Diabetes Surveillance

Population-based estimates and projections for New Zealand, 2001–2011

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Foreword

Diabetes has been identified as a priority health objective since the release of the New Zealand Health Strategy by the Minister of Health on 14 December 2000. More recently, 'improving diabetes services' has been identified as one of 10 targets for the health sector.

Interventions to reduce the burden of diabetes – whether aimed at primary prevention of the condition, early intervention to slow the progression of the disease, or more effective treatment of micro- and macrovascular complications – will benefit from a better understanding of the descriptive epidemiology of diabetes in New Zealand. To provide such information, Public Health Intelligence (PHI) developed a multi-state life table model of diabetes in 2002. This model, based on data from the 1996/97 New Zealand Health Survey (NZHS), has proved useful for planning and funding diabetes services and formulating diabetes prevention policies.

PHI has now updated the model, using data from the 2002/03 NZHS. This report provides estimates of diabetes incidence, prevalence, mortality and survival for the early 2000s, and projections of these parameters out to 2011, based on the updated model.

It is hoped that these estimates and projections will prove useful to the policy, research and clinical communities – indeed, all those involved with planning, funding, delivering or evaluating diabetes policies and services. Comments on this report, and requests to run further scenarios on the model, are welcomed.

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Acknowledgements

The modelling was done and the report written by Martin Tobias, Li-Chia Yeh and Craig Wright (Public Health Intelligence, Ministry of Health).

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Disclaimer

Opinions expressed in this report are those of the authors and do not necessarily reflect the views of peer reviewers or the policy advice of the Ministry of Health. The Ministry of Health accepts no liability for decisions or actions based on the contents of this report.

Note on terminology

This report refers solely to diagnosed (known) type 2 diabetes, with onset between the ages 25–89 years, even when this is not explicitly stated

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Background

Surveillance of type 2 diabetes and its risk factors provides essential information needed to plan, resource and evaluate diabetes prevention and treatment services. In 2002, Public Health Intelligence built a multi-state life table model to provide estimates for the descriptive epidemiology of diagnosed type 2 diabetes in New Zealand, based on the 1996/97 New Zealand Health Survey. This model has proved useful for planning and resourcing diabetes services, and for developing diabetes prevention strategies. This report now updates our model, using data from the 2002/03 New Zealand Health Survey.

The objective of this report is to provide the policy, research and clinical communities with internally consistent estimates of diagnosed type 2 diabetes incidence, prevalence, survival and mortality for New Zealand in 2001, and projections to 2011.

Methods

Self-reported doctor-diagnosed diabetes prevalence by five-year age group (from age 25) and sex was estimated from the 2002/03 New Zealand Health Survey, a nationally representative household survey of approximately 13,000 adults.

Estimates of the relative risk (RR) of mortality conditional on diabetes were produced by record linkage using the National Health Index, and were also obtained from a systematic review of cohort studies undertaken by the Asia Pacific Cohort Studies Collaboration.

Using the above information, multi-state life tables were constructed, from which internally consistent estimates of diabetes incidence, prevalence, survival and mortality for 2001 were extracted. A cohort method was used to project these parameters to 2011, under scenarios allowing for the anticipated growth in obesity prevalence and reduction in diabetes case fatality.

Results

The model estimates that three adults per 1000 were newly diagnosed with type 2 diabetes in 2001, corresponding to approximately 7000 diagnoses per year and a life table risk of over 15%. In that year, the model estimates that 125,000 people were living with this diagnosis (an age-standardised rate of 4.5 per 100 adults) and 1500 died as a result (although only half of these deaths were so coded in the Mortality Collection).

Under the ‘most likely’ scenario (reflecting anticipated growth in the obesity epidemic and improvements in both diabetes and general health care), the model estimates over 11,000 new diagnoses, 180,000 prevalent diagnoses and 1900 deaths attributable to diagnosed disease in 2011.

Conclusions

The prevalence of diagnosed type 2 diabetes is projected to increase by approximately 45% over the decade from 2001 to 2011. About two-thirds of this growth reflects non-modifiable demographic trends (including the increasing size and ageing of the population), but the remaining one-third reflects the projected growth in obesity prevalence. Improvements needed in diabetes surveillance include the use of objective tests (such as HbA1c) in the New Zealand Health Survey and Adult Nutrition Survey, along with nested follow-up studies to enable a more robust estimation of trends in incidence, prevalence and progression of undiagnosed diabetes and of pre-diabetic states.

Introduction

Surveillance of type 2 diabetes and its risk factors provides essential information needed to plan, resource and evaluate diabetes prevention and treatment services. Yet population-based diabetes surveillance must overcome serious challenges. The measurement of incidence is difficult because the disease is initially asymptomatic, may not require pharmacologic treatment, and typically does not lead to hospital admission until complications occur. Also, diabetes-related mortality is frequently misclassified and miscoded, so routine vital statistics cannot be used to measure mortality (Chen et al 2004). Finally, the only national source of diabetes prevalence data is the New Zealand Health Survey (NZHS), which can only provide estimates of diagnosed (known) diabetes based on self-report.

Despite these data quality issues, we have previously built a multi-state life table model that provides an internally consistent account of the descriptive epidemiology of type 2 diabetes in New Zealand (Ministry of Health 2002). This model has been successfully applied to the planning, resourcing, monitoring and evaluation of diabetes services and targets at national and District Health Board levels (Ministry of Health 2003a). We can now update this model with new prevalence data from the 2002/03 NZHS and new estimates of the mortality hazard of diabetes from the Asia Pacific Cohort Studies Collaboration, and from linking routine health service databases.

The overall objectives of this study are to:

- produce internally consistent estimates of type 2 diabetes incidence, prevalence, survival and mortality for New Zealand in 2001 and projections to 2011, using our multi-state life table model
- compare these estimates with corresponding estimates derived by linking routine health service databases
- produce estimates and projections of observed (as opposed to life table) diabetes prevalence for national and sub-national populations, for monitoring and evaluation purposes, using smoothed estimates from the 2002/03 NZHS and growth parameters from the multi-state life table model.

Data Sources and Methods

Definitions

We model only diagnosed type 2 diabetes. Note that the empirical prevalence data are based on self-report of a medical diagnosis of diabetes, and typing is based on excluding disease with onset younger than 25 years of age (to roughly exclude type 1 diabetes) or during pregnancy (to roughly exclude gestational diabetes).

Input prevalence estimates

Empirical estimates of doctor-diagnosed self-reported diabetes prevalence (excluding diabetes diagnosed during pregnancy) by five-year age group (from age 25) and sex were extracted from the 2002/03 NZHS, a nationally representative household survey of approximately 13,000 adults (Ministry of Health 2005).

These estimates were smoothed by kernel smoothing (Buskirk 1998). A gaussian kernel function with a band width of nine was found to produce the optimal smooth. Both unsmoothed (empirical) and smoothed estimates are shown in Table 1.

Table 1: Unsmoothed and smoothed empirical prevalence estimates, diagnosed type 2 diabetes, 2001

Age (years)	Males		Females		Total	
	Unsmooth	Smooth	Unsmooth	Smooth	Unsmooth	Smooth
15–24	N/A	N/A	N/A	N/A	N/A	N/A
25–29	N/A	N/A	N/A	N/A	N/A	N/A
30–34	N/A	0.9	1.0	0.7	0.6	0.8
35–39	N/A	1.7	1.5	1.4	1.3	1.6
40–44	3.9	3.3	2.5	2.7	3.2	3.0
45–49	2.9	4.6	4.9	3.8	4.0	4.2
50–54	7.3	6.6	4.4	5.4	5.9	6.0
55–59	10.6	8.7	5.8	7.1	8.1	7.9
60–64	11.8	10.8	7.0	8.8	9.4	9.8
65–69	13.6	13.5	12.1	11.0	12.8	12.3
70–74	16.5	14.1	11.3	11.5	13.6	12.8
75–84	10.8	12.5	10.5	10.2	10.6	11.3
85+	N/A	8.5	N/A	7.0	7.7	7.7

Source: 2002/03 New Zealand Health Survey N/A = suppressed because count <10

The smoothed prevalence estimates were used both as an input to the multi-state life table model and as the basis for estimates (including ethnic-specific estimates) and projections for evaluation (as opposed to planning) purposes. The latter application is described in a later section of this report.

Input mortality hazard estimates

Estimates of the relative risk (RR) of mortality conditional on diabetes were produced by record linkage using the National Health Index (NHI). That is, people living with diabetes were first identified by combining records of hospital admissions for diabetes (as primary or secondary diagnosis) with records of community prescriptions for insulin, oral hypoglycaemic drugs or blood glucose self-testing equipment, thereby creating a 'virtual diabetes register'. These individuals were then linked to the mortality database by NHI to establish vital status over the following five years.

In addition, RR estimates were available from a systematic review of cohort studies from the Asia–Pacific region (including Australia and New Zealand) carried out by the Asia Pacific Cohort Studies Collaboration (APCSC 2003). While age-by-sex patterns were similar, the APCSC estimates were uniformly lower than our own record linkage estimates. Data reconciliation was achieved by taking the average of the two.* The mortality RR estimates are shown in Table 2.

Table 2: Relative risk estimates for mortality conditional on diagnosed type 2 diabetes

Age (years)	APCSC		PHI		Average	
	Males	Females	Males	Females	Males	Females
20–24	3.08	3.08	4.11	4.03	3.8	3.65
25–29	2.99	2.99	4.53	3.86	3.8	3.7
30–34	2.89	2.89	4.95	3.69	3.7	3.6
35–39	2.79	2.79	4.61	3.96	3.5	3.4
40–44	2.68	2.68	4.27	4.24	3.2	3.2
45–49	2.54	2.54	3.78	4.04	2.9	3.0
50–54	2.36	2.36	3.30	3.83	2.5	2.7
55–59	2.17	2.17	2.76	3.28	2.1	2.4
60–64	1.99	1.99	2.22	2.72	1.8	2.1
65–69	1.80	1.80	1.91	2.31	1.5	1.8
70–74	1.62	1.62	1.60	1.91	1.3	1.5
75–79	1.49	1.49	1.38	1.63	1.2	1.3
80–84	1.40	1.40	1.17	1.36	1.15	1.2
85+	1.30	1.30	1.08	1.18	1.1	1.15

Sources: Asia Pacific Cohort Studies Collaboration; PHI analysis based on New Zealand data (PHI); smoothed average

* PHI's record linkage estimate is likely to be biased high because it is based solely on people with pharmacologically treated diabetes. By contrast, the APCSC estimate is likely to be biased low because it includes people whose diabetes would have remained undiagnosed (for a time) had they not participated in the study.

Multi-state life table construction

Multi-state life tables for 2001 (male and female) were constructed by conventional demographic methods (Shryock and Siegel 1976). The mathematics of the multi-state life table and its use for descriptive epidemiology have been summarised elsewhere (Roberts and Tobias 2001).

In brief, construction of the life tables required age- and sex-specific all-cause mortality rates for the general population in 2001 (provided by Statistics New Zealand) and similar estimates for diabetes prevalence and mortality RR (derived as explained above). In addition, remission rates were assumed to be zero by definition (ie, a person with diabetes cannot transition back to the non-diseased state).

Given these inputs – prevalence, mortality RR and (zero) remission – multi-state life tables could be constructed and a wide range of internally consistent outputs extracted, including incidence, life table risk, age of onset, prevalence, survival, duration and mortality. Note that the output prevalence rates may differ from those input, because the model forces these rates to be consistent with the other life table outputs (eg, incidence and mortality rates).

The burden of diagnosed type 2 diabetes on the New Zealand population in 2001 was then estimated by applying these internally consistent output rates to the 2001 estimated usually resident mid-year population. In addition, separate 'diabetes' and 'non-diabetes' life tables were constructed to assess the impact of diabetes on life expectancy.

2011 projections

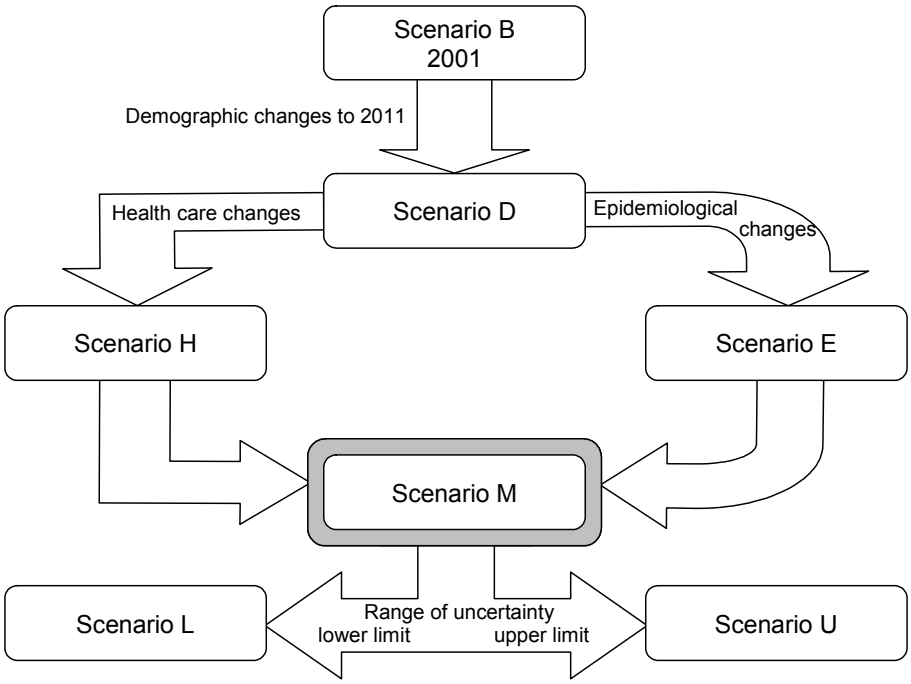
A cohort-based approach was used to project the (diagnosed type 2) diabetes burden to 2011, anchoring on census years 2001 (base), 2006 and 2011. This was done by applying diabetes incidence and mortality rates to the general and 'diabetes' populations respectively, year by year. For the former, Statistics New Zealand series 5 population projections were used (these assume 'medium' fertility, mortality and net migration).

Four main scenarios were run (see Figure 1):

- a 'demographic' scenario, in which diabetes incidence rates and mortality hazard ratios remain stable
- a 'health care' scenario, in which the excess risk of mortality associated with diabetes is progressively reduced in line with expected improvements in diabetes care (2% per annum decrease in diabetes mortality, corresponding to a 25% reduction in RR over the decade) (Ministry of Health 2002)
- an 'epidemiologic' scenario, in which incidence is increased in line with the projected growth in the obesity epidemic (3% per annum increase) (Ministry of Health 2004)
- a 'most likely' scenario, in which both of the above trends occur.

In addition, a sensitivity analysis was carried out for the most likely scenario, with the growth rates in incidence rates and mortality hazard ratios inflated and deflated by 50%.

Figure 1: Projection scenarios, 2001–2011



Key to scenarios:

B 2001 baseline (multi-state life table)

D 2001 incidence, 2001 RR, 2006 or 2011 total mortality

E Increased incidence (driven by obesity), 2001 RR, 2006 or 2011 total mortality

H 2001 incidence, decreased RR (driven by health care improvements), 2006 or 2011 total mortality

M Increased incidence as for scenario E, decreased RR as for scenario H, 2006 or 2011 total mortality

L Lowest realistic increase in incidence and decrease in RR (50% less change than scenario M), 2006 or 2011 total mortality

U Highest realistic increase in incidence and decrease in RR (50% more change than scenario M), 2006 or 2011 total mortality

Notes:

Three percent per year increase in diagnosed diabetes incidence rates (scenarios E and M), driven by corresponding growth in obesity prevalence, implies a projected increase in obesity prevalence overall from 21% to 27% of the adult population over the projection period (see Ministry of Health 2004).

Two percent per year decrease in diagnosed diabetes-attributable mortality rates (scenarios H and M), driven by improvements in early recognition and treatment for diabetes, corresponds to a 25% reduction in RR over the projection period (see Ministry of Health 2002).

Estimates for 2001

Modelled estimates for 2001

Modelled estimates for diagnosed type 2 diabetes incidence, prevalence and mortality rates and counts are presented in Tables 3, 4 and 5, respectively. In each case, only results for the age range 25–89 years are shown (ages less than 25 and over 89 were excluded because of contamination by type 1 diabetes and lack of robust prevalence and mortality hazard data, respectively).

As can be seen from Table 3, the model estimates that approximately 3 adults per 1000 were newly diagnosed with type 2 diabetes in 2001, corresponding to approximately 7000 new diagnoses per year. Table 4 shows that the prevalent pool of diagnosed type 2 diabetes was estimated at 4.5 per 100 adults (25–89 years), corresponding to a total count of approximately 125,000 people. Deaths directly attributable to type 2 diabetes (Table 5) were estimated at 0.5 per 1000 adults, or approximately 1500 per year (just under 6% of all deaths).

Table 3: Modelled diagnosed type 2 diabetes incidence rates and counts, by age (25–89 years) and sex, 2001

Age (years)	Males		Females	
	Rates	Counts	Rates	Counts
25–29	0.9	112	0.7	91
30–34	1.5	204	1.2	178
35–39	2.3	336	1.9	294
40–44	3.1	446	2.5	375
45–49	3.8	490	3.0	396
50–54	4.5	549	3.6	438
55–59	5.2	486	4.0	376
60–64	5.5	429	4.1	328
65–69	5.3	334	3.8	249
70–74	4.7	270	3.4	214
75–79	4.0	167	3.0	164
80–84	3.5	82	2.5	98
85–89	3.0	34	2.0	47
25+	3.2	3938	2.5	3250

Note: Rates are per 1000. The 25+ rate is age-standardised to the WHO world population (25–89 years) (WHO 2000).

Table 4: Modelled diagnosed type 2 diabetes prevalence rates and counts, by age (25–89 years) and sex, 2001

Age (years)	Males		Females	
	Rates	Counts	Rates	Counts
25–29	0.4	497	0.3	417
30–34	1.0	1346	0.8	1174
35–39	1.9	2774	1.6	2402
40–44	3.1	4507	2.6	3850
45–49	4.7	6038	3.8	5061
50–54	6.5	7922	5.3	6468
55–59	8.4	7872	6.9	6454
60–64	10.4	8073	8.4	6738
65–69	12.1	7596	9.8	6406
70–74	13.2	7594	10.7	6753
75–79	13.8	5784	11.3	6207
80–84	14.0	3260	11.5	4518
85–89	13.8	1559	11.4	2677
25+	5.1	64,821	4.1	59,126

Note: Rates are per 100. The 25+ rate is age-standardised to the WHO world population (25–89 years).

Table 5: Modelled diagnosed type 2 diabetes mortality rates and counts, by age (25–89 years) and sex, 2001

Age (years)	Males		Females	
	Rates	Counts	Rates	Counts
25–29	0.0	0	0.0	0
30–34	0.0	0	0.0	0
35–39	0.1	15	0.0	0
40–44	0.1	14	0.1	15
45–49	0.3	39	0.2	26
50–54	0.5	61	0.3	37
55–59	0.8	75	0.6	56
60–64	1.3	101	0.9	72
65–69	2.0	126	1.3	85
70–74	2.7	155	1.6	101
75–79	3.2	134	2.0	110
80–84	3.6	84	2.4	94
85–89	3.6	41	2.5	58
25+	0.6	844	0.4	654

Note: Rates are per 1000. The 25+ rate is age standardised to the WHO world population (25–89 years).

Table 6 summarises other statistics extractable from the multi-state life table and separate diabetes and non-diabetes life tables (these are for the whole population, all ages). These are self-explanatory other than the 'life table risk', which is the probability that an individual will be diagnosed with type 2 diabetes in his or her lifetime, taking into account risks of mortality from all causes. This statistic is derived directly from the multi-state life table, and is more valid than the conventional cumulative incidence or 'lifetime probability' statistic, which does not take mortality into account.

Table 6: Life table risk of diagnosed type 2 diabetes, median age at onset, median duration, and life expectancy impact of diagnosed type 2 diabetes at median age of onset, 2001

	Males	**Females**
Life table risk (%)	17.3	14.4
Median age of onset (years)	52.6	52.5
Median survival duration (years)	22.6	25.8
LE at median age (years):		
• diabetes	24.8	28.2
• non-diabetes	29.8	33.4
• difference (impact of diabetes)	5.0	5.2

Notes: LE = life expectancy; all estimates are for life expectancy at age 53 years, the estimated median age of diagnosis of type 2 diabetes from the model.

As can be seen from Table 6, the life table risk of being diagnosed with type 2 diabetes was estimated to be 17% for males and 14% for females in 2001. The median age of onset was 53 years in both sexes, and both males and females with diabetes lost on average five years of their life expectancy because of this condition.

Comparison of modelled with register estimates for 2001

Table 7 compares modelled with empirical estimates of diagnosed type 2 diabetes incidence, prevalence and mortality (25–89 years) for 2001. The empirical estimates for incidence and prevalence were derived by linking health service records, as described under 'Methods'.* The empirical mortality data is the average count of deaths for 2001–03 coded to diabetes, as recorded on the New Zealand Health Information Service mortality database.

* Because of low NHI (National Health Index) completion rates in 2001, the empirical incidence and prevalence data are actually back-cast from 2006, using the modelled growth rate for 2001–2006 in reverse (ie, 2006 record linkage data have been deflated by 20% to obtain the 2001 estimate – this is a conservative deflator).

Table 7: Comparison of modelled with register estimates (counts), sexes pooled, 2001

Age (years)	Incidence			Prevalence			Mortality		
	Model	Reg	Diff	Model	Reg	Diff	Model	Reg	Diff
25–29	203	280	–77	914	2732	–1818	0	1	–1
30–34	382	340	42	2520	4078	–1558	0	3	–3
35–39	630	448	182	5176	5826	–650	15	3	12
40–44	821	497	324	8357	7229	1128	29	10	19
45–49	886	607	279	11,099	8337	2762	65	17	48
50–54	987	675	312	14,390	10,248	4142	98	33	65
55–59	862	723	139	14,326	12,290	2036	131	40	91
60–64	757	669	88	14,811	12,409	2402	173	70	103
65–69	583	674	–91	14,002	13,290	712	211	84	127
70–74	484	468	16	14,347	11,686	2661	256	116	140
75–79	331	408	–77	11,991	10,719	1272	244	124	120
80–84	180	274	–94	7778	7103	675	178	125	53
85–89	81	105	–24	4236	3142	1094	99	104	–5
25–89	7188	6168	1020	123,947	109,088	14,859	1498	730	768

Note: Reg = virtual register (record linkage); Diff = difference between model estimate and record linkage estimate.

As expected, only half (48%) of diabetes-attributable deaths appear to be so recorded in the New Zealand Health Information Service Mortality Collection (730 versus 1498 deaths). PHI's record linkage process (linking hospital separations and Pharmhouse databases) appears to underestimate diabetes incidence and prevalence by only 10–15%, although the differences between model output and 'register' are greater if age- (and sex-) specific patterns are examined.

Projections for 2006 and 2011

Incidence

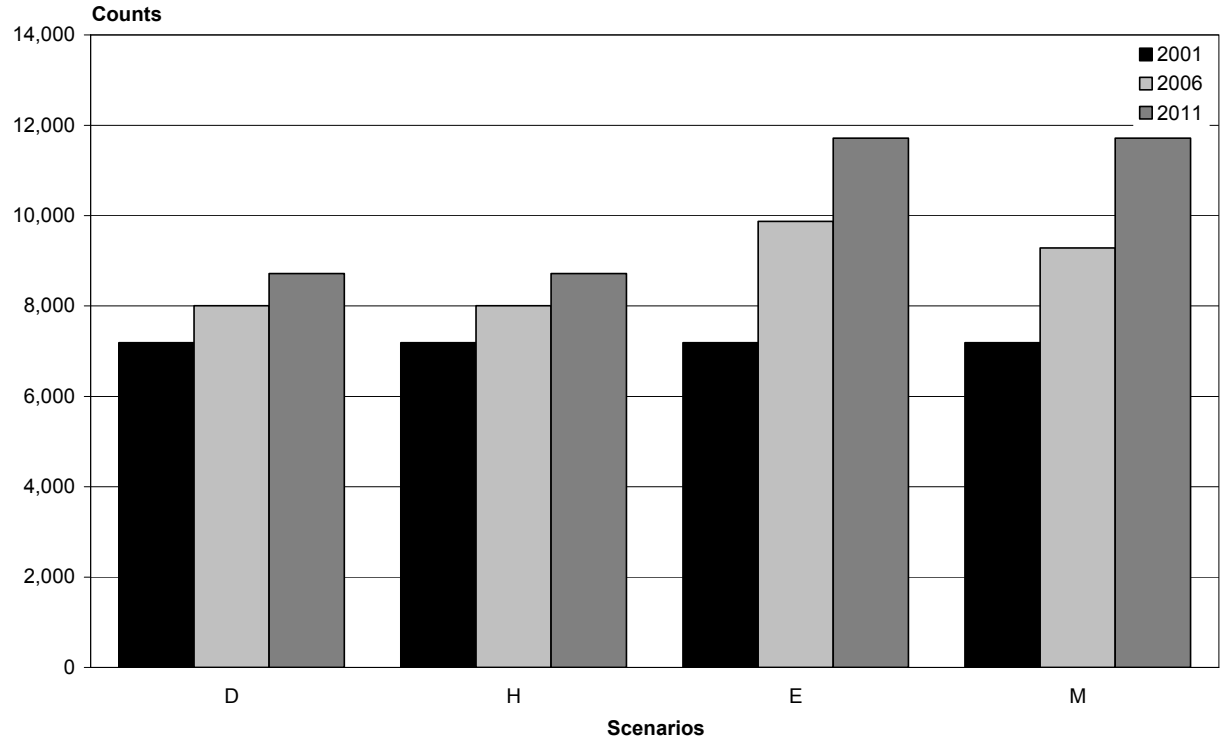
Projections of incidence counts (actually counts of diagnoses) by scenario for 2006 and 2011 are summarised below (Table 8 and Figure 2). Only total counts are shown; age-specific projections are available on request.

Table 8: Diagnosed type 2 diabetes incidence, modelled for 2001 and projected for 2006 and 2011, by sex, ages 25–89 years

Scenario	Male			Female		
	Count	Change	%	Count	Change	%
2001	3938			3250		
2006						
D	4387	449	11.4	3620	370	11.4
H	4387	449	11.4	3620	370	11.4
E	5085	1148	29.1	4197	946	29.1
M	5085	1148	29.1	4197	946	29.1
L	4726	788	20	3900	650	20
U	5467	1529	38.8	4511	1261	38.8
2011						
D	4785	847	21.5	3934	683	21
H	4785	847	21.5	3934	683	21
E	6430	2492	63.3	5286	2036	62.7
M	6430	2492	63.3	5286	2036	62.7
L	5553	1615	41.0	4565	1315	40.5
U	7430	3493	88.7	6109	2859	88

Note: For key to scenarios, see Figure 1 (page 5).

Figure 2: Modelled and projected diagnosed type 2 diabetes incidence, sexes and ages pooled, key scenarios



Note: For key to scenarios, see Figure 1 (page 5).

Based on an anticipated growth in the obesity epidemic of 3% per year, the most likely projection is for a 63% increase in new diagnoses per year over the decade, from approximately 7200 in 2001 to 11,700 in 2011, corresponding to an average annual growth rate of 5%. Note that this does not allow for any increase in the diabetes detection rate (ie, reduction in the undiagnosed to diagnosed diabetes ratio).

Prevalence

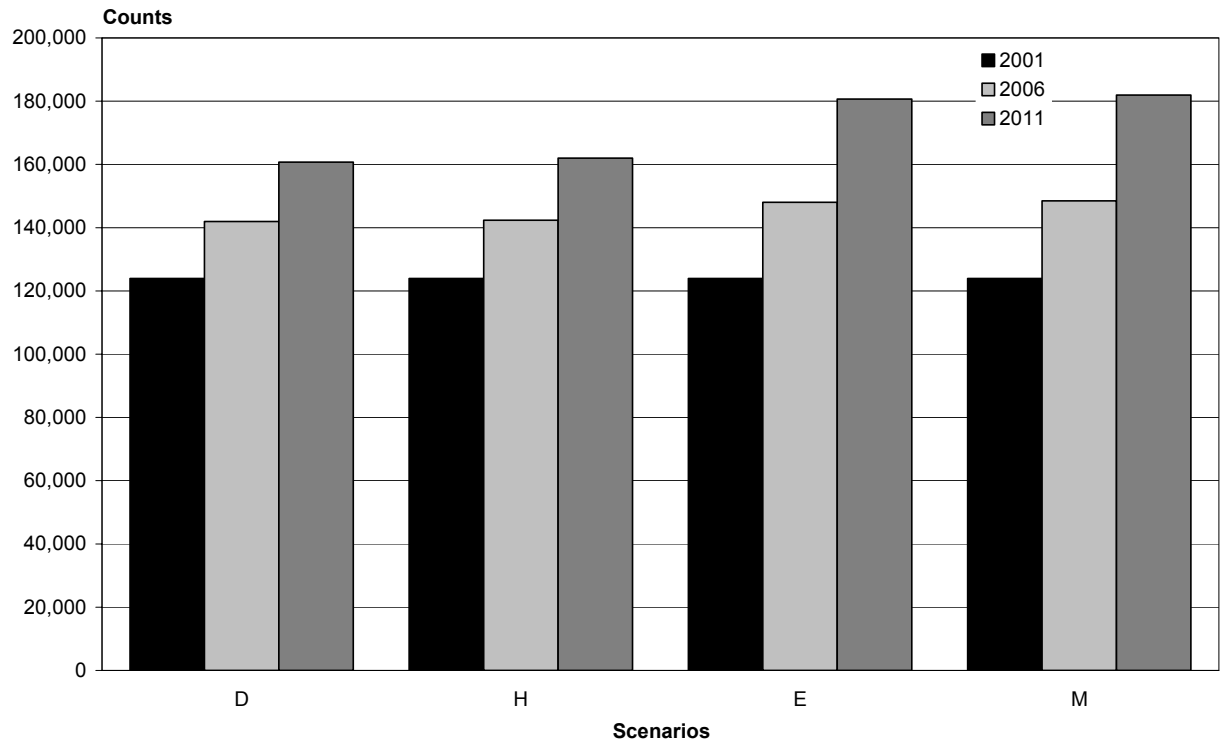
Projections of prevalence counts (of diagnosed type 2 diabetes) by scenario for 2006 and 2011 are summarised below (Table 9 and Figure 3). Only total counts are shown; age-specific projections are available on request.

Table 9: Diagnosed type 2 diabetes prevalence, modelled for 2001 and projected for 2006 and 2011, by sex, ages 25–89 years

Scenario	Male			Female		
	Count	Change	%	Count	Change	%
2001	64,819			59,125		
2006						
D	74,750	9931	15.3	67,226	8101	13.7
H	74,978	10,159	15.7	67,409	8284	14.0
E	78,059	13,240	20.4	69,986	10,861	18.4
M	78,287	13,467	20.8	70,168	11,043	18.7
L	76,470	11,650	18.0	68,656	9531	16.1
U	80,206	15,387	23.7	71,765	12,640	21.4
2011						
D	85,106	20,287	31.3	75,633	16,508	27.9
H	85,820	21,000	32.4	76,202	17,077	28.9
E	95,981	31,162	48.1	84,703	25,578	43.3
M	96,695	31,875	49.2	85,272	26,147	44.2
L	90,596	25,776	39.8	80,199	21,074	35.6
U	103,471	38,652	59.6	90,909	31,784	53.8

Note: For key to scenarios, see Figure 1 (page 5).

Figure 3: Modelled and projected diagnosed type 2 diabetes prevalence, sexes and ages pooled, key scenarios



Note: For key to scenarios, see Figure 1 (page 5).

Demographic forces acting alone are projected to increase the number of people living with a diagnosis of type 2 diabetes (ages 25–89 years) by approximately 30% over the decade from 2001 to 2011. Improvement in health care (scenario H) has a small incremental effect, while an increase in obesity prevalence (scenario E) has a much greater impact. The most likely scenario (M) is therefore a growth in prevalence of over 45%, from approximately 124,000 people in 2001 to almost 182,000 in 2011, an increase of 58,000 over the decade, corresponding to an average annual growth rate of 4%. The sensitivity analysis (scenarios L and U) indicates that this estimate is reasonably robust, with the plausible range being from 171,000 to 194,000 (ie, ±6%).

Mortality

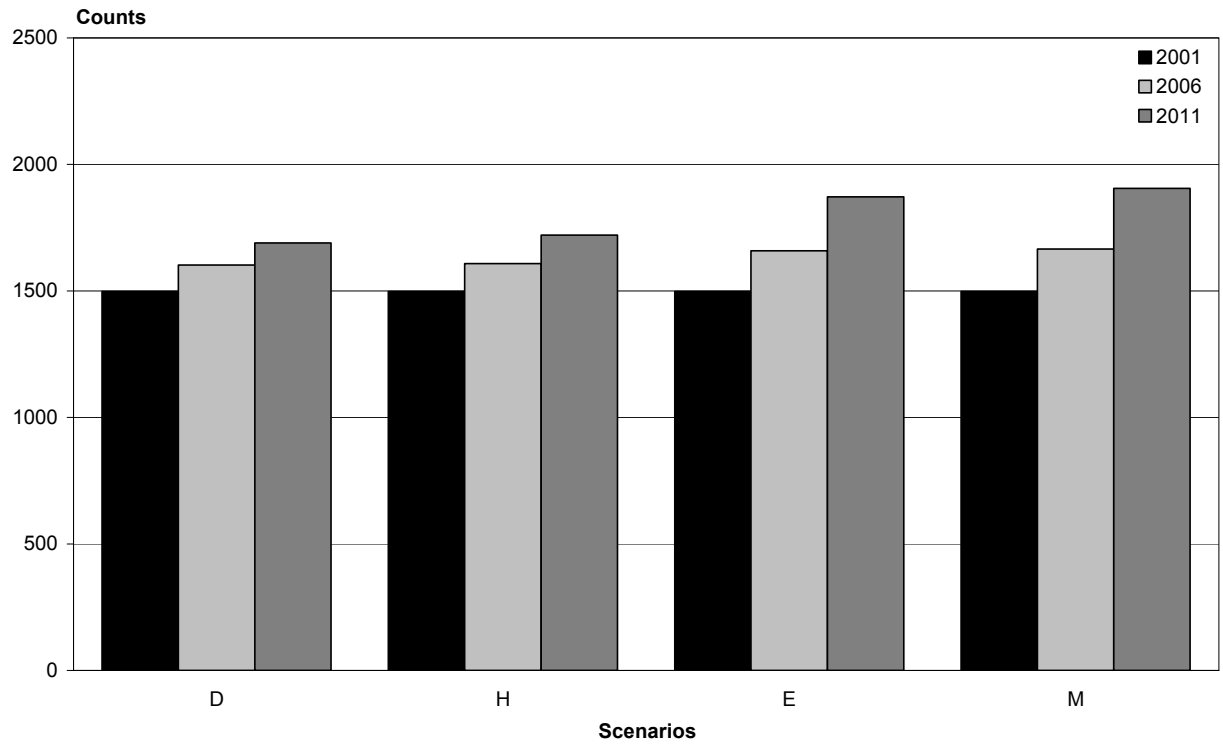
Projections of diagnosed type 2 diabetes-attributable mortality counts by scenario for 2006 and 2011 are summarised below (Table 10 and Figure 4). Only total counts are shown; age-specific projections are available on request.

Table 10: Diagnosed type 2 diabetes-attributable mortality, modelled for 2001 and projected for 2006 and 2011, by sex, ages 25–89 years

Scenario	Male			Female		
	Count	Change	%	Count	Change	%
2001	844			654		
2006						
D	906	62	7.3	696	42	6.4
H	915	71	8.4	693	39	6.0
E	940	96	11.4	719	65	9.9
M	950	106	12.6	716	62	9.5
L	977	133	15.0	734	80	12.2
U	923	79	9.4	695	41	6.3
2011						
D	964	120	14.2	726	72	11.0
H	991	147	17.4	730	76	11.6
E	1072	228	27.0	800	146	22.3
M	1101	257	30.5	804	150	22.9
L	1167	323	38.3	855	201	30.7
U	1036	192	22.7	753	99	15.1

Note: For key to scenarios, see Figure 1 (page 5).

Figure 4: Modelled and projected diagnosed type 2 diabetes-attributable mortality, sexes and ages pooled, key scenarios



Note: For key to scenarios, see Figure 1 (page 5).

Based on anticipated improvement in diabetes care leading to an annual reduction in case fatality of 2%, the model projects approximately 30% growth in the mortality burden attributable to diagnosed diabetes over the decade from 2001 to 2011 for males and 23% for females, corresponding to an increase in count from approximately 1500 to approximately 1900 deaths per year, pooling sexes (an average annual growth rate of 2.5%).

Prevalence driver analysis

As prevalence projections (a measure of future burden) are of most policy interest, Figure 5 shows the contribution of different drivers to the estimated growth in count of diagnosed type 2 diabetes (ages 25–89 years) from 2001 to 2011 under scenario M. The shares have been estimated by comparing growth under scenario M with that under scenarios D, H and E.

Figure 5: Prevalence driver contributions (%), 2001 to 2011

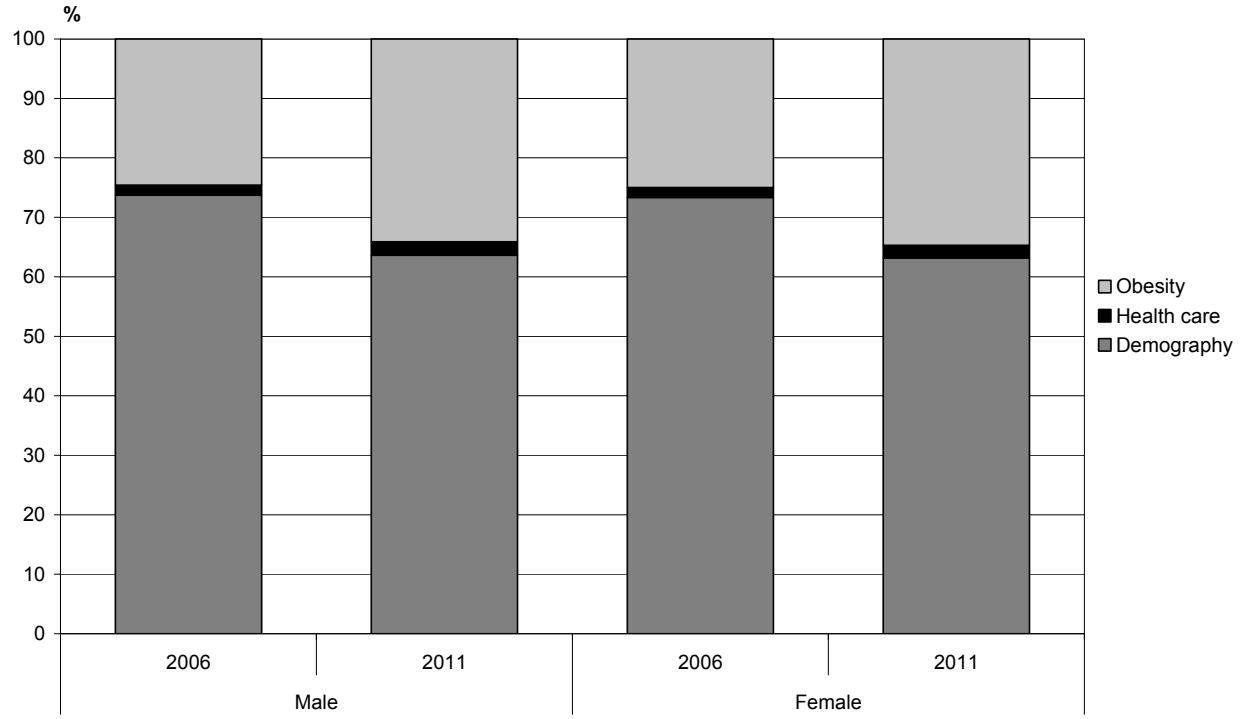


Figure 5 shows that non-modifiable demographic trends (including trends in population size and ageing) account for almost two-thirds of the projected increase in diagnosed diabetes prevalence over the decade. Anticipated progression of the obesity epidemic accounts for just over one-third of the growth in diabetes prevalence at the population level. Expected reduction in diabetes case fatality contributes very little to the expansion in prevalence – only about 2%.

Applications to Policy and Planning

Key findings

The model estimates that three adults per 1000 were newly diagnosed with type 2 diabetes in 2001, corresponding to approximately 7000 diagnoses per year and a life table risk of over 15%. In that year, the model estimates that 125,000 people were living with this diagnosis (an age-standardised rate of 4.5 per 100 adults) and 1500 died as a result (although only half of these deaths were so coded in the Mortality Collection). The true burden of insulin resistance (including diagnosed and undiagnosed diabetic and pre-diabetic states) is likely to have been substantially higher than these estimates (for example, the new diagnosis rate of 3 per 1000 adults may reflect a 'true' underlying incidence rate of up to 6 per 1000, and so on).

Under the 'most likely' scenario (reflecting anticipated growth in the obesity epidemic along with improvements in both diabetes and general health care), the model estimates over 11,000 new diagnoses, 180,000 prevalent diagnoses and 1900 deaths attributable to diagnosed disease in 2011. These estimates correspond to average annual percentage changes (growth rates) over the decade of approximately 5.0%, 4.0% and 2.5% per year for incidence, prevalence and mortality of diagnosed diabetes, respectively. Note that these estimates assume a stable ratio of diagnosed to undiagnosed diabetes (ie, no increase in the diabetes detection rate), and so should be considered conservative.

Comparison with other studies

The modelled incidence rate estimates for New Zealand in 2001 are lower than corresponding estimates for the US (Geiss et al 2006), Canada (Lipscombe and Hux 2007) and Australia (International Diabetes Institute 2006). This may reflect a higher diabetes detection rate (a lower ratio of undiagnosed to diagnosed diabetes) in these countries than in New Zealand, the use of different diagnostic criteria, or inclusion of undiagnosed diabetes in the studies. Diabetes prevalence and mortality rate estimates are correspondingly higher in the comparison countries, although the differences are not as great as for incidence.

Of perhaps more interest is a comparison of the outputs of the New Zealand 1996 (Ministry of Health 2002) and 2001 diabetes models, even though these are not strictly comparable because of differences in the mortality hazard ratio input estimates between the two models, confining us to a semi-quantitative comparison at best. Over the five years from 1996 to 2001, the models indicate substantial growth in diagnosed type 2 diabetes incidence and even greater growth in prevalence, while the mortality risk appears to have declined such that the mortality burden remains more or less stable. The growth in incidence and prevalence from 1996 to 2001 is greater than that projected for the next five years (2001–2006), and may reflect increased detection (ie, some reduction in the undiagnosed to diagnosed ratio) as well as possibly some underestimation of the 1996 values by the earlier model. By contrast, the mortality trend suggests that earlier intervention and improved treatment for people with diabetes are having a measurable effect. The greater relative increase in prevalence than incidence is consistent with the estimated mortality trend (from 1996 to 2001).

Limitations

The multi-state life table model has the advantage that output incidence, prevalence and mortality estimates are internally consistent. However, this model also has several limitations, which should be borne in mind when applying the model for planning or other purposes.

Firstly, the model is restricted by its input data to diagnosed diabetes, and the age restriction provides only a rough approximation to type 2 disease. Estimation of the total social burden of insulin resistance (including undiagnosed diabetes and pre-diabetic states) can only be made exogenously to the model.

Secondly, there were insufficient input data (prevalence rates and mortality hazard ratios by five-year age group) to build separate models for different ethnic or socioeconomic groups or regions, so the model is unable to examine inequalities in diabetes risks or burdens between subgroups.

Thirdly, the model is deterministic and so does not directly output confidence intervals for either the 2001 estimates or the 2006 and 2011 projections. However, sensitivity analysis can be used to quantify data, if not model uncertainty, as was done for the 'most likely' projection scenario, with reasonable results.

Finally, a multi-state life table is an equilibrium model (Roberts and Tobias 2001). Thus the model outputs a 'synthetic' prevalence estimate for 2001 generated by assigning 2001 incidence and mortality estimates to all previous years. This creates prevalence estimates that are free of cohort effects – and well suited to future scenario planning for that reason. However, the synthetic prevalence may differ from the observed prevalence (which incorporates past cohort effects), making the former less suitable for evaluation and performance monitoring purposes (see 'Applications to Evaluation and Performance Assessment').

Planning applications: running scenarios (simulations)

The model is potentially useful for health planners, not because it can accurately predict future absolute burdens (which no model can claim), but because of the insights it provides into the drivers of the type 2 diabetes epidemic. Thus we estimate that almost two-thirds of the growth in the number of people living with a diagnosis of diabetes from 2001 to 2011 will result from non-modifiable demographic trends. Improvement in health care, converting diabetes from a more to a less fatal disease, is estimated to contribute only about 2% to this growth. Increasing obesity prevalence – the major modifiable cause – is estimated to contribute just over one-third of the growth in diagnosed diabetes prevalence anticipated under the 'most likely' scenario (note that this assumes no increase in the diabetes detection rate).

The model can be used to simulate the impact (effectiveness) of any proposed strategy or intervention intended to control obesity, improve nutrition or promote physical activity, such as those being funded by the Ministry of Health under the Healthy Eating – Healthy Action Strategy (Ministry of Health 2003b). For example, if an intervention to reformulate certain manufactured foods and improve nutritional labelling results in a 1 percentage point reduction in obesity prevalence by 2011 compared to the counterfactual (ie, overall obesity prevalence reaches 26% rather than the 27% otherwise expected), the model indicates that approximately 3000 people will not be diagnosed with type 2 diabetes by that year who otherwise would have been (ie, the prevalence count increases by 55,000 under this counterfactual or 'intervention' scenario, rather than 58,000 under the 'business as usual' scenario).

This example illustrates how the model may be applied for planning purposes, such as policy and programme option appraisal, cost-effectiveness analysis, target setting and resource allocation. However, for reasons cited above, care should be exercised when applying the model for evaluation or performance monitoring (eg, of District Health Boards). Instead, such objectives are better served by using empirical estimates (eg, of diagnosed diabetes prevalence), adjusted as necessary to remove 'noise' (eg, by smoothing), confounding and other biases (Murray 2007). The next section of this report provides such smoothed estimates for diagnosed diabetes prevalence, by major ethnic group, derived from the 2002/03 NZHS.

Applications to Evaluation and Performance Assessment

The life table or synthetic prevalence estimates output by the multi-state life table model represent 'predicted' statistics (Murray 2007). As such, these estimates are useful for planning (eg, 'what if' scenarios) but not for evaluation. For assessing District Health Board performance or similar evaluative applications, smoothed empirical estimates of prevalence (so-called 'adjusted' statistics) are more appropriate.

Empirical estimates of diagnosed type 2 diabetes prevalence, 2001

Table 11 provides our best empirical estimates of diagnosed diabetes prevalence (in 2001) by age, sex and ethnicity, derived by kernel smoothing of the empirical data from the 2002/03 NZHS. The ethnic estimates in particular should be used with caution because of the thin data on which they are based.

Table 11: Smoothed empirical estimates of diagnosed diabetes prevalence rates (per 100), ages 30–89 years, by sex and ethnicity, 2001

Age (years)	Males	Females	Total
Total population			
30–34	0.9	0.7	0.8
35–39	1.7	1.4	1.6
40–44	3.3	2.7	3.0
45–49	4.6	3.8	4.2
50–54	6.6	5.4	6.0
55–59	8.7	7.1	7.9
60–64	10.8	8.8	9.8
65–69	13.5	11.0	12.3
70–74	14.1	11.5	12.8
75–84	12.5	10.2	11.3
85+	8.5	7.0	7.7
European			
30–34	0.7	0.5	0.6
35–39	1.3	0.9	1.1
40–44	2.5	2.0	2.3
45–49	3.1	2.6	2.8
50–54	4.2	3.4	3.8
55–59	6.9	5.7	6.3
60–64	9.1	7.4	8.3
65–69	11.2	9.1	10.1
70–74	12.4	10.1	11.3
75–84	11.4	9.3	10.3
85+	8.5	7.0	7.7

Age (years)	Males	Females	Total
Māori			
30–34	1.8	1.3	1.5
35–39	3.6	2.5	3.0
40–44	6.9	4.9	5.8
45–49	9.8	6.9	8.2
50–54	13.8	9.7	11.6
55–59	18.4	12.9	15.5
60–64	22.8	16.0	19.1
65–69	28.5	20.0	23.9
70–74	29.8	20.9	25.0
75–84	26.3	18.5	22.1
85+	18.0	12.6	15.1
Pacific			
30–34	1.4	2.1	1.8
35–39	2.8	4.2	3.5
40–44	5.3	8.1	6.8
45–49	7.5	11.4	9.6
50–54	10.6	16.1	13.5
55–59	14.1	21.4	18.0
60–64	17.5	26.5	22.3
65–69	21.9	33.1	27.8
70–74	22.9	34.6	29.1
75–84	20.2	30.6	25.7
85+	13.8	20.9	17.5
Asian			
30–34	1.5	1.6	1.5
35–39	3.0	3.2	3.1
40–44	5.8	6.2	6.0
45–49	8.2	8.8	8.5
50–54	11.6	12.4	12.0
55–59	15.5	16.5	16.0
60–64	19.1	20.5	19.8
65–69	23.9	25.6	24.7
70–74	25.0	26.8	25.9
75–84	22.1	23.6	22.9
85+	15.1	16.1	15.6

Notes:

1. Rates are per 100.
2. ‘Asian’ includes South Asian ethnic groups (who have prevalences similar to Māori and Pacific ethnic groups), as well as East and South-East Asian ethnic groups (who have prevalences similar to the European ethnic group).

Estimates of diagnosed type 2 diabetes prevalence, 2006

For evaluation or performance monitoring, estimates of current rather than past prevalence are required. Table 12 provides four such estimates (total population only):

- smoothed empirical estimates from 2001 (see Table 11), projected to 2006 by applying the growth rate estimated from the multi-state life table model* (see Table 9) [**E**]
- the multi-state life table projection ('predicted statistics') from Table 9 [**M**]
- the PHI record linkage estimates for 2006, derived by linking hospitalisation and pharmacy data (data supplied by Craig Wright) [**R**]
- the *Get Checked* estimates for 2006 (*Get Checked* is a free annual check for people living with diabetes; a comprehensive set of data is collected at the time of each annual examination) (data supplied by Sandy Dawson) [**G**].

Table 12: Comparison of different estimates for diagnosed diabetes prevalence in 2006, by age and sex

Age (years)	Male				Female			
	E	**M**	**R**	**G**	**E**	**M**	**R**	**G**
25–29	N/A	398	964	1132	N/A	261	2451	1079
30–34	1481	1140	1476	1749	1259	929	3621	1798
35–39	3003	2486	2426	2734	2626	2172	4856	2912
40–44	5749	4630	3767	3977	4895	4147	5269	4210
45–49	7181	7148	5130	5305	6076	6110	5291	5507
50–54	9749	8984	6695	6397	7958	7532	6115	6343
55–59	9829	10,778	8192	7612	8070	8913	7170	7149
60–64	10,183	10,680	8171	7376	8531	8721	7465	6811
65–69	10,280	9865	8760	7050	8735	8251	7853	6570
70–74	9784	8363	7423	5542	8758	7230	7185	5343
75–84	9857	11,820	10,413	7756	11,593	12,499	11,740	8053
85+	1552	2486	1998	1419	2922	4766	3864	2145
30+	78,648	78,380	64,451	56,917	71,423	71,270	70,429	56,841
	Sexes pooled							
30+	150,071	149,650	134,880	113,758				

Note: See text above for key to estimates (ie, definitions of E, M, R and G).

* The modelled prevalence growth rate is 4% per year. However, 5% might be more appropriate, in order to allow for some diagnostic transfer (ie, a declining trend in the ratio of undiagnosed to diagnosed diabetes).

The record linkage process (R) appears to overestimate prevalence at younger ages relative to most of the other methods, especially for females. This may reflect the inclusion of gestational diabetes along with (some) type 1 diabetes in the record linkage, as well as data quality issues relating to the prescription of oral hypoglycaemics, insulin syringes and glucose test strips in the Pharmhouse database (the latter potentially affecting all age groups). Further work is under way to improve the reliability of the record linkage process, especially for younger age groups.

However, the *Get Checked* data do suggest that the survey-based estimates (both empirical and modelled) underestimate prevalence in younger age groups (under 35 years) in 2006, indicating that the rate of growth of diagnosed diabetes prevalence from 2001 to 2006 may have been underestimated by these methods for these age groups.

Overall, the empirical and modelled prevalence estimates are very similar, at approximately 150,000 prevalent diagnoses in 2006, pooling all ages (from 25 years) and both sexes. Record linkage, as currently applied, appears to underestimate prevalence by approximately 10% overall (135,000 versus 150,000).

Applications to Monitoring

The combination of 'predicted statistics' for planning (unfortunately not available by ethnicity, socioeconomic group or region) and 'adjusted statistics' for evaluation and performance monitoring (Murray 2007), as outlined in this report, should enable many policy and programme objectives to be met. Nevertheless, large gaps remain in our databases, especially in relation to inequalities, and further improvement in diabetes surveillance methodology is urgently required.

One critical improvement would be the extension of the NZHS (and the national nutrition surveys) to include further objective measures such as HbA1c, in addition to body mass index and waist circumference. These cross-sectional prevalence surveys could also be extended to include nested cohort (follow-up) studies of participants identified (through HbA1c or similar testing) with probable (pre)diabetes. This would provide valuable information on diabetes risk factors, the prevalence of pre-diabetic states, disease progression rates, and the ratio of undiagnosed to diagnosed diabetes. Knowledge of the population distribution of HbA1c levels would also be useful in its own right. Such surveys would also provide an additional source of information on the distribution of HbA1c levels among people with self-reported (known) diabetes (ie, the quality of glycaemic control).

A second critical development would be to further improve the existing record linkage studies carried out by PHI. The current method could be extended to use anonymous probabilistic data matching methods to link hospital separations, pharmacy prescriptions, laboratory results, specialised registers (eg, for renal dialysis and transplantation) and mortality data, along with Bayesian mark-recapture methods, to estimate diabetes prevalence. This approach could also provide better information on diabetes micro- and macrovascular complication rates, medical and surgical intervention rates, health outcomes and costs.

Finally, there is scope to integrate diabetes primary health care data (including the *Get Checked* databases operated by primary health organisations) and hospital diabetes clinic data to provide evaluative information on access to and the quality of diabetes health care (eg, in comparison to published New Zealand guidelines) (New Zealand Guidelines Group 2003), including degree of metabolic control and distribution of absolute cardiovascular risk.

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