	Remote	0.87	0.91	0.95

Source: AIHW unpublished data.

Table 4.10 **Angioplasty without stent**

Year	*Remoteness area*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1996-97	Major City	0.95	0.98	1.01
	Regional	0.72	0.75	0.79
	Remote	0.36	0.47	0.58
1997-98	Major City	1.06	1.09	1.12
	Regional	0.75	0.79	0.83
	Remote	0.28	0.39	0.50
1998-99	Major City	1.08	1.12	1.16
	Regional	0.73	0.77	0.81
	Remote	0.51	0.66	0.82
1999-00	Major City	1.05	1.09	1.13
	Regional	0.76	0.81	0.86
	Remote	0.30	0.45	0.59
2000-01	Major City	1.02	1.07	1.11
	Regional	0.80	0.86	0.91
	Remote	0.54	0.74	0.94
2001-02	Major City	1.04	1.09	1.13
	Regional	0.76	0.82	0.87
	Remote	0.32	0.48	0.65
2002-03	Major City	1.02	1.07	1.12
	Regional	0.74	0.80	0.86
	Remote	0.52	0.76	1.01
2003-04	Major City	1.06	1.12	1.17
	Regional	0.67	0.73	0.80
	Remote	0.41	0.66	0.91

Source: AIHW unpublished data.

Table 4.11 **Angioplasty with stent**

Year	*Remoteness area*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1996-97	Major City	0.96	0.99	1.01
	Regional	0.69	0.72	0.76
	Remote	0.37	0.50	0.63
1997-98	Major City	1.05	1.08	1.10
	Regional	0.79	0.82	0.85
	Remote	0.43	0.52	0.62
1998-99	Major City	1.08	1.10	1.12
	Regional	0.79	0.81	0.84
	Remote	0.50	0.59	0.67
1999-00	Major City	1.07	1.08	1.10
	Regional	0.78	0.81	0.83
	Remote	0.45	0.53	0.60
2000-01	Major City	1.04	1.06	1.08
	Regional	0.85	0.87	0.89
	Remote	0.76	0.85	0.94
2001-02	Major City	1.03	1.05	1.07
	Regional	0.86	0.88	0.90
	Remote	0.66	0.74	0.82
2002-03	Major City	1.02	1.04	1.05
	Regional	0.85	0.87	0.89
	Remote	0.69	0.76	0.84
2003-04	Major City	1.03	1.05	1.06
	Regional	0.85	0.87	0.88
	Remote	0.64	0.71	0.78

Source: AIHW unpublished data.

Table 4.12 **Coronary artery bypass graft**

Year	*Remoteness area*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1996-97	Major City	0.98	1.00	1.02
	Regional	0.70	0.73	0.75
	Remote	0.42	0.52	0.61
1997-98	Major City	1.05	1.07	1.09
	Regional	0.79	0.82	0.85
	Remote	0.40	0.48	0.57
1998-99	Major City	1.05	1.07	1.09
	Regional	0.84	0.86	0.88
	Remote	0.51	0.59	0.68
1999-00	Major City	1.03	1.05	1.07
	Regional	0.86	0.88	0.90
	Remote	0.46	0.53	0.61
2000-01	Major City	1.00	1.02	1.04
	Regional	0.92	0.94	0.97
	Remote	0.65	0.74	0.84
2001-02	Major City	0.99	1.01	1.03
	Regional	0.94	0.96	0.99
	Remote	0.71	0.81	0.91
2002-03	Major City	0.97	0.99	1.01
	Regional	0.98	1.01	1.03
	Remote	0.72	0.82	0.92
2003-04	Major City	0.96	0.98	1.00
	Regional	0.99	1.02	1.04
	Remote	0.70	0.80	0.90

Source: AIHW unpublished data.

Table 4.13 **Implantable cardiac defibrillators**

Year	*Remoteness area*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1996-97	Major City	0.94	1.08	1.22
	Regional	0.45	0.61	0.78
	Remote	0.00	0.00	0.00
1997-98	Major City	1.02	1.14	1.26
	Regional	0.55	0.69	0.83
	Remote	0.01	0.58	1.16
1998-99	Major City	1.02	1.12	1.22
	Regional	0.68	0.79	0.91
	Remote	0.04	0.35	0.67
1999-00	Major City	1.02	1.11	1.20
	Regional	0.69	0.79	0.90
	Remote	0.19	0.61	1.03
2000-01	Major City	0.92	1.00	1.09
	Regional	0.85	0.97	1.09
	Remote	0.51	0.99	1.48
2001-02	Major City	0.92	1.00	1.08
	Regional	0.87	0.98	1.09
	Remote	0.46	0.86	1.26
2002-03	Major City	0.94	1.00	1.06
	Regional	0.89	0.98	1.07
	Remote	0.46	0.79	1.11
2003-04	Major City	0.96	1.02	1.08
	Regional	0.87	0.94	1.02
	Remote	0.57	0.86	1.15

Source: AIHW unpublished data.

Table 4.14 **Lens insertion and or removal**

Year	*Remoteness area*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1996-97	Major City	0.97	0.98	0.98
	Regional	0.83	0.84	0.85
	Remote	0.65	0.70	0.75
1997-98	Major City	1.04	1.05	1.06
	Regional	0.87	0.88	0.90
	Remote	0.65	0.70	0.75
1998-99	Major City	1.01	1.02	1.03
	Regional	0.95	0.96	0.97
	Remote	0.65	0.69	0.73
1999-00	Major City	1.02	1.03	1.04
	Regional	0.94	0.95	0.95
	Remote	0.75	0.79	0.82
2000-01	Major City	0.97	0.98	0.99
	Regional	1.02	1.03	1.04
	Remote	1.04	1.08	1.13
2001-02	Major City	0.98	0.99	1.00
	Regional	0.97	0.98	0.99
	Remote	0.98	1.03	1.07
2002-03	Major City	0.98	0.98	0.99
	Regional	0.99	1.00	1.00
	Remote	1.00	1.04	1.08
2003-04	Major City	0.97	0.98	0.99
	Regional	1.00	1.01	1.02
	Remote	0.79	0.83	0.86

Source: AIHW unpublished data.

Table 4.15 **Hip replacement** [a]

Year	*Remoteness area*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1996-97	Major City	0.91	0.92	0.94
	Regional	0.91	0.94	0.96
	Remote	0.51	0.61	0.71
1997-98	Major City	0.98	1.00	1.01
	Regional	0.98	1.00	1.03
	Remote	0.47	0.56	0.65
1998-99	Major City	0.99	1.01	1.02
	Regional	0.96	0.98	1.00
	Remote	0.65	0.74	0.82
1999-00	Major City	1.00	1.02	1.03
	Regional	0.95	0.97	1.00
	Remote	0.50	0.58	0.65
2000-01	Major City	0.94	0.95	0.97
	Regional	1.07	1.09	1.12
	Remote	0.70	0.79	0.88
2001-02	Major City	0.94	0.95	0.97
	Regional	1.05	1.08	1.10
	Remote	0.82	0.91	0.99
2002-03	Major City	0.94	0.95	0.97
	Regional	1.06	1.09	1.11
	Remote	0.74	0.82	0.90
2003-04	Major City	0.94	0.96	0.98
	Regional	1.06	1.08	1.10
	Remote	0.74	0.82	0.90

a Includes both primary and revision hip replacement surgery.

Source: AIHW unpublished data.

Table 4.16 **Knee replacement**

Year	*Remoteness area*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
1996-97	Major City	0.88	0.90	0.92
	Regional	0.98	1.01	1.04
	Remote	0.62	0.75	0.88
1997-98	Major City	0.95	0.97	0.99
	Regional	1.03	1.06	1.09
	Remote	0.61	0.73	0.85
1998-99	Major City	0.98	1.00	1.02
	Regional	0.97	0.99	1.02
	Remote	0.60	0.69	0.78
1999-00	Major City	0.98	0.99	1.01
	Regional	1.00	1.02	1.04
	Remote	0.58	0.67	0.76
2000-01	Major City	0.92	0.94	0.96
	Regional	1.09	1.11	1.14
	Remote	0.89	0.99	1.09
2001-02	Major City	0.92	0.94	0.95
	Regional	1.08	1.11	1.13
	Remote	0.81	0.90	0.99
2002-03	Major City	0.93	0.94	0.95
	Regional	1.09	1.11	1.13
	Remote	0.82	0.90	0.99
2003-04	Major City	0.92	0.93	0.94
	Regional	1.11	1.13	1.15
	Remote	0.76	0.84	0.92

a Includes both primary and revision knee replacement surgery.

Source: AIHW unpublished data.

4.3 Separations by patient funding status

Table 4.17 **Catheterisation (number separations)**

Year	*Public*	*Private insurance or self insured*	*Department of Veterans' Affairs*	*Other*
1993-94	48874	47694	4908	6964
1994-95	59304	49400	6850	5826
1995-96	65286	57614	8472	1878
1996-97	65780	60860	8192	3010
1997-98	73298	68060	10742	3160
1998-99	71740	67022	11956	1556
1999-00	67820	70778	12454	1226
2000-01	66462	73130	12828	7498
2001-02	63578	87746	12778	2310
2002-03	67066	94296	13162	2560
2003-04	72782	102582	13176	1638

Source: AIHW unpublished data.

Table 4.18 **Angiography**

Year	*Public*	*Private insurance or self insured*	*Department of Veterans' Affairs*	*Other*
1993-94	22480	20407	2235	1788
1994-95	28436	21378	3190	1708
1995-96	31730	27025	4183	889
1996-97	32113	29892	4033	1455
1997-98	35451	33223	5228	1542
1998-99	34252	32876	5854	723
1999-00	32605	34863	6145	561
2000-01	32247	36116	6357	3665
2001-02	30852	43397	6331	1121
2002-03	32644	46690	6537	1234
2003-04	35604	50736	6526	794

Source: AIHW unpublished data.

Table 4.19 **Angioplasty without stent**

Year	*Public*	*Private insurance or self insured*	*Department of Veterans' Affairs*	*Other*
1993-94	3832	4510	441	472
1994-95	5051	4488	609	342
1995-96	5887	5563	710	94
1996-97	4228	3792	506	141
1997-98	3650	3225	494	171
1998-99	2510	1915	325	34
1999-00	2130	1521	301	33
2000-01	1600	1370	262	139
2001-02	1098	1873	187	26
2002-03	965	1323	148	20
2003-04	929	1027	139	8

Source: AIHW unpublished data.

Table 4.20 **Angioplasty with stent**

Year	*Public*	*Private insurance or self insured*	*Department of Veterans' Affairs*	*Other*
1993-94	1	0	0	0
1994-95	599	558	82	42
1995-96	1570	1802	237	27
1996-97	3636	4089	441	187
1997-98	6264	6310	891	249
1998-99	7341	6729	1041	147
1999-00	8682	7576	1377	113
2000-01	9450	8160	1417	793
2001-02	9333	10493	1594	222
2002-03	10516	13136	1845	236
2003-04	12060	15423	1964	96

Source: AIHW unpublished data.

Table 4.21 **Coronary artery bypass graft**

Year	*Public*	*Private insurance or self insured*	*Department of Veterans' Affairs*	*Other*
1993-94	7087	6939	849	1296
1994-95	7477	7083	1018	1046
1995-96	8215	7757	1470	234
1996-97	8088	7529	1450	332
1997-98	8754	7153	1331	249
1998-99	8759	6863	1582	193
1999-00	8679	6854	1607	162
2000-01	8207	6437	1427	625
2001-02	7825	7110	1276	64
2002-03	7875	6755	1201	91
2003-04	7759	6575	1086	53

Source: AIHW unpublished data.

Table 4.22 **Implantable cardiac defibrillators**

Year	*Public*	*Private insurance or self insured*	*Department of Veterans' Affairs*	*Other*
1993-94	80	65	11	5
1994-95	91	77	15	4
1995-96	125	109	22	4
1996-97	185	148	22	5
1997-98	295	221	40	9
1998-99	396	240	56	6
1999-00	405	352	69	6
2000-01	407	255	68	65
2001-02	435	442	79	5
2002-03	563	734	112	15
2003-04	707	969	144	10

Source: AIHW unpublished data.

Table 4.23 **Lens insertion and or removal**

Year	*Public*	*Private insurance or self insured*	*Department of Veterans' Affairs*	*Other*
1993-94	20670	37050	6364	8979
1994-95	22786	45558	8604	6475
1995-96	27499	55238	11227	987
1996-97	28289	60318	11795	2501
1997-98	30091	65285	14394	2627
1998-99	34655	63590	17148	3014
1999-00	32728	72671	17770	1564
2000-01	34251	79499	17614	2171
2001-02	36701	85683	17687	2400
2002-03	39781	92972	18397	1527
2003-04	39794	98419	18136	679

Source: AIHW unpublished data.

4.4 Separation rates by Indigenous status

Table 4.24 Hip replacement [a]

Year	*Status*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
2001-02	Indigenous	0.21	0.28	0.34
	Non-Indigenous	1.00	1.00	1.01
2002-03	Indigenous	0.18	0.23	0.28
	Non-Indigenous	1.00	1.01	1.02
2003-04	Indigenous	0.26	0.33	0.40
	Non-Indigenous	1.00	1.01	1.02

a Includes both primary and revision hip replacement surgery.

Source: AIHW unpublished data.

Table 4.25 Knee replacement [a]

Year	*Status*	*Lower Confidence Limit*	*Age Standardised Separation Rate*	*Upper Confidence Limit*
2001-02	Indigenous	0.20	0.26	0.31
	Non-Indigenous	1.00	1.01	1.02
2002-03	Indigenous	0.14	0.18	0.22
	Non-Indigenous	1.00	1.01	1.02
2003-04	Indigenous	0.27	0.34	0.40
	Non-Indigenous	1.00	1.01	1.02

a Includes both primary and revision knee replacement surgery.

Source: AIHW unpublished data.