The Demand for Renal Replacement Therapy:

Projections for 2005–2019

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Authors

The modelling was done and the report prepared by Sue Paul and Martin Tobias.

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Disclaimer

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Objectives

The objectives of this study were to:

1. Project incident and prevalent renal replacement therapy (RRT) rates and counts from 2000-04 to 2015-19.
2. Estimate the contribution of demographic and nondemographic drivers to the trend in demand (count).

Methods

Data on incident and prevalent RRT patients, and on deaths among these patients, were extracted from the Australian and New Zealand Dialysis and Transplant Registry (ANZDATA) for the period 1965 to 2004. Data were grouped into five year age groups and five year periods, so generating ten year overlapping cohorts. Population projections used were Statistics New Zealand series 4.

Age / period / cohort regression modelling was used to project incidence. Small numbers of registrations in some age groups and periods resulted in poor model fit, so modelling had to be restricted to the 15-69 age group. Prevalence was modelled as a function of incidence and mortality, using a partial cohort component approach.

Driver share analysis was done by comparing growth in count versus rate (to distinguish changes in population from changes in risk), and changes in crude versus age standardised rates (to distinguish trends in population size from structural ageing of the population).

Results

Both incident and prevalent RRT rates and counts are projected to increase steadily to the projection horizon (2019), driven by an increasing period rather than cohort effect.

The demand for RRT (both incident and prevalent count) is projected to increase over the projection period (2005 – 2019) at an average annual percentage rate of 5% per annum (uncertainty interval 4 – 6% per annum). While this estimate relates only to the 15-69 age group, it would be reasonable to assume that the growth rate will be similar for the 70+ age group as well.

About 40% of this growth rate is estimated to result from nonmodifiable demographic forces (with increases in population size and changes in population age structure contributing about equally).

The remaining 60% reflects a combination of epidemiological trends and health service factors. Assuming that the latter (ie increases in eligibility for and acceptability of RRT) are close to saturation, perhaps half of the total increase in demand over the projection period may be attributable to epidemiological factors, chiefly the rising prevalence of type 2 diabetes, itself fuelled by the growing obesity epidemic.

Introduction

Motivation

Early in 2006 Public Health Intelligence was requested by Dr Grant Pidgeon on behalf of the Renal Advisory Board to prepare 10–15 year projections of the growth in demand for renal replacement therapy (RRT, comprising dialysis and renal transplantation).

The intention, therefore, of this report is to inform policy work within the Ministry and its advisory board. However, it is only one input to such work and provides only an overall estimate of the likely growth in demand for RRT over the next 10 years. No attempt has been made to model demand separately by ethnic group; to distinguish diabetic from non-diabetic demand; or to project need for dialysis versus transplantation.

Background

Chronic kidney disease (CKD) is believed to be widely prevalent in the New Zealand population, along with other similar populations, such as Australia (Cass et al 2006). However, only a small proportion – perhaps as low as 2% – of people with CKD go on to develop renal failure (defined as a glomerular filtration rate of < 15 ml / min / 1.73 m^2) (Cass et al 2006). Also, not everyone who develops end stage renal disease (ESRD, essentially synonymous with renal failure) is eligible for, is offered, or accepts, RRT.

Therefore, the number of new patients receiving RRT (incident RRT) is influenced by demographic drivers (changes in the size and age structure of the population) and health service variables (changes in access to, and acceptability of, RRT), as well as epidemiological factors (changes in the underlying risk of CKD, and of progression of CKD to ESRD).

Furthermore, the number of patients receiving RRT at any point in time (prevalent RRT) is also influenced by changes in survival times on RRT (itself dependent on the mix of RRT modalities, quality of care, causal spectrum of renal failure, co-morbidities and age).

The causal spectrum of ESRD has changed dramatically over the 40 or so years that RRT has been available in New Zealand. Diabetes has overtaken glomerulonephritis as the leading cause of ESRD, with hypertension remaining in third place. Yet better management of blood pressure and blood glucose in the future may preserve kidney function, despite increasing prevalence of metabolic and cardiovascular disease. Reflux nephropathy and polycystic kidney disease may also be declining in prevalence, while analgesic nephropathy has disappeared as a cause of incident ESRD (or RRT). In New Zealand, in 2002, approximately 45% of incident RRT was attributed to diabetes, 22% to glomerulonephritis, 9% to hypertension, 5% to cystic kidney disease and 19% to all other causes (ANZDATA 2006).

Objectives

The objectives of this study were to:

1. project incident and prevalent RRT rates and counts to 2015
2. estimate the contribution of different drivers to the trend in demand (count).

Methods

Data

The data used in this report were extracted from the Australia New Zealand Dialysis and Transplant Registry (ANZDATA) (www.anzdata.org.au). All patients with ESRD who receive RRT in New Zealand and Australia are registered on this database (there is no register for those who are not offered, or refuse, RRT). The registry collects a wide range of demographic and clinical data, including outcomes data.

Registrations

The number of new patients registering for RRT was requested by five-year age group, gender and single calendar year (1965–2004). Ages of patients at registration ranged from 0 to 89 years. The number of registrations at extreme ages was very low, so the ages modelled had to be restricted to the 15–69 age range so as not to compromise the quality of the regression results (see below).

Also, for the purposes of this study, we have restricted the analysis to patients who started and ended their treatment in New Zealand. The number of patients who started in Australia and ended in New Zealand (and vice versa) was very low, so exclusion of this group is unlikely to impact on the results presented in this report.

The analysis has also been pooled over all causes of RRT. Because of the small number of registrations for each individual cause (with the possible exception of diabetes) it was not possible to do a cause-specific analysis.

Prevalent cases

In addition to the number of new patients registering for RRT, the stock of patients at the end of each calendar year was also extracted. Specifically, we were provided with the number of prevalent patients on RRT by five-year age group and gender as at 31 December (1965–2004).

While the number of new registrations enables us to calculate incidence (or inflows), the stock (or prevalence) is a function of both incidence and survival, and so provides a more comprehensive measure of burden. However, for policy and planning purposes, it is necessary to estimate (and project) both incidence and prevalence.

Statistical methods

Age standardisation

For some analyses, age-specific incidence or prevalence rates have been summarised using direct age standardisation. This allows comparison over time unconfounded by changes in the age structure of the population. The reference, or standard population, used is the New Zealand 2000–2004 population.

Modelling incidence

A classical age-period-cohort (APC) model was used to analyse and project incidence separately for each gender.

The APC model is a Poisson regression model where age, period and birth cohort are used as explanatory variables to explain differences in the response variable – in this case RRT registrations.

The mean number of new RRT registrations, μ_{ap} in each age group in each period, is modelled as being the product of age, period and cohort effects. Under the assumption that the number of cases in each age group in each period is approximately Poisson with mean $R_{ap}n_{ap}$, where R_{ap} is the risk of initiating RRT in the group, and n_{ap} is the number of person-years (population) at risk, the appropriate model to fit is a generalised linear model with a log link function, with the number of person-years modelled as an offset:

$$y_{ap} \sim \text{Poisson}(\mu_{ap})$$
$$\log(\mu_{ap}) = \alpha_a + \pi_p + \gamma_c + \log(n_{ap})$$

where α_a is the age parameter in the a-th age group (a = 1, 2, ..., A), π_p is the period parameter in the p-th period (p = 1, 2, ..., P) and γ_c is the c-th cohort parameter (c = 1, 2, ..., C, where c = A + p – a and C = A + P – 1).

For this study, new RRT registrations were grouped into five-year age bands (15–19, 20–24, …, 65–69) and five-year periods (1965–1969, 1970–1974,….,2000–2004), which leads to overlapping 10-year birth cohorts.

Projected incidence (from 2005–2019) is modelled by applying the APC model to the existing data, and projecting period and cohort effects using simple linear regression on the last three periods and birth cohorts (Osmond 1985). That is:

$$\hat{\pi}_p = \beta_{0p} + \beta_{1p} p$$
$$\hat{\gamma}_c = \beta_{0c} + \beta_{1c} c,$$

for p>P and c>C.

One of the drawbacks of using APC models is their sensitivity to cells with zero counts. If a particular age group (or period) has no registered cases the regression models will return very large error values, and the results will be uninterpretable. For this reason, the age range modelled had to be restricted to 15–69 years. Even after restricting to this age range there were three cells with zero counts (all in the 50+ age range). To avoid restricting the modelled age range any further, ‘contrived cases’ were added to these cells. That is, in the three cells with zero counts, we assumed there was one person registering for RRT. Such a negligible imputation is unlikely to have a large effect on the projections, but it does provide a convenient way of maximising the range of ages modelled.

Another well-known problem with APC models is non-identifiability. Given an age group and a period, we automatically know what the associated birth cohort is. More generally, given any two of the age, period and cohort indices, the third one is determined.

This implies that different sets of effect estimates can be found that will give the same set of fitted values (and projections). A variety of approaches can be used to obtain identifiable effects (see for example, Holford (1991) and Osmond and Gardner (1982)). For the purposes of this report, 'corner-point constraints' have been used to obtain identifiable effects. That is, the first period effect is set to zero, as is the first cohort effect. In addition, stability of age effects over the projection period has been assumed.

Note, however, that the main purpose of this study is to project incidence (and prevalence), so estimation of age, period and cohort effects is of secondary importance.

Alternatives to the classical APC model

Alternatives to the classical frequentist approach include Bayesian models, which use autoregressive priors for the age, period and cohort parameters to obtain distributions of the desired parameters using Markov Chain Monte Carlo techniques (Gelman et al 1995).

A Bayesian random walk 2 model was also built for incidence. This is used for two purposes:

- to provide an estimate of uncertainty around the projected rates (the 95% Bayesian credible interval, which is similar to the frequentist 95% confidence interval, yet is easier to calculate in the context of APC models)
- to validate the frequentist incidence projections.

Only classical APC projections are included in the body of this report. The Bayesian incidence projections are provided in the Annexe.

Modelling prevalence

Prevalence is modelled as a function of incidence and age-specific mortality rates. Simply, if we know the current number of patients at the start of any period, along with the expected number of inflows (new cases) and outflows (deaths) during that period, we are then able to estimate the prevalent stock at the beginning of the following year.

Mortality projections

The ANZDATA provided historical age specific mortality rates.

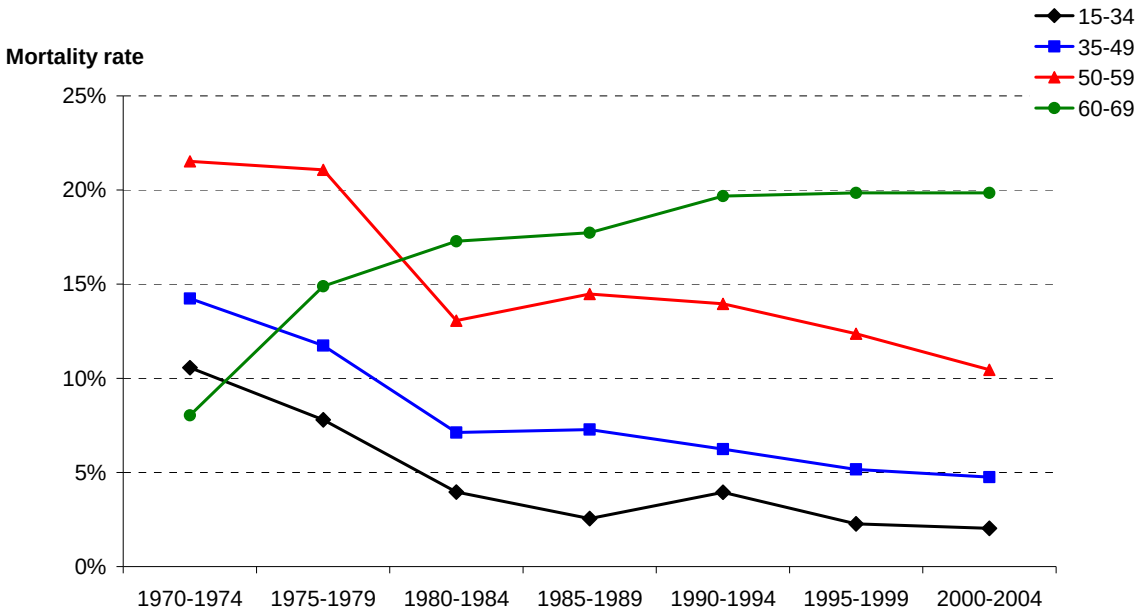
Mortality rates by age group over the 1970–1974 to 2000–2004 periods are shown in Figure 1. The data has been pooled over both modalities of RRT (transplant and dialysis). The wide age groups were chosen to ensure narrower confidence intervals around mortality rate estimates. Genders are pooled for the mortality analysis on the assumption that survival of RRT patients does not vary significantly between genders.

From Figure 1, we can see that there has been a decrease in mortality for all age groups, except the 60–69 group. In this case, mortality rates increased from 8% in 1970–1974 to 20% in 1990–1994. The mortality rate for this group has remained stable since. Given the flat mortality trend over the last 15 years, we have assumed that mortality rates for 60–69 year olds will remain stable at 20% between 2005 and 2019.

For the 15–49 age group there has been a very steep decline in mortality over the last 30 years. Mortality rates have dropped from 11% for 15–34 year olds (14% for 35–49 year olds) in 1970–1974 to 2% (5% for 35–49 year olds) in 2000–2004. If a simple linear regression (SLR) model were fit for these groups (using period as a dummy predictor), then the projected mortality rates would drop below zero due to the steepness of the fitted slope. Because the 2000–2004 mortality rates for 15–49 year olds are already so low, we have assumed that there will be no further decreases in mortality over the next 15 years.

For the 50–59 age group, the mortality rate decreased from 22% in 1970–1974 to 11% in 2000–2004. Fitting an SLR to this group's data yields projected mortality rates of 8% in 2005–2009, 6% in 2010–2014 and 5% in 2015–2019. To ensure that mortality rates do not drop too steeply, the regression results are used for the first two periods only. No mortality improvement occurs thereafter.

Figure 1: Historical mortality rates by age and five-year period



The mortality assumptions for the projection periods are summarised in Table 1. In brief, no further improvement in mortality risk is anticipated, except for a small decline in the 50–59 age group.

Table 1: Projected mortality rates 2005–2019

	2005–2009	2010–2014	2015–2019
15–34	2%	2%	2%
35–49	5%	5%	5%
50–59	8%	6%	6%
60–69	20%	20%	20%

Prevalence projections

A partial cohort component approach was used, whereby the current stock of RRT patients (2004) is simulated over the projection period (2005–2019). Each year new patients are added in (according to incidence projections). The number of deaths from the resulting pool of patients is modelled using the mortality rates in Table 1. At the end of each year, the current pool of patients is aged by one year. The ageing transition is required to ensure that age-specific incidence and mortality is applied to the correct cohorts each year.

Because the incidence projections are restricted to the 15–69 age group, the prevalence projections must, likewise, be restricted. Thus, when simulating prevalent patients, we assume that youngest patients (aged 15) will only result from newly registered cases, and exclude any inflows from the 0–14 age group. We do not anticipate this to introduce much error into the model, as the current stock of RRT patients aged 0–14 is very low – in 2004 there were 16 patients aged 0–14 from a total of 2977. Similarly, each time a patient turns 70 in the simulation, we 'delete' them from the model because we are unable to model them further due to lack of projected incidence for the 70+ age group.

The relationship between incidence, survival and prevalence is set out in more detail below. For any single-year age group a (a = 15, 16, ..., 68, 69) and single calendar year p (p = 2005, 2006, ..., 2018, 2019), let:

- $RRT_{a,p}$ = number of RRT patients aged a currently being treated in period p
- $reg_{a,p}$ = number of new registrations from age group a in period p. These are forecasted from the age period cohort model projections. New cases are calculated by multiplying the projected incidence rate by population projections for the year concerned ($reg_{ap} = \text{incidence rate}_{ap} \times \text{population at risk}_{ap}$)
- $D_{a,p}$ = number of deaths in age group a and period p
- $M_{a,p}$ = the mortality rate for age group a in period p – roughly denoting the proportion of patients aged a surviving from period p to p+1.

Then the number of prevalent RRT cases in period p+1 for age group a can be written as:

$$\mathrm{RRT}_{a,p+1} = \mathrm{reg}_{a,p+1} + \mathrm{RRT}_{a-1,p} - D_{a,p+1};$$
$$D_{a,p+1} = \left(\mathrm{reg}_{a,p+1} + \mathrm{RRT}_{a-1,p}\right) \times M_{a,p+1}.$$

Incidence

Regression results and RRT incidence projections (ages 15–69 years) are presented in this section. All rates are five-year averages, and all counts are five-year totals.

However, we first present a brief exploratory analysis of the registration data over the 1965–2004 period (for the 15-69 year age range), focusing on the most recent observed period (2000–2004).

Exploratory analysis

Figure 2 summarises the number of new patients registering for RRT by gender over the 1965–2004 period. The number of registrations has risen steadily for both genders over this period. The total number of RRT registrations has increased from 117 in 1965–1969 to 1771 in 2000–2004, an average annual percentage increase of 36% per annum over the whole observation period (although much less in recent periods).

Figure 3 graphs crude and age-standardised incidence (expressed as patients per million population or pmp) for males and females over the modelled period. The pattern mirrors the results of Figure 2. Incidence has increased steadily over the last 30 years, increasing from 7 pmp for both males and females in 1965–1969 to 86 pmp for males and 60 pmp for females in 2000–2004. The increase in incidence rate and count has been slightly steeper for males, particularly from 1985–1989 onward.

Figure 2: Number of RRT registrations by gender, 1965–2004

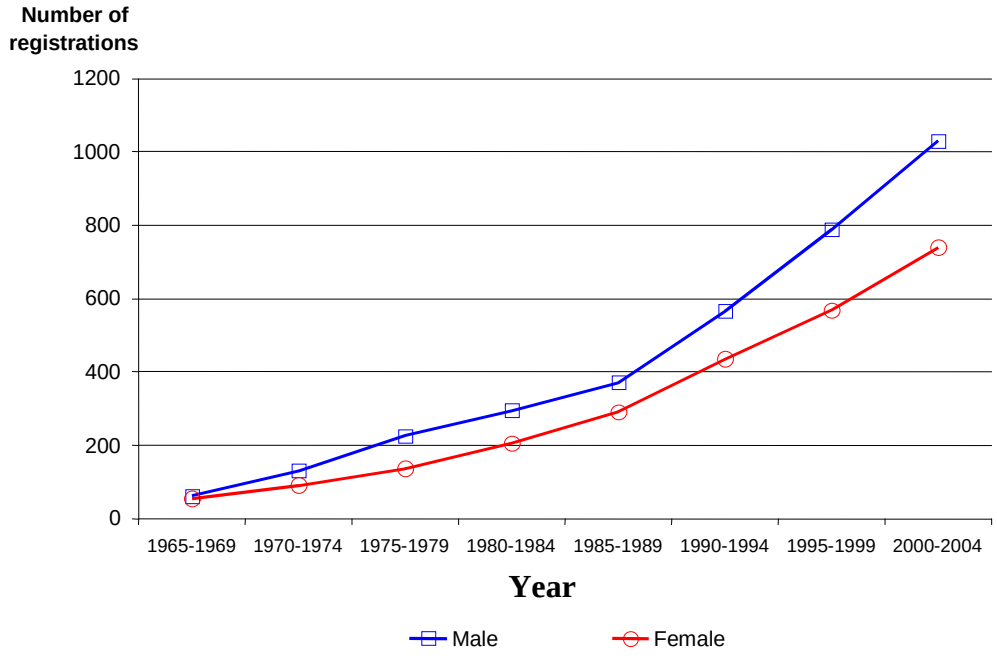


Figure 3: Crude and age standardised incidence of RRT by gender, 1965–2004

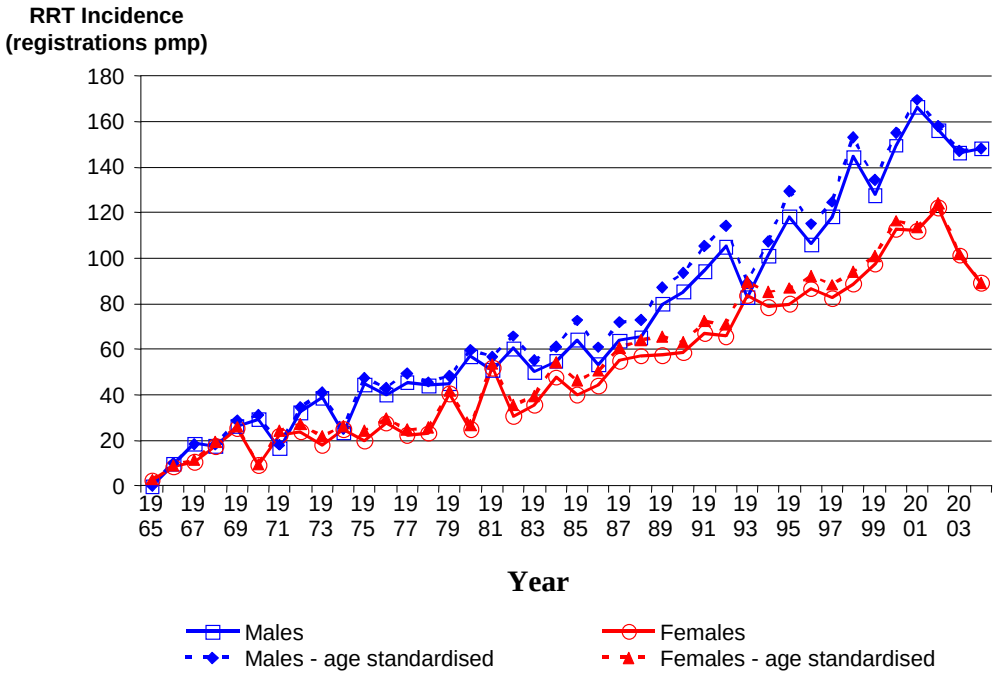
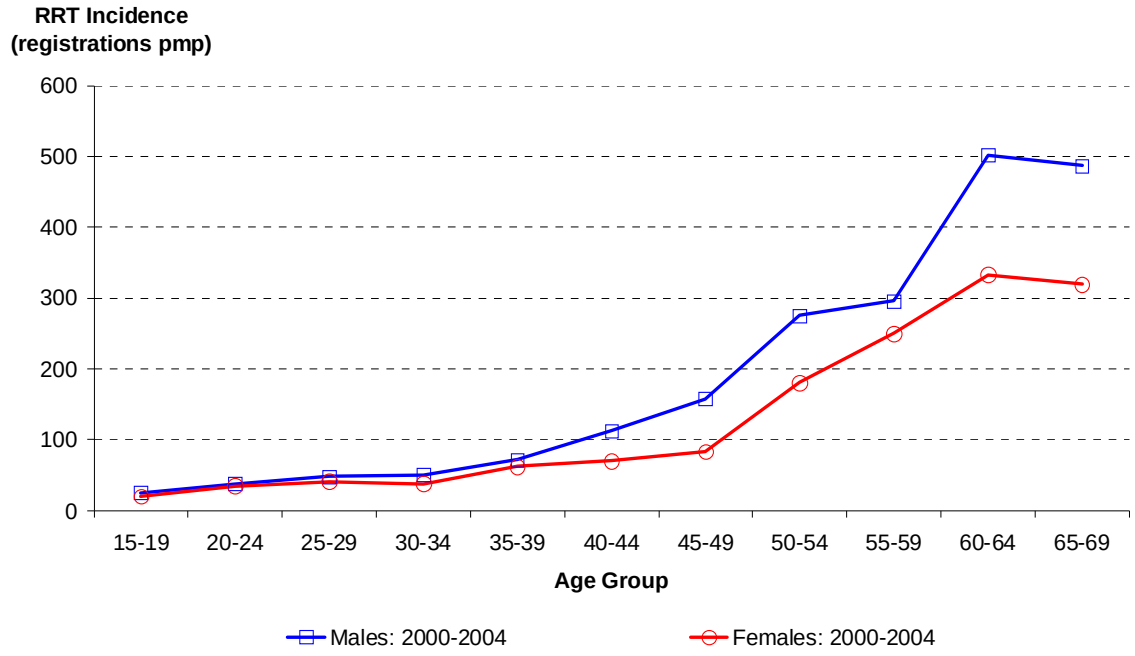


Figure 3 shows rates by single calendar year. It appears that incidence may have stabilised, or even fallen, since 2001. It is too early, however, to determine whether this represents the emergence of a new long-term trend or merely represents a transient effect.

The age distribution of RRT registrations has changed sharply over the observation period, because eligibility criteria have altered therefore broadening the age range of patients, especially at the upper end of the range. Figure 4 shows age-specific RRT incidence for the 2000–2004 period. The age-specific pattern is similar for both genders. Incidence is low in the younger ages (15–34) and increases exponentially with age thereafter. A flattening, and even decline, is noted at the oldest ages.

Figure 4: Age specific incidence of RRT, 2000–2004

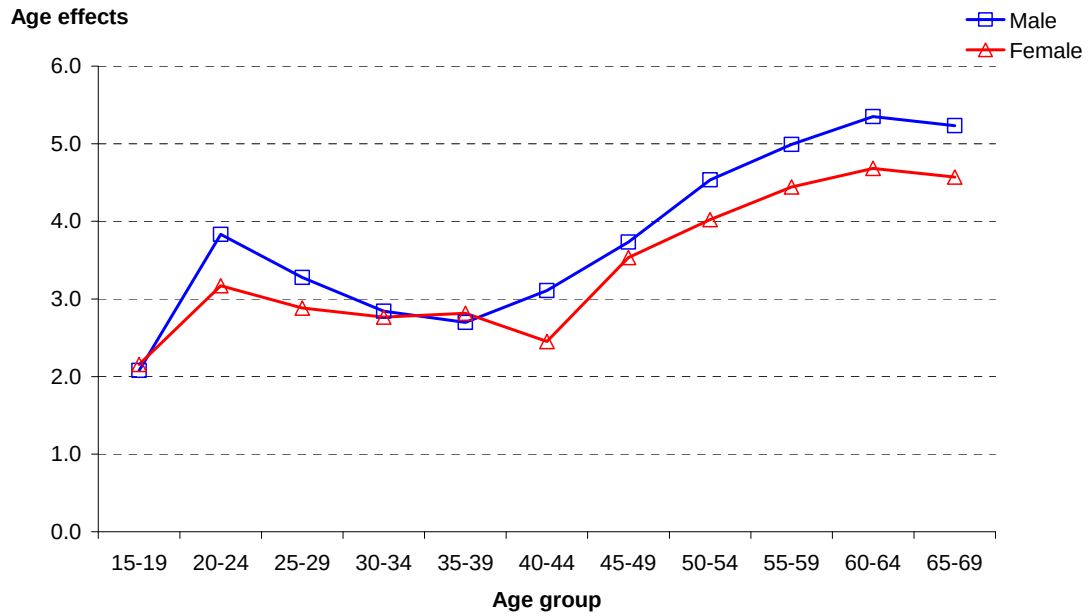


Regression modelling

Age, period and cohort effects

Age

Figure 5: Age effects



The age effects can be thought of as the age-specific incidence rates for a population with a period effect set to unity (rather than zero, because of the way the regression model is fit, ie, the period effect of the first period (1965–1969) = 1), and a cohort effect also set to unity (for the same reason, ie, the cohort effect of the earliest cohort (1895–1904) = 1).

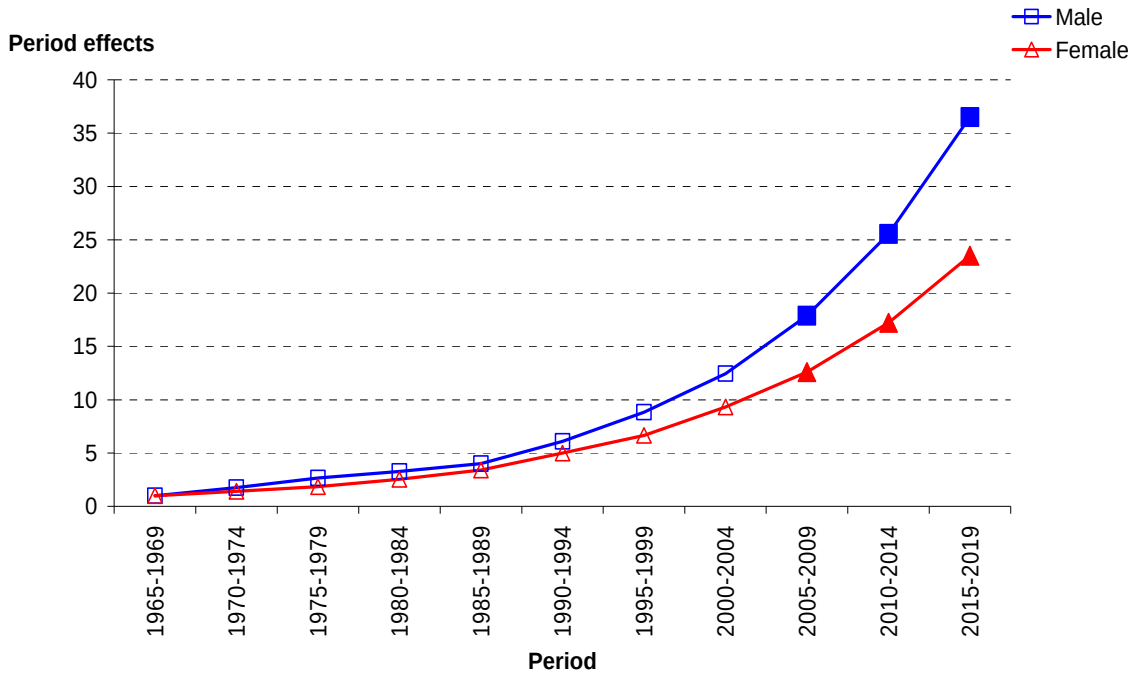
The age effects are similar to the age-specific trend seen in Figure 4, except for a small peak around the 20–24 age group and a shallow trough around the 40–44 age group. The peak at age 20–24 may reflect childhood causes of ESRD, including reflux nephropathy and Alport's disease. The plateauing at age 65–69 may reflect the impact of earlier referral and rationing biases. We have no obvious explanation for the trough at age 40–44.

Period

Period effects are summarised in Figure 6. These can be interpreted as the relative risk faced by an 'average' person with the first period as the reference. For example, Figure 6 shows that an average person in 1990–1994 was five times more likely to register for RRT than their counterpart in 1965–1969.

Figure 6 indicates that there has been a steady increase in risk for both genders over the last 40 years. For example, a male in 2000–2004 was approximately 12 times more likely than a male in 1965–1969 to register for RRT. The corresponding relative risk for females is 10.

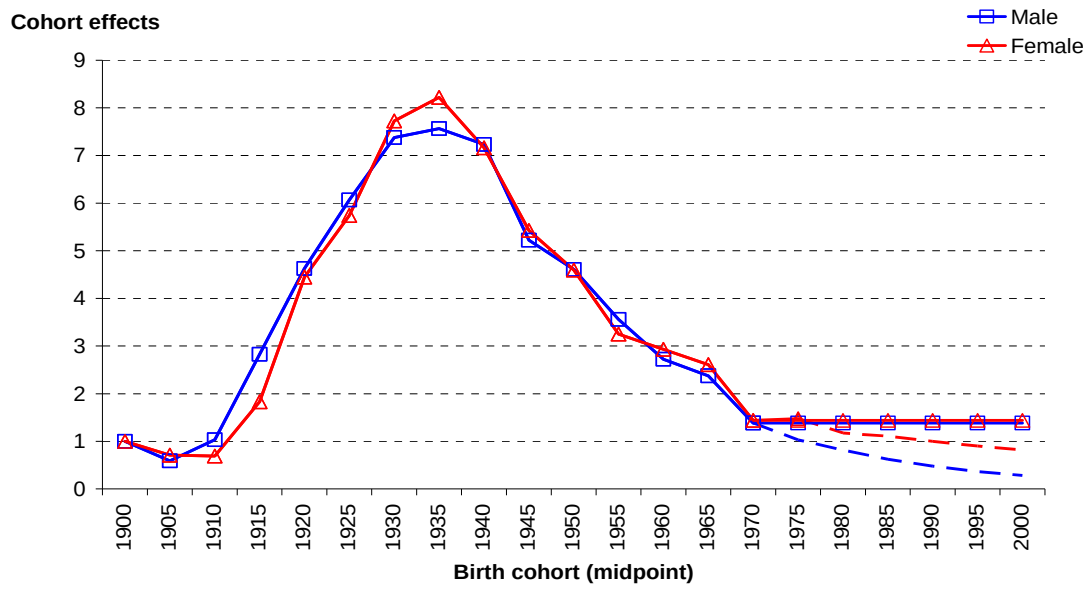
Figure 6: Period effects (last three points denote projections)



Cohort

Cohort effects are illustrated in Figure 7. Cohort effects can be interpreted as the relative risk faced by a person belonging to a particular birth cohort compared with a reference cohort. In this case, the earliest cohort (persons born between 1895 and 1904) is the reference. The effects in Figure 7 represent the relative risk for a person in any birth cohort (for any given age group) compared with those (in the same age group) born between 1895 and 1904. For example, a person born in 1945 is almost five times more likely to require RRT than someone in the reference cohort.

Figure 7: Cohort effects (last three points denote projections)



The dotted lines (from the 1970 cohort onward) on the graph indicate the actual cohort effects produced by the regression and projection models. The bold lines represent 'censored' cohort effects assumed by us. Censoring was done because the estimated effects for the most recent cohorts are less robust, being based on fewer cases (as these cohorts are still relatively young), so the assumption of stability was preferred to the further decline generated by the model. In practice, it makes little difference whether modelled or censored cohort effect estimates are used for the most recent cohorts.

Figure 7 shows that cohort effects increased progressively, from the earliest cohort to the cohort centred around 1935, and have since declined again, such that the 1970 cohort was almost back to the same cohort-specific risk level as the reference cohort.

Therefore, it appears that the increase in RRT registrations in recent years has been driven primarily by period effects. Note that, for persons born prior to 1960, the cohort-specific risk is still much higher than that of the earliest cohort. However, for the most recent cohorts (born 1970 or later) it appears that the cohort effects are not as significant as the period effects.

In interpreting these results, it is important to exercise caution. First, note that some of the increase in risk may be due to increased access to RRT in recent years. For example, RRT may not have been readily available to older patients in the 1960s. Part of the earlier cohort effects may therefore be attributable to access improvements rather than increased biological risk.

Secondly, changes in the causal mix of RRT cases may complicate the picture. For example, rising cohort effects due to diabetes may be obscured by declining cohort effects attributable to glomerulonephritis and other causes of renal failure.

Thirdly, a portion of both eligibility (access) and diabetes effects may be captured by the model as period, rather than as cohort, effects.

Finally, as indicated above, the effect estimates for recent cohorts are not robust (because they are based on fewer cases, given that these cohorts are still young) and the possibility that much of the recent diabetes epidemic effect has simply been missed cannot be excluded.

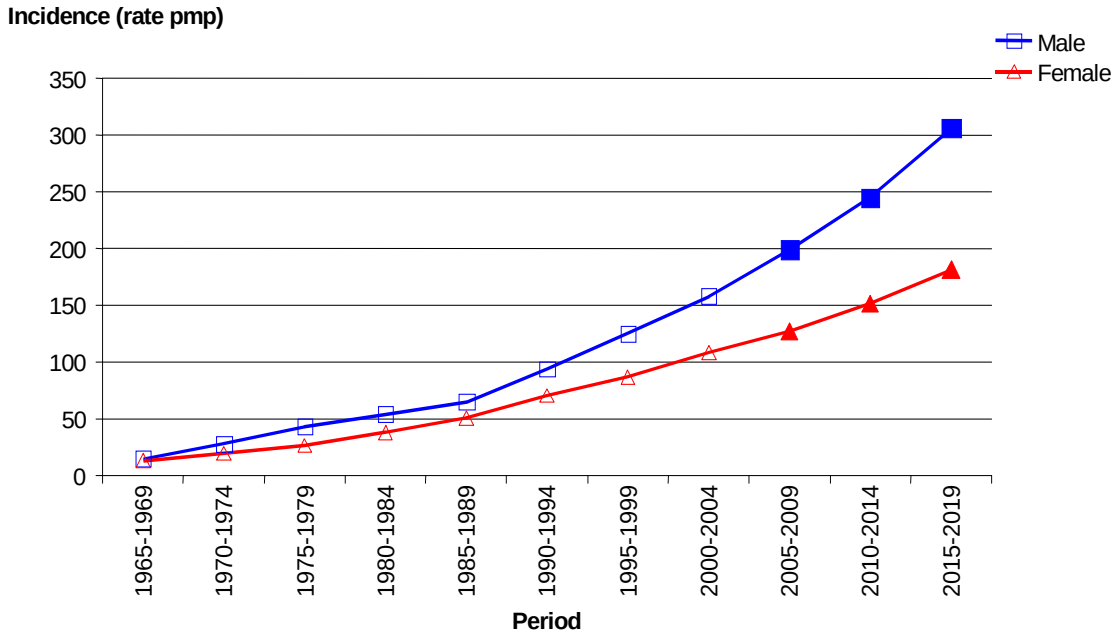
Projections

Note that all projections are restricted to the 15–69 age group. The projected number of new cases will be an underestimate of the total number of new RRT cases because they exclude children (0–14) and (more importantly) the oldest age group (70+).

Incidence rates

Figure 8 summarises fitted and projected crude RRT incidence rates. Projections (2005–2019) are denoted by bold coloured points.

Figure 8: Fitted and projected crude RRT incidence rate (15-69 years), 1965-69 to 2015-19



Age-specific incidence, crude incidence (pooled over all ages) and age standardised incidence estimates over the projection period are provided in Table 2.

Table 2: Projected age-specific, crude and age standardised RRT incidence rates

Age	Male (pmp)			Female (pmp)		
	2005–2009	**2010–2014**	**2015–2019**	**2005–2009**	**2010–2014**	**2015–2019**
15–19	51.33	73.34	104.80	39.07	53.32	72.79
20–24	94.72	135.35	193.39	57.47	78.45	107.08
25–29	80.99	115.72	165.35	52.26	71.33	97.37
30–34	70.26	100.39	143.44	50.14	68.45	93.43
35–39	66.70	95.31	136.18	51.06	69.70	95.14
40–44	131.95	109.76	156.83	80.77	60.71	82.86
45–49	181.51	226.38	188.30	130.55	158.81	119.36
50–54	288.74	315.15	393.06	164.94	202.86	246.77
55–59	411.15	454.10	495.62	257.35	248.77	305.96
60–64	499.68	629.78	695.57	320.73	370.26	357.90
65–69	676.94	698.39	880.23	412.85	427.15	493.10
Crude rates	199.14	244.97	306.49	127.57	151.94	181.91
Age-standardised rates	189.73	222.22	269.45	121.31	137.68	159.73

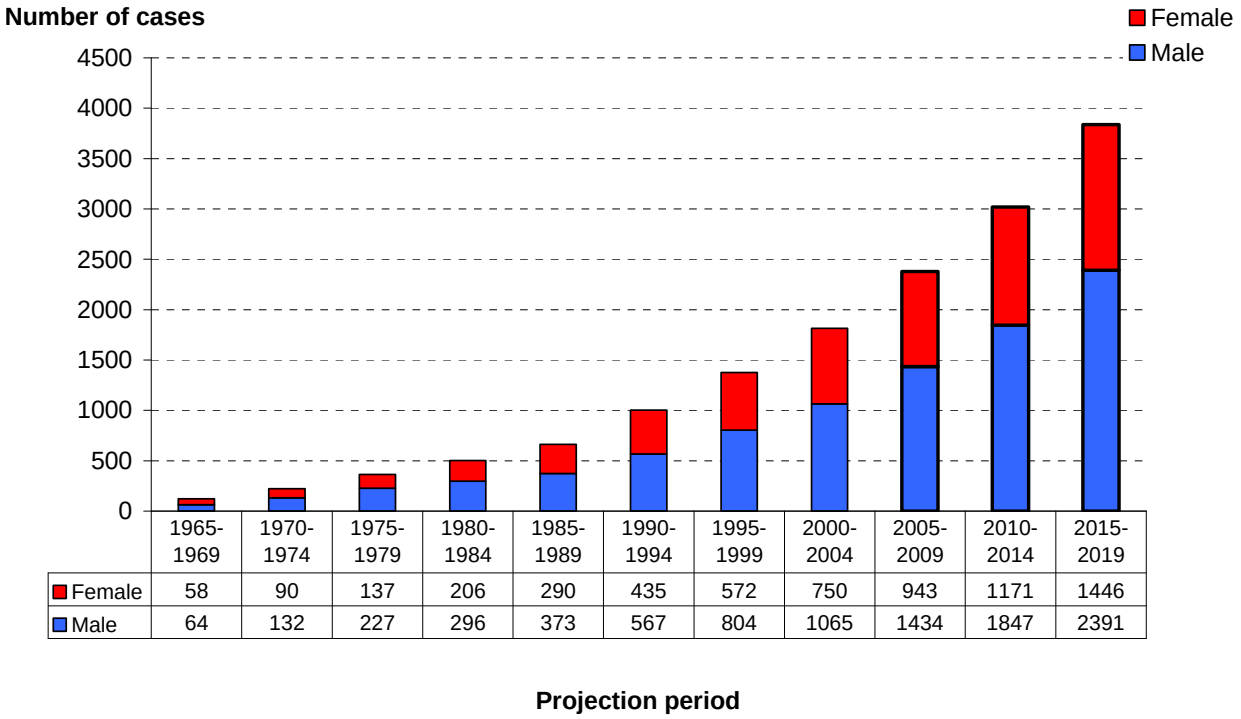
Incidence is projected to increase for both males and females over the next 15 years. The increase is markedly sharper for males than females. Male (crude) incidence increases from 200 pmp in 2005–2009 to 310 pmp in 2015–2019. Female incidence increases from 130 pmp in 2005–2009 to 180 pmp.

The corresponding age standardised rates are comparatively lower. This represents the effect of structural ageing of the population. This is discussed in more detail later in this chapter.

Incidence counts

Figure 9 shows the modelled number of new cases by gender and period. The last three bars (outlined in bold) denote projection points.

Figure 9: Observed and projected number of new registrations (15–69 years)



	1965-1969	1970-1974	1975-1979	1980-1984	1985-1989	1990-1994	1995-1999	2000-2004	2005-2009	2010-2014	2015-2019
Female	58	90	137	206	290	435	572	750	943	1171	1446
Male	64	132	227	296	373	567	804	1065	1434	1847	2391

The number of new RRT cases is projected to keep increasing over the next 10 years. The total number of cases in the 15–69 age group by five-year period will grow from 2377 in 2005–2009 to 3837 in 2015–2019, a 61% relative increase (corresponding to an annual average percentage increase of 5% per annum).

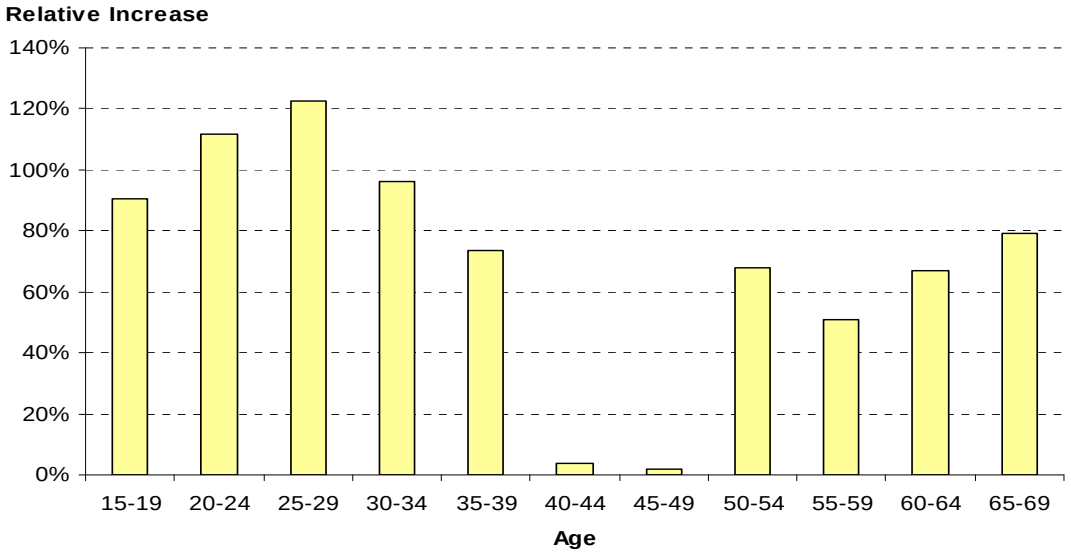
That is, the average annual count of new cases among people aged 15–69 years is projected to increase from 475 per year on average in 2005–2009 to 765 per year on average in 2015–2019. Given that the 70+ age group could not be formally modelled, a conservative 'default' option would be to assume a similar rate of growth for this age group as well.

Males will comprise the majority (over 60%) of new cases. The age-specific breakdown of new cases (within the 15–69 age range) is provided in Table 3 and Figure 10.

Table 3: Projected number of new RRT cases over five-year period by age and gender

Age	Male			Female		
	2005–2009	2010–2014	2015–2019	2005–2009	2010–2014	2015–2019
15–19	42	59	83	30	40	54
20–24	70	107	154	40	59	79
25–29	51	76	119	33	46	68
30–34	45	64	95	35	45	62
35–39	49	64	90	41	51	66
40–44	101	83	110	66	50	63
45–49	137	176	145	103	131	99
50–54	191	238	306	112	159	202
55–59	245	295	368	156	166	237
60–64	243	363	438	160	219	234
65–69	260	322	483	167	205	282
Total cases	1434	1847	2391	943	1171	1446

Figure 10: Relative increase in the total number of new RRT cases between 2005–2009 and 2015–2019



While the number of new cases increases across all age groups, the relative increase varies with age (Figure 10). For individuals aged 50 and over, the relative increase in the number of new cases ranges from 65% to 80%. Greater relative increases are projected for the 20–29 age group, while much smaller relative increases are projected for the 40–49 age group.

It is possible that these age differences reflect the peak and trough seen in the age effects curve around ages 20–24 and 40–44 respectively (see Figure 5). However, these 'kinks' may reflect historical changes in eligibility criteria and case mix, which may not persist into the future. So it is possible that future growth rates may be more

consistent across all age groups, rather than showing the age patterning generated by the model.

Driver share analysis

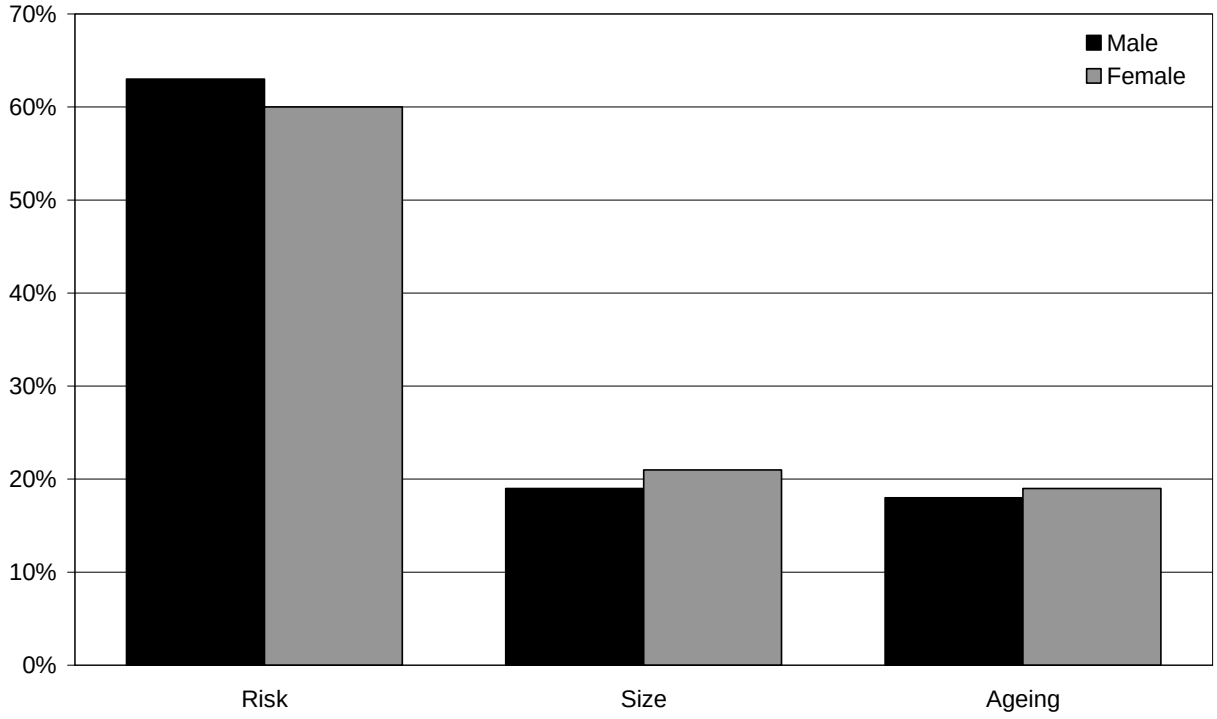
By comparing the relative increases in incidence *count* versus *rate* over the projection period, it is possible to separate the effects of trends in population size from changes in risk. Similarly, by comparing the relative increases in *crude* versus *age standardised* incidence rates it is possible to isolate the effect of population ageing.

We use this approach to separate the 'shares' contributed by the three drivers to the total growth in demand from the 2005–2009 base period to the 2015–2019 projection horizon (Figure 11). Statistics New Zealand series 4 projections were used to estimate changes in population size and age structure over the projection period. Note that the contribution from 'risk' includes both trends in epidemiological risk factors (such as diabetes) and in health service factors (such as access).

Figure 11 shows that almost two-thirds of the growth in demand is attributable to trends in 'risk'. Both increases in population size and structural ageing of the population contribute approximately one-fifth of the total growth.

That is, if growth in demand for RRT resulted solely from demographic forces (increase in population size and structural ageing of the population), it would be less than half (about 40%) of that projected. The model is silent as to what accounts for the non-demographic growth (accounting for about 60% of the total growth projected). However, further loosening of eligibility criteria probably accounts for little, since access is already reasonably similar for all age groups. Instead, most non-demographic growth probably reflects the impact of the type 2 diabetes epidemic, along with other epidemiological trends, such as increasing survival of people with heart disease. It seems reasonable to conclude that anticipated growth in type 2 diabetes prevalence may account for up to half of the total projected growth in RRT demand.

Figure 11: Driver share analysis of increase in RRT incidence, 2005-09 to 2015-19



Prevalence

RRT prevalence projections (ages 15–69 years) are presented in this section. All rates are five-year averages, and all counts are five-year totals (unless otherwise specified).

However, we first present a brief exploratory analysis of the ANZDATA prevalence data, focusing on the most recent five-year period.

Exploratory analysis

Figures 12 and 13 summarise the number of prevalent RRT cases and corresponding prevalence rate (pmp) respectively over the last 40 years.

The pattern is similar to the incidence data. That is, the number of prevalent cases (and the prevalence rate) has increased steadily since 1965. The number of prevalent RRT patients has risen from two in 1965, to approximately 2500 in 2004. Examining rate, rather than count (Figure 13), the recent slowing (since 2001) noted for incidence is seen again, especially among females.

The increase in prevalence is driven by the increasing number of new cases each year (as evidenced by the increasing incidence over time), and improvement in survival for RRT patients (see Figures 2 and 1).

Figure 12: Number of prevalent RRT cases by gender, 1965–2004

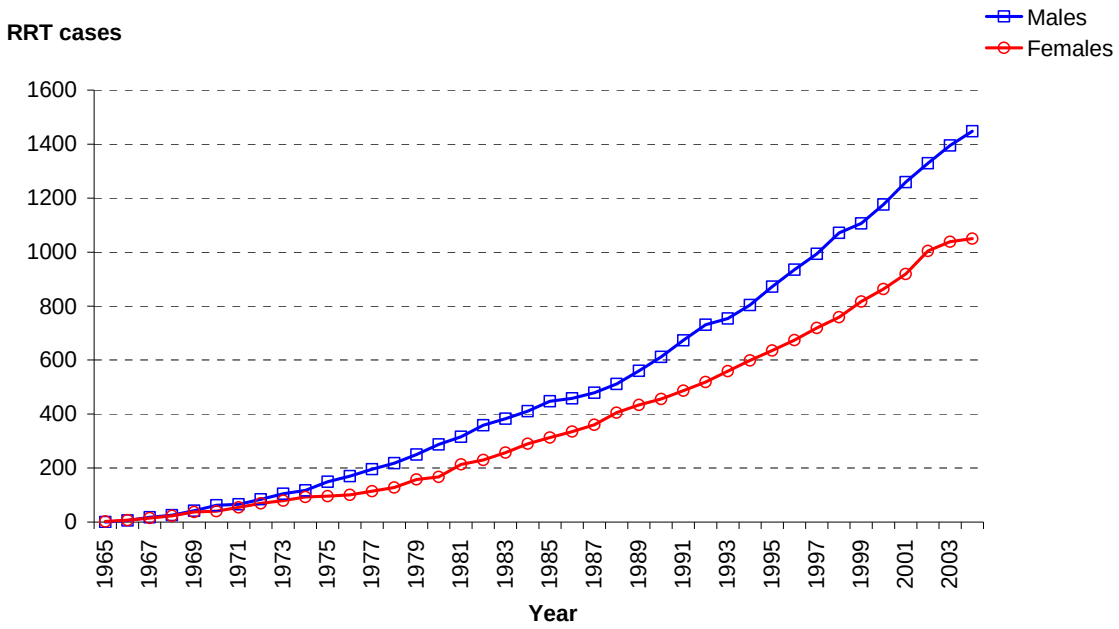
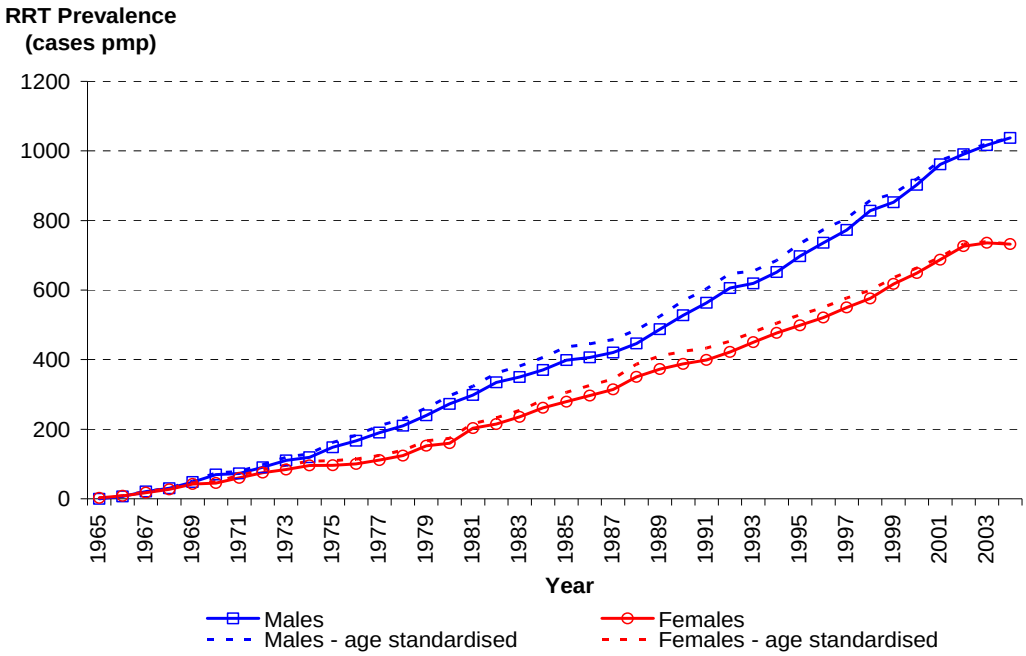
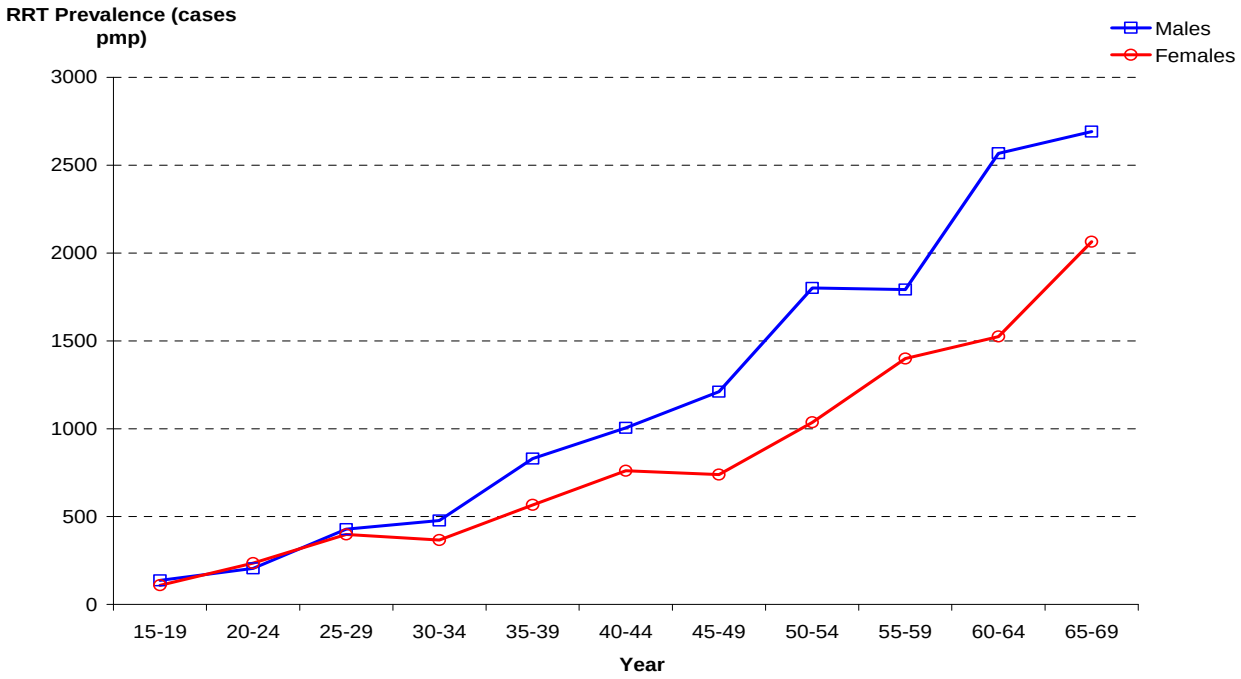


Figure 13: Crude and age standardised prevalence rates of RRT by gender, ages 15-69 years, 1965–2004



As with incidence, the age distribution of prevalent cases has shifted over time towards older ages. Age-specific prevalence rates for the most recent year (2004) are shown in Figure 14. The age-specific trends for prevalence and incidence are similar, with rates increasing more-or-less exponentially with age and then flattening slightly at older ages, at least for males.

Figure 14: Age-specific prevalence of RRT by gender, 2004



Regression modelling

Prevalence is projected to increase for both males and females over the next 15 years (Figure 15 and Table 4). Male (crude) prevalence increases from 1100 cases pmp in 2005 to 1921 pmp in 2019, a relative increase of 75%. Female prevalence rises from 767 pmp in 2005 to 1217 pmp – a smaller relative increase of 58%.

The corresponding age standardised rates are comparatively lower, again reflecting the population ageing effect.

Figure 15: Projected prevalence rates by gender, ages 15-69 years, 2005 to 2019

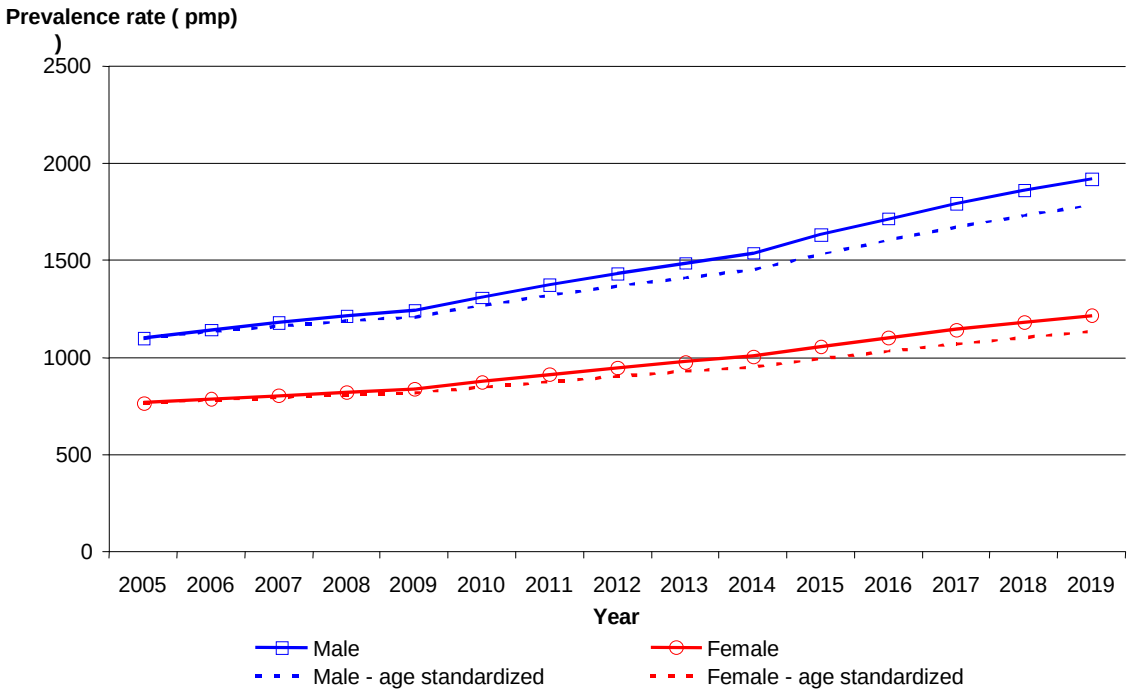


Table 4: Crude and age standardised prevalence rates (pmp) by gender and year

Year	Male	Female	Total
2005	1099.9 (1099.9)	767.4 (767.4)	931.4 (931.4)
2006	1142.7 (1133.5)	788.3 (782.4)	963.2 (955.5)
2007	1179.6 (1161.4)	805.5 (794.3)	990.2 (975.3)
2008	1213.4 (1188.2)	823.2 (806.5)	1015.9 (994.7)
2009	1242.4 (1208.8)	840.2 (817.5)	1038.9 (1010.3)
2010	1310.8 (1267.7)	876.3 (847.1)	1091 (1054.3)
2011	1374.8 (1320.6)	912.8 (876)	1141.2 (1094.9)
2012	1434.6 (1368.9)	946.5 (903.7)	1187.9 (1132.9)
2013	1487.1 (1410.5)	978.2 (928.5)	1230 (1166.1)
2014	1539.1 (1451.3)	1008.6 (951.9)	1271.2 (1198)
2015	1631.6 (1530.8)	1057.6 (994.2)	1341.7 (1258.8)
2016	1715.6 (1603.1)	1102.4 (1032.4)	1406 (1313.9)
2017	1792.9 (1670.5)	1144.9 (1070)	1465.7 (1366.4)
2018	1860.8 (1731.3)	1183.3 (1103)	1518.9 (1413.4)
2019	1920.6 (1785.6)	1216.6 (1133.4)	1565.3 (1455.7)

Note: Age standardised rates are shown in parentheses.

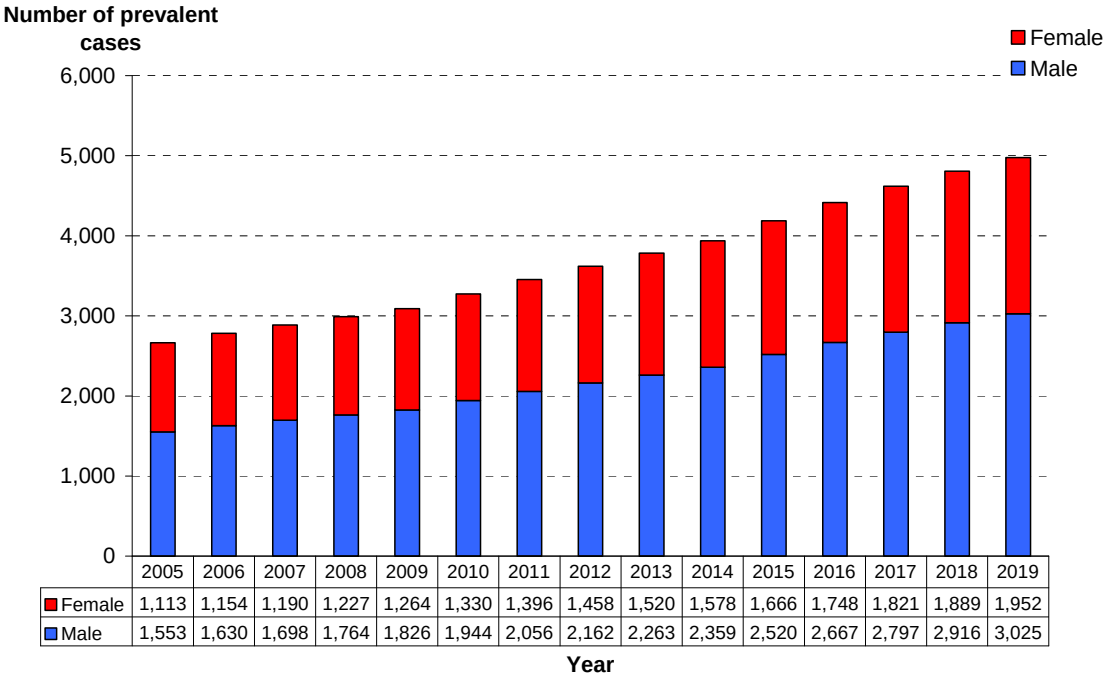
Figure 16 summarises the projected number of prevalent cases (2005–2019) by gender. Prevalent RRT cases are projected to rise from 2655 in 2005 to 4977 in 2019 – a relative increase of 87% over the period, corresponding to an average annual percentage increase of 5% per annum (similar to that found for incidence). Male cases will rise from 1553 to 3025, a relative increase of 95%. For females, the rise is less steep, with cases increasing from 1113 in 2005 to 1952 in 2019 – a relative increase of 75%.

Males will account for the majority of cases – comprising 58% of the total in 2005, rising to 61% in 2019.

The age composition of RRT cases is illustrated in Table 5. The 50+ age group makes up over half the RRT pool – accounting for 55% of all cases (within the 15–69 age

range) in 2005 and 58% in 2019. The rise in share is attributable to the increase in incidence for this group, as well as the improvement in survival rates (see Table 1).

Figure 16: Projected number of prevalent cases by gender



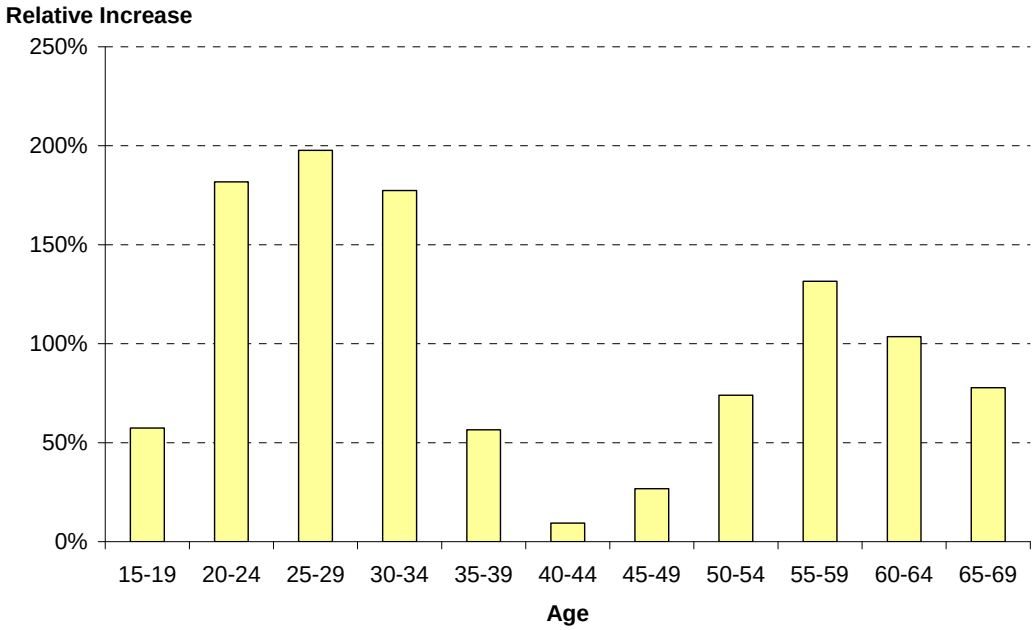
	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019
Female	1,113	1,154	1,190	1,227	1,264	1,330	1,396	1,458	1,520	1,578	1,666	1,748	1,821	1,889	1,952
Male	1,553	1,630	1,698	1,764	1,826	1,944	2,056	2,162	2,263	2,359	2,520	2,667	2,797	2,916	3,025

Table 5: Projected number of prevalent cases by five-year age group

Year	15–19	20–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60–64	65–69	Total
2005	50	83	118	137	216	299	306	380	401	347	327	2665
2006	51	100	112	159	199	311	325	391	456	337	343	2784
2007	52	109	124	166	191	305	352	402	486	355	347	2888
2008	48	124	131	171	197	291	377	417	512	375	350	2991
2009	46	128	150	174	194	285	400	431	530	404	347	3090
2010	47	145	171	188	195	278	429	465	556	448	352	3274
2011	51	155	193	189	218	261	446	501	585	491	361	3452
2012	54	165	211	205	227	254	445	542	615	521	383	3621
2013	56	170	233	219	234	259	434	578	649	544	407	3783
2014	57	175	246	243	240	259	427	610	683	564	435	3937
2015	64	189	273	271	260	268	409	652	732	586	481	4186
2016	70	205	296	301	269	293	389	677	782	613	520	4415
2017	74	217	318	327	291	306	381	680	836	642	546	4619
2018	77	227	335	358	311	317	386	671	885	674	565	4805
2019	79	235	352	381	339	327	389	660	928	707	581	4977

Relative increases between 2005 and 2019 for each age group are shown in Figure 17.

Figure 17: Relative increase in the number of prevalent cases between 2005 and 2019



The pattern here is similar to the age-specific relative increase in the number of new RRT (incident) cases over the same period (see Figure 11). Again, surprisingly large relative increases are seen for the younger age groups, and surprisingly small relative increases for the 40–49 age group. The explanation may be the same as for incidence, that is, the 'kinks' seen in the age effects curve – suggesting that such extreme variation in growth rate by age is more likely to be artefactual than real.

Driver share analysis

As with incidence, the growth in number of prevalent cases can be decomposed into the shares contributed by different demographic and non-demographic drivers.

This analysis shows (unsurprisingly) a very similar picture to that seen for incidence. That is, pooling genders and age groups, about 60% of the increase in prevalent cases from 2005–2009 to 2015–2019 is attributable to changes in 'risk'; approximately 20% is attributable to expected increase in population size (based on the Statistics New Zealand series 4 projection); and the remaining approximately 20% is attributable to anticipated structural ageing of the population (again based on the series 4 projection, ie, mid-range estimates for fertility, mortality and net migration).

Discussion

The modelling has revealed a pattern of period and cohort effects, and of differential relative growth rates for some age groups, which have no ready explanation. Further research, perhaps using more and better differentiated data, may yield further insight into these patterns.

However, the projections are largely unaffected by these considerations, especially if summed over all age groups within the 15–69 age range. Furthermore, the Bayesian and frequentist projections are reasonably close (see Annexe), suggesting that they are probably fairly robust.

The key finding of the model is that the burden (incident and prevalent count) of RRT will increase at an average rate of 5% per year for (at least) the next 10–15 years.

Although we have only been able to model a restricted age range (15–69 years), a reasonable 'default' option would be to assume a similar rate of increase for the 70+ age group as well. Provided this assumption is made, expected total incident and prevalent case numbers can readily be calculated.

How uncertain is this estimate of 5% for the average annual percentage increase? The Bayesian 95% credible interval shows that it becomes increasingly uncertain more than five years out, and the projections cannot be considered robust more than 10 years out.

Beyond stochastic uncertainty, the model may have underestimated the growth in demand for several reasons. Firstly, the most recent cohorts have only reached young ages, so there is little data associated with them; their cohort effects may therefore have been underestimated. Secondly, the assumption that the 70+ age group will follow a similar trajectory to younger age groups may be overly conservative. Finally, growth in prevalence may have been underestimated over and above any underestimation of incidence, because the assumption of little further improvement in RRT survival (reflecting change in modality distribution or better modality-specific survival) may turn out to be too conservative.

On the other hand, if access contributes substantively to the rising period effect, and increases in access are approaching saturation, then the period effect will not continue to rise as projected and we will have overestimated the growth in demand.

A reasonable conclusion would be that both incident and prevalent burdens are unlikely to grow at less than 4% per year, or much more than 6% per year, over the next decade.

How does this (overall) growth rate estimate of 5% per year compare with other countries? It can be difficult to compare studies because of different methods, different time periods and ways of presenting the model output. Perhaps the closest comparison is with a recent simulation model that predicted a 4.5–6% average annual growth rate in prevalent RRT for England over the period 2000–2010 (Roderick et al 2004). An earlier simulation for Australia (Branley et al 2000) suggested a higher growth rate, but this was for an earlier period (1995–2007). A more recent Australian simulation (Cass et al

2006) suggests growth rates between 4% and 8%, depending on the scenario modelled. Recent slowing in the observed growth rate of RRT incidence in Australia suggests that the lower estimate may be more realistic, despite projected increases in obesity and type 2 diabetes prevalence. The New Zealand data also shows plateauing or even decreasing rates from 2001 to 2004 (the latest data available at the time of writing), although this may merely represent a transient, rather than an established, trend (see Figure 5). However, it needs to be acknowledged that the recent (post 2001) slowing has little influence on the model. Should this turn out to be a true turning point, it will have been missed entirely by the model.

Assuming that our overall annual growth rate estimate of 5% is correct, we further estimate that approximately two-thirds of projected growth over the next decade reflects potentially modifiable non-demographic factors, including the rising prevalence of diabetes and improved survival of people with coronary heart disease, and possibly further broadening of eligibility criteria and acceptance of RRT. The remaining one-third represents non-modifiable demographic forces (increase in population size, and structural ageing of the population).

While this information may provide useful input into the planning of RRT services, this study has been unable to address inequalities in the growth in demand across ethnic and socioeconomic groups, an issue that also deserves attention.

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Annexe: Bayesian Incidence Projections

Methods

Incidence was also modelled under a Bayesian paradigm. The Bayesian approach is covered very briefly here. More details can be found in Berzuini and Clayton (1994), Besag et al (1995) and Knorr-Held and Rainer (2001).

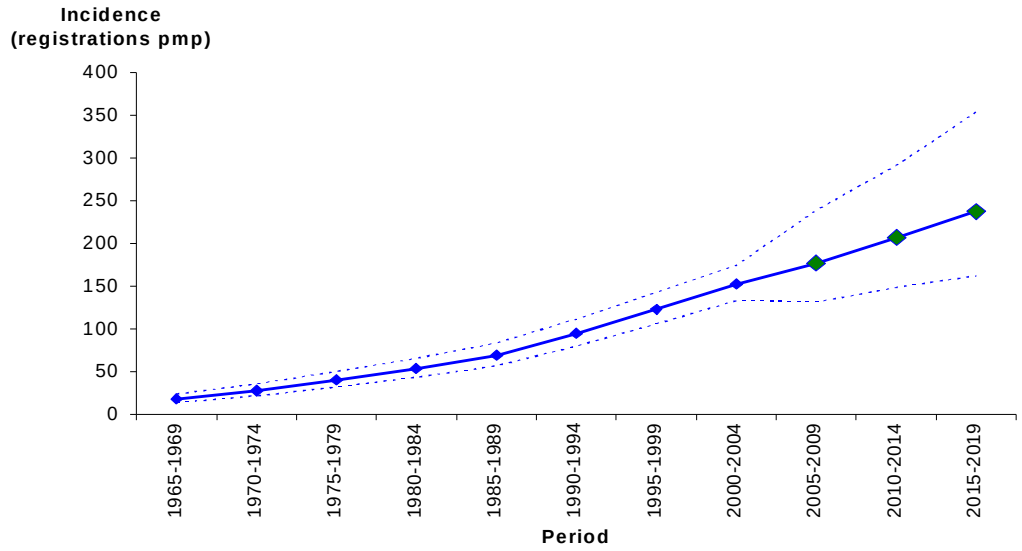
Under the Bayesian approach, prior (probability) distributions are assigned to the parameters of interest (age, period and cohort effects). Given a parametric assumption around the likelihood function (or the distribution of the data), a posterior distribution of the parameters of interest (in this case, age, period and cohort effects and the fitted and projected incidence) can be derived. Given the posterior distribution, we can make estimates of incidence by looking at the median of the relevant distribution. Similarly, by looking at the 2.5th and 97.5th percentiles of these distributions, we can gain some idea of the error around our estimates (ie, the Bayesian 95% credible interval).

BAMP[1] Random Walk 2 models (Bayesian Age Period Cohort Modelling and Prediction) models were used to project RRT incidence.

Figure 18 summarises fitted and projected RRT incidence (pmp) by gender. The dotted lines denote the 95% credible intervals or error bounds.

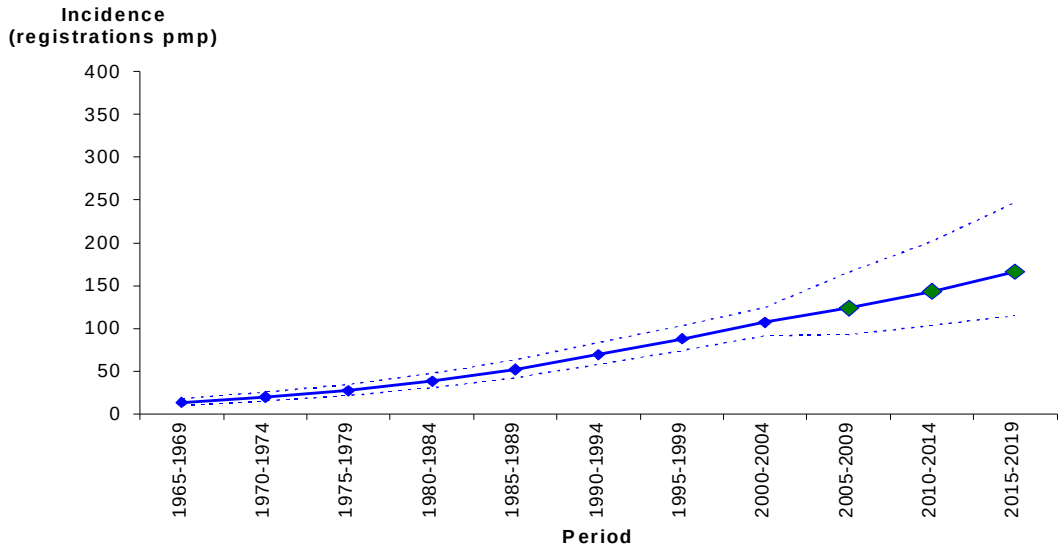
Figure 18: Fitted and projected RRT incidence under Bayesian model

Males



[1] http://www.statistik.lmu.de/sfb386/software/bamp/bamp/

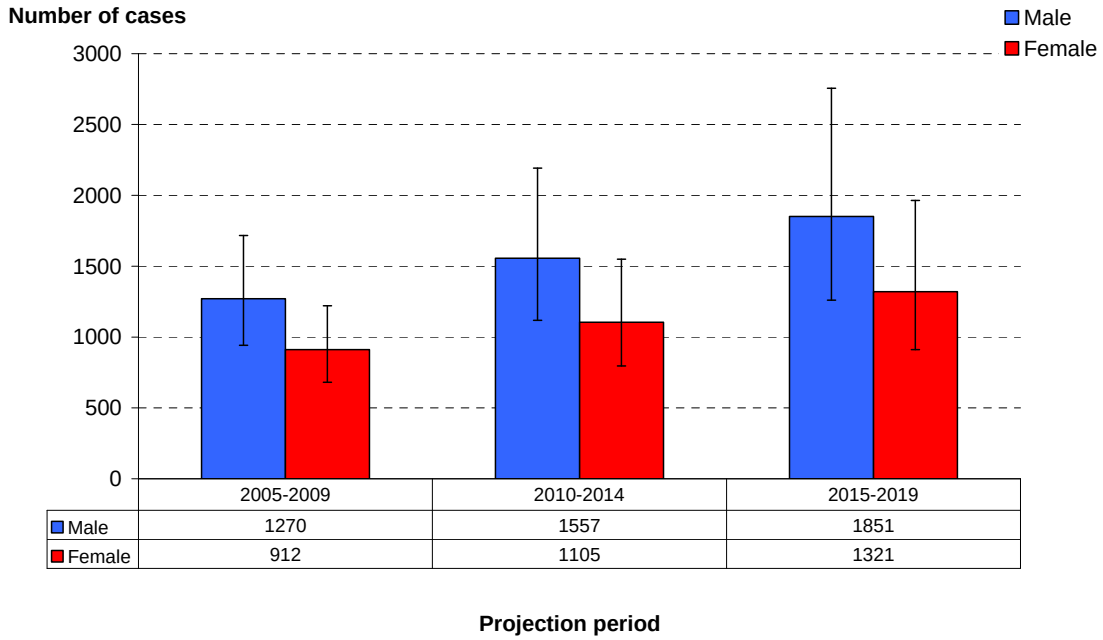
Females



Note: Projection points are denoted by points in bold. Dotted lines represent 95% credible intervals.

From Figure 19 we can see that the errors get wider as the projection points get further out. The projected number of cases by gender is shown below (Figure 19).

Figure 19: Projected number of cases under Bayesian model



Comparing classical and Bayesian results

In the following section the classical results presented in the body of this report are compared with the Bayesian results.

Figure 20 compares fitted and projected incidence rates, while Figure 21 compares fitted and projected cases under both approaches.
Bayesian and classical results are very similar over the fitted period (1965–2004). Over the projection period, however, the classical model produces higher numbers of registrations. The reason is that the classical model extrapolates a linear trend out of the period effects, and will therefore have a sharper upward trend.

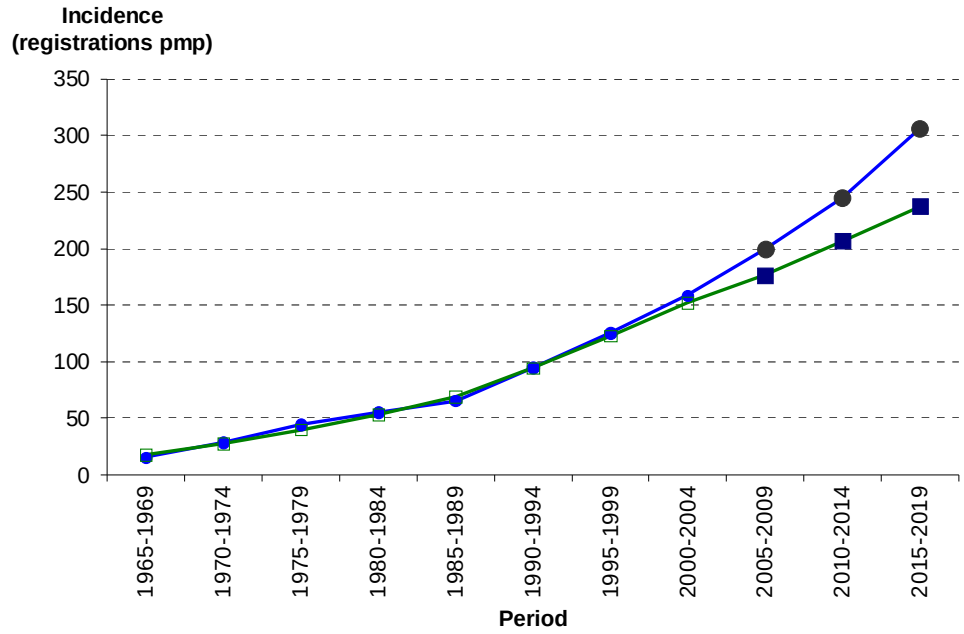
For females, however, the difference between Bayesian and classical models is not so large.

Not surprisingly, the projected number of cases under the Bayesian model is lower than the classical approach.

Overall, however, the Bayesian and frequentist results are similar, and we do not feel that it is necessary to recalculate prevalence using Bayesian incidence projections.

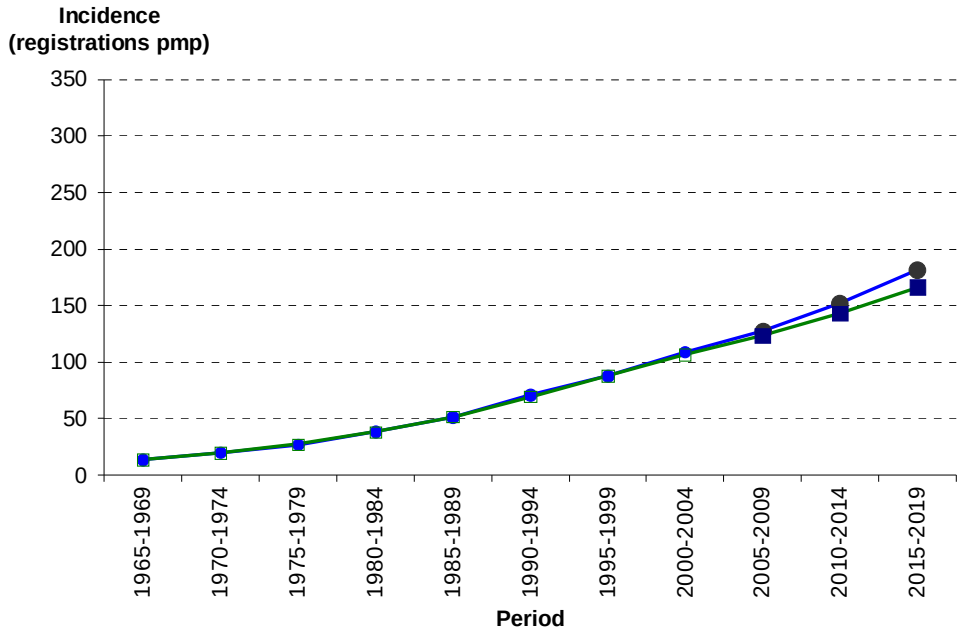
Figure 20: Comparing fitted and projected incidence under Classical and Bayesian models

Males



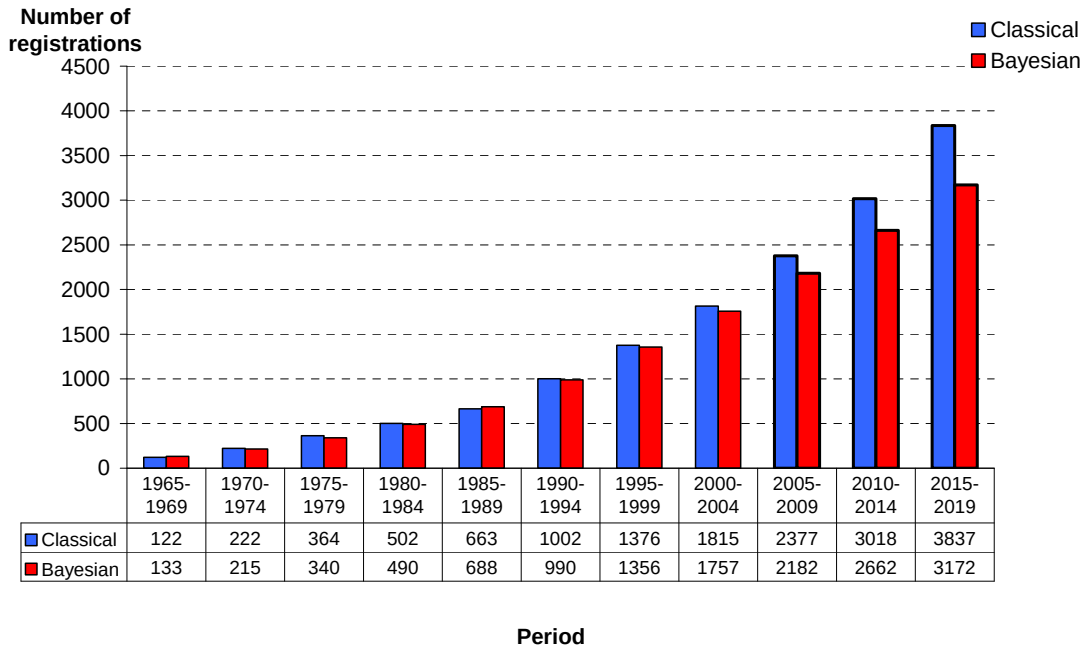
Note: Projection points are denoted by bold points.

Females



Note: Projection points are denoted by bold points.

Figure 21: Comparing the fitted and projected number of new registrations under Classical and Bayesian models



	1965-1969	1970-1974	1975-1979	1980-1984	1985-1989	1990-1994	1995-1999	2000-2004	2005-2009	2010-2014	2015-2019
Classical	122	222	364	502	663	1002	1376	1815	2377	3018	3837
Bayesian	133	215	340	490	688	990	1356	1757	2182	2662	3172

Note: Projection points are denoted by bold outlined bars.

Age-specific incidence projections are summarised in Table 6. Projected number of new registrations by age is provided in Table 7.

Table 6: Age specific incidence projections

Age	Male			Female		
	2005–2009	**2010–2014**	**2015–2019**	**2005–2009**	**2010–2014**	**2015–2019**
15–19	18.91 (14.7, 24.01)	17.19 (13.23, 21.9)	18.3 (14.01, 23.37)	10.07 (7.49, 13.16)	10.79 (8.16, 14.23)	12.92 (9.89, 16.66)
20–24	27.91 (22.21, 35.19)	30.87 (24.93, 39.33)	27.02 (21.3, 33.8)	13.11 (9.73, 17.03)	14.05 (10.95, 18.3)	17.56 (13.74, 22.99)
25–29	32.72 (26.25, 40.75)	37.57 (30.21, 46.09)	41.8 (34.18, 51.79)	18.54 (14.29, 24.07)	19.63 (15.24, 25)	21.45 (16.75, 26.9)
30–34	44.94 (36.44, 55.79)	41.06 (32.58, 49.99)	49.85 (41.04, 60.42)	22.46 (17.35, 29.29)	26.52 (20.93, 33.51)	28.65 (22.56, 36.09)
35–39	58.92 (48.35, 71.45)	54.37 (44.76, 66.7)	64.4 (52.81, 76.63)	21.98 (16.98, 28.65)	34.28 (26.95, 44.19)	38.33 (30.26, 46.91)
40–44	69.88 (57.01, 85.02)	70.11 (56.95, 85.26)	90.29 (75.65, 106.98)	18.13 (14.02, 24.11)	31.69 (25.03, 40.01)	46.6 (37.11, 58.27)
45–49	96.2 (79.7, 114.8)	114.88 (94.96, 136.31)	132.99 (113.73, 155.07)	14.31 (10.93, 19.09)	29.15 (22.7, 37.37)	47.44 (37.94, 59.23)
50–54	90.53 (73.07, 109.17)	144.65 (121.06, 171.29)	184.12 (156.9, 212.15)	8.73 (6.3, 11.94)	22.03 (16.93, 28.64)	39.51 (31.12, 49.37)
55–59	96 (79.87, 117.06)	165.44 (140.08, 196.29)	209.7 (178.89, 244.27)	5.27 (3.36, 7.81)	13.03 (9.67, 17.48)	31.57 (24.59, 40.7)
60–64	69.29 (54.96, 85.51)	128.46 (105.78, 154.21)	248.8 (215.41, 285.22)	3.36 (1.75, 5.74)	7.73 (5.04, 11.54)	18.54 (13.68, 24.84)
65–69	53.86 (41.97, 69.49)	98.9 (79.1, 121.29)	204.24 (173.31, 239.36)	2.14 (0.86, 4.59)	4.96 (2.59, 8.29)	11.5 (7.56, 17.09)
Crude	176.41 (130.74, 238.36)	206.54 (148.16, 290.83)	237.3 (161.58, 353.35)	123.42 (92.18, 165.29)	143.22 (103.34, 201.38)	166.06 (114.59, 247.14)
ASR	167.72 (124.22, 226.82)	185.16 (132.45, 261.65)	202.23 (137, 303.55)	117.53 (87.7, 157.57)	129.2 (92.9, 182.55)	143.07 (97.7, 216.1)

Note: 95% credible intervals are provided in parentheses.

Table 7: Projected number of new registrations by age and gender

Age	Males			Females		
	2005–2009	**2010–2014**	**2015–2019**	**2005–2009**	**2010–2014**	**2015–2019**
15–19	21 (12, 37)	23 (10, 52)	24 (7, 77)	18 (10, 32)	19 (8, 47)	21 (6, 73)
20–24	26 (17, 38)	31 (17, 57)	34 (14, 78)	22 (14, 33)	25 (14, 47)	27 (11, 69)
25–29	29 (21, 40)	33 (22, 52)	40 (22, 75)	26 (19, 36)	29 (18, 45)	34 (17, 65)
30–34	40 (29, 55)	42 (30, 62)	49 (32, 78)	37 (28, 51)	39 (27, 55)	43 (27, 69)
35–39	62 (46, 85)	61 (44, 86)	66 (44, 98)	55 (41, 73)	57 (41, 78)	61 (41, 90)
40–44	95 (71, 129)	97 (70, 135)	96 (67, 138)	77 (58, 103)	84 (61, 114)	87 (61, 126)
45–49	134 (100, 184)	150 (107, 209)	155 (109, 220)	98 (74, 129)	117 (86, 161)	128 (90, 180)
50–54	167 (124, 224)	209 (153, 291)	233 (165, 329)	111 (83, 148)	146 (108, 203)	176 (126, 245)
55–59	213 (159, 279)	256 (185, 351)	321 (221, 454)	137 (104, 180)	165 (121, 224)	217 (155, 305)
60–64	236 (178, 315)	314 (229, 430)	379 (264, 546)	155 (118, 205)	200 (147, 270)	239 (170, 337)
65–69	247 (185, 330)	341 (251, 468)	454 (316, 663)	176 (132, 231)	224 (164, 306)	288 (207, 406)
Total	1270 (942, 1716)	1557 (1118, 2193)	1851 (1261, 2756)	912 (681, 1221)	1105 (795, 1550)	1321 (911, 1965)

Note: 95% credible intervals provided in parentheses.