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The prevalence of diabetes in Australia: Insights from the Fremantle Diabetes Study Phase II

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Introduction

Diabetes is a major global health problem that has been characterised as one of the largest epidemics in history.¹ The progressively increasing number of people diagnosed with diabetes means that more resources are required to manage the disease and its complications;² the total annual cost of diabetes in Australian adults was estimated at A\$14.6 billion in 2010.³ An accurate assessment of the burden of diabetes is, therefore, essential for the planning and prioritisation of health services. As there are significant geographical differences in trends in diabetes prevalence,⁴ there is a need for epidemiological studies to be performed at the individual country level.

There is evidence that the prevalence of diabetes in Australia has increased over the past few decades, although there are apparent inconsistencies. Studies employing biochemical screening of adults aged ≥ 25 years in Busselton, Western Australia, in 1981⁵ and of individuals of European ancestry aged ≥ 15 years in rural Victoria in 1992⁶ both showed a total prevalence (known and newly diagnosed) of 3.4%. Using a similar approach, the Australian Diabetes, Obesity and Lifestyle Study (AusDiab) conducted in 1999–2000 across Australia found an overall prevalence of 7.4% in adults aged ≥ 25 years.⁷ More recently, the

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Conflicts of interest
None disclosed.

Australian Health Survey (AHS) conducted in 2011-12 found that 4.0% of the total population reported having been told by a health professional that they had diabetes and that it was long-term.⁸ These data were used to provide an estimate of 4.2% in a subset of adults aged 18 years with the addition of biochemical screening, although those who were diagnosed with, but not taking medication for, diabetes and who did not have a fasting serum glucose ≥ 7.0 mmol/L were excluded.⁹ The National Diabetes Services Scheme (NDSS) delivers diabetes-related products at subsidised prices and provides information and support services to people with diabetes. Registration is free and open to all Australians diagnosed with diabetes. Notwithstanding incomplete and inappropriate registration, the current (2017) estimate for the percentage of Australians who are on the NDSS is 5.1%.¹⁰

An assessment of the differences between these various prevalence estimates needs to take into consideration the population screened or surveyed (total versus adult, ethnicity), definitions of diabetes (self-reported versus biochemically ascertained, including changes in glycaemic thresholds as occurred in 1998¹¹), and the possibility of selection bias when a minority of eligible participants are recruited.¹² Each approach has limitations, and it may be prudent to use available data from a variety of sources to inform estimates of the change in prevalence and, more importantly for health services planning, the contemporary burden of diabetes in Australia.

With this objective in mind, we have utilised a further method of ascertaining diabetes prevalence in Australia, namely through systematic active detection of known diabetes cases. The Fremantle Diabetes Study Phase II (FDS2) is an ongoing longitudinal observational study carried out in a postcode-defined geographical area surrounding the port city of Fremantle in Western Australia (WA).¹³ The primary aim of the present study was to utilise FDS2 ascertainment data, in conjunction with contemporaneous AHS, NDSS and Australian Bureau of Statistics (ABS) data, to generate a relatively contemporary estimate of diabetes prevalence in Australia. A secondary aim was to determine the proportions of type of diabetes from the same data sources.

Materials and Methods

Study population

Based on Australian Census data, there were 149,111 people living in the FDS2 postcode-defined study catchment area in 2006¹⁴ and 168,473 in 2011.¹⁵ Interpolating these data to 2008, there were approximately 157,000 people living in the area at the start of FDS2 recruitment. Socio-economic data relating to income, employment, housing, transportation and a range of other variables in this area show an average Index of Relative Socio-economic Advantage and Disadvantage¹⁶ of 1033 with a range by postcode of 977-1113, figures similar to the Australian national mean $\pm$ SD which are set at 1000 $\pm$ 100.

The FDS2 recruitment strategy comprised identification of any resident in the study catchment area who had a clinician-verified diagnosis of diabetes between 2008 and 2011 from public hospital inpatient/outpatient clinic lists and laboratory databases, notifications by local primary care/specialist physicians and allied health services including diabetes education, dietetics and podiatry, advertisements in pharmacies and local media, third party

mail-outs through the NDSS and National Diabetes Register of insulin-treated patients, and word of mouth. The FDS2 was approved by the Human Research Ethics Committee of the Southern Metropolitan Area Health Service. All participants gave written informed consent.

Methods

All FDS2 participants were scheduled for face-to-face assessments at entry and then biennially, interspersed with biennial postal questionnaires.¹³ Face-to-face assessments included a standardised comprehensive questionnaire, physical examination and fasting biochemical tests performed in a single nationally accredited laboratory. Type of diabetes was ascertained at study entry based on diabetes treatment history (especially insulin and its initiation relative to diagnosis), body mass index (BMI), age at diagnosis, nature of first presentation, and/or self-identification including a previous clinician diagnosis of Maturity Onset Diabetes of the Young (MODY) or neonatal diabetes, with available case records consulted for evidence of ketonaemia, as well as autoantibodies, serum insulin and/or C-peptide concentrations, and genotyping, if available. Diabetes was consequently classified as type 1, type 2, latent autoimmune diabetes of adults (LADA), monogenic or secondary. Ethnicity was categorised based on self-selection, country/countries of birth, and parents' birth and grandparents' birth, and language(s) spoken at home as Anglo-Celt, Southern European, Other European, Asian, Aboriginal or mixed/other.¹³

All participants aged <35 years at diagnosis were assessed using the UK MODY risk prediction models.^{17, 18} Those identified as probable MODY underwent subsequent genetic testing with analysis of all coding regions and exon/intron boundaries, as well as partial/whole gene deletions and duplications, of the known monogenic diabetes genes *GCK*, *HNF1A*, *HNF4A*, *HNF1B*, *NEUROD1*, *INS*, *INSR*, *KCNJ11*, *ABCC8*, *PDX1*, *CEL*, *PAX6*, *GATA6*, *TRMT10A*, *WFS1*, *ZFP57*, *PCBD1*, *LMNA*, *PPARG*, *PLIN1* and *POLD1*, and the m.3243A>G MIDD mutation, by targeted next generation sequencing (Agilent custom capture v5.1/Illumina HiSeq, Agilent Technologies LDA UK Ltd, Stockport, Cheshire, UK)¹⁹ performed at the Molecular Genetics Laboratory, Royal Devon and Exeter NHS Foundation Trust, UK. Serum glutamic acid decarboxylase (GAD65) autoantibodies were determined using stored sera from the baseline assessment and a commercially available ELISA kit (RSR GAD65, RSR Limited, Cardiff, UK) run on an EVOLIS automated ELISA workstation (Bio-Rad laboratories, Australia). GAD65 positivity was defined as a titre ≥ 5 U/mL. The mean inter- and intra-assay coefficients of variation were 6.5% and 6.4%, respectively.

Basic confidential data (age, sex, country of birth, type of diabetes) were also collected from available sources for the non-recruited residents with known diabetes in the FDS2 catchment area.

Results

Diabetes prevalence

We identified 4,639 patients with diabetes (excluding gestational diabetes) during FDS2 registration between 2008 and 2011 with 1,668 (36%) being recruited. A further 64

surviving participants from the first phase of FDS who lived outside the study area were also recruited, but these were excluded from the present analysis.¹³

Using the 4.0% overall prevalence from AHS in both 2007-2008²⁰ and 2011-2012,⁸ as well as ABS-based population estimates at the same times, 6,280 and 6,739 residents had known diabetes in the FDS2 catchment area at the beginning and end of the FDS2 recruitment period, respectively (see Table 1). Data from the NDSS relating to the numbers of people residing within the FDS2 catchment area by age, sex, and type of diabetes in 2010, 2011 and 2012, suggested that 7,211, 7,774 and 7,180 persons, respectively, were identified as having diabetes with corresponding diabetes prevalence rates of 4.4%, 4.6%, and 4.2% (see Table 1). The lower 2012 figure was derived after extensive in-house NDSS database verification including removal of deceased persons, duplicates, those with gestational diabetes who were post-partum, and those without evidence of diabetes. Compared with these rates, the 4,639 identified (recruited and non-recruited) in FDS2 gave a prevalence estimate of 2.9%, or between 28% and 37% lower than those derived from other sources (see Table 1).

FDS2 participants and non-participants

Adjusted to the beginning of recruitment period in 2008, the 4,639 people with diabetes identified in the catchment area were of mean±SD age 61.6±16.2 years, 52.3% were males, 89.7% had clinically-defined type 2 diabetes, 8.6% type 1 diabetes, 0.6% LADA, and 1.1% secondary or other types. The age distribution of these participants compared with those in the NDSS is shown in Figure 1. Diabetes type could not be ascertained in 96 non-participants (3.2%), while 4 participants (0.2%) required further information before categorisation. The majority of residents with diabetes were born in Australian/New Zealand (56.4%), 17.7% in Southern Europe, 13.9% in the UK/Ireland, 3.1% in other European countries, 4.8% in Asia, and 4.0% in other regions of the world. Aboriginal/Torres Strait Islanders comprised 4.6% of all identified residents with diabetes.

There were no significant differences between participants and non-participants in age at the beginning of recruitment in 2008, in sex or in diabetes type (excluding those with unknown type) (P 0.17; see Table 2). Region of birth was significantly different by participant status (P <0.001), with a greater proportion of participants than non-participants being born in Australia/New Zealand and in the UK/Ireland, while a smaller proportion were born in Southern Europe. A higher proportion of participants than non-participants were Aboriginal/Torres Strait Islanders (P <0.001). A significant reason for non-participation was death before recruitment, with 12.2% of those not recruited dying within the recruitment period compared with 2.9% of participants (P <0.001).

FDS2 participants

Over half (52.2%) of FDS2 participants were males, they were of mean±SD age 63.6±13.6 years at study entry with a median [inter-quartile range] diabetes duration of 9.0 [3.0-16.0] years. The majority (1,499 or 89.9%) had clinically-defined type 2 diabetes, 12 (0.7%) had LADA, 132 (7.9%) had type 1 diabetes, 17 (1.0%) secondary diabetes, 4 (0.2%) monogenic diabetes, and 4 (0.2%) required confirmatory evidence before categorisation.

Most (87.4% of 1,608 responders) of the FDS2 participants self-reported being NDSS members although this has not been validated from NDSS sources. This proportion did not differ significantly by diabetes type, diabetes treatment, or ancestry (P 0.30). The majority of FDS2 participants (83.6%) reported self-monitoring their blood glucose levels a median 7 [3-14] times/week, 92.6% of those self-monitoring reported being NDSS members. Thus, the NDSS database may have less than 90% ascertainment due to not everyone with diabetes being registered. If 12.6% of Australians with known diabetes do not register with the NDSS, the 2012 NDSS prevalence could be inflated to $4.2\%/0.874 = 4.8\%$ or 1.1 million Australians.

FDS2 participants with diet-based management

Nearly one-quarter (22.6%) of FDS2 participants managed their diabetes based on diet alone, while almost a half (49.0%) were taking oral glucose-lowering medications (OGLMs), two (0.1%) were taking OGLMs with a non-insulin injectable, one-eighth (12.8%) used insulin alone, two were taking mixed insulin with a non-insulin injectable, 15.2% both insulin and OGLMs, and two were taking basal insulin and OGLMs with a non-insulin injectable.

Of those who managed their diabetes based on diet alone, 69.1% had a fasting serum glucose <7.0 mmol/L. Thus, 15.6% of the FDS2 cohort would not be counted as having diabetes by the AHS combination of self-report and fasting serum glucose.⁹ Since 20.3% of this group were not NDSS members, they would not be identified by either the AHS or the NDSS as having diabetes, i.e. $15.6\% \times 20.3\% = 3.2\%$ of the FDS2 cohort would not be captured by the combined sources. For the 206 (12.4%) diet-treated with $HbA_{1c} <6.5\%$ and fasting serum glucose <7.0 mmol/L (below the threshold for diabetes diagnosis²¹), 99.0% had type 2 diabetes, one was in the honeymoon stage of type 1 diabetes (when compensatory residual pancreatic beta cell function can reduce exogenous insulin needs), and one had secondary diabetes due to pancreatitis. Most (68.1%) of this latter group were diagnosed incidentally, self-reported NDSS membership was 80.5% and 78.5% self-monitored their blood glucose level 4 [2-7] times/week.

Figure 2 illustrates the potential for not identifying all Australians with known diabetes using the NDSS and AHS, and the overlap between these two methods. Three-quarters (75.0%) of FDS2 participants with known diabetes would have been captured by both the NDSS and AHS, 3.2% would not have been captured by either, 9.4% by the AHS only, and 12.4% by the NDSS only.

Confirmation of diabetes type

Testing for GAD65 antibodies identified 158 (9.5%) positive results (72 in clinically-defined type 2 diabetes, one confirming LADA in a participant requiring further information before categorisation, eight in known LADA, and 77 in type 1 diabetes). Genotyping for MODY¹⁸ revealed one case of a novel *GCK* gene mutation (c.1253G>T; p. Ser418Ile) and a previously reported missense mutation known to be associated with familial partial lipodystrophy (*LMNA*, c.1444C>T; p.Arg482Trp²²). Including this information refined the proportions with type 1 diabetes, type 2 diabetes, LADA, MODY and secondary diabetes to

7.9% (no change), 85.8%, 4.9%, 0.4% and 1.0% (no change), respectively. The proportions of participants with clinically-defined type 2 diabetes who had LADA or monogenic diabetes were 4.8% and 0.1%, respectively.

Discussion

All of the recent methods of estimating known diabetes prevalence in Australia have limitations. The AHS combination of self-report and fasting serum glucose ≥ 7.0 mmol/L excludes those who are well controlled on diet-based management,^{8,9} a percentage of total cases in FDS2 of 15.6%. Thus the AHS prevalence of 4.2% for known diabetes in those aged ≥ 18 years may be closer to 5.0%, but this figure excludes younger age-groups in which the prevalence of diabetes is relatively low. The reduction in NDSS prevalence with more rigorous data validation in 2012 (4.2%) compared with the preceding two years (up to 4.6%) suggests that there may still be inappropriate registrations, but FDS2 data suggest that 12.6% of Australians with known diabetes do not register with the NDSS. This could inflate the 2012 NDSS prevalence to 4.8%. The 4.0% ascertained from self-report for Australians of all ages by the AHS in 2011-12⁸ is a likely underestimate of the true prevalence of known diabetes. Based on these considerations, we estimate that the prevalence of known diabetes in Australia in 2011-12 was that derived from NDSS and FDS2 data, specifically 4.8% or 1.1 million Australians.

The 2.9% prevalence derived from FDS2 identification methods represents 62% ascertainment of our overall estimate of 4.7% of all patients with diabetes in the FDS2 catchment area informed by assessment of NDSS and AHS data. This rate of ascertainment is consistent with a UK study in which case identification from similar health database and other sources to those used in FDS2 was associated with $<50\%$ ascertainment relative to that in capture-recapture analysis.²³ The estimated mean age of NDSS registrants in Figure 1 (64.4 years) was close to that for all people with diabetes identified in FDS2 (66.3 years extrapolated to the end of 2012 or 64.5 years excluding those who had died by this time) and the two distributions were generally similar. However, the FDS2 identification rate was low for the 0-10 year olds, perhaps reflecting their exclusive use of paediatric health services outside the FDS2 catchment area, with an increase in the rate for the 16-25 year age group that was very close to that in the NDSS during the transition to local adult care. There were similarly high ascertainment rates in older people, probably consistent with greater use of health care services generally.

In the case of FDS2 participants, there was evidence that the young and the old did not volunteer as readily as those in other age groups (see Figure 1). Recruitment to FDS2 was highest (28% of NDSS registrants) for 66-75 year olds, a period when health status facilitates participation in such studies and most have retired. In adults aged 21-65 years, participation rates were lowest (14-23% of the NDSS) for those of working age (21-50 years), with rates rising in the 51-65 year olds with the onset of retirement (23-24%). The lowest rates, however, were for ≤ 21 year olds (0-14%) due to the difficulties in identifying the small numbers of children and adolescents with diabetes and the challenge of engaging with both the young people and their parents.

Data from Australia^{9, 24} and other developed countries²⁵ suggest that the prevalence of previously undiagnosed diabetes may be <25% that of known diabetes. These observations appear at odds with AusDiab data suggesting an equivalent (100%) relative prevalence.⁷ This and the relatively high rate of known and newly diagnosed diabetes in AusDiab (7.4% in adults aged ≥ 25 years⁷) may reflect selection bias¹² including higher proportions of older men and, to a lesser extent, women and of participants who suspected they had diabetes compared to those who did not participate.⁷ It is also likely that increasing awareness of the diagnosis in primary care since AusDiab, and thus greater targeted screening, has reduced the proportion of cases of type 2 diabetes that remain undiagnosed.

The distribution of type of diabetes showed a higher percentage of type 1 diabetes in the AHS and NDSS databases compared with FDS2 participants (10.0% to 12.4% versus 7.9%; see Table 1). This is very likely to reflect misclassification of insulin-treated type 2 diabetes as type 1 in the AHS and NDSS²⁶ compared with the algorithmic approach to establishing diabetes type used in FDS2.¹³ Further diagnostic refinement through GAD65 antibody assays and MODY genotyping in FDS2 did not change the percentage of participants with type 1 diabetes but reduced the type 2 percentage, suggesting that relatively few type 1 patients will be misclassified if validated clinical algorithms are applied.

The present study had limitations. As acknowledged, ascertainment of diabetes in epidemiological studies, surveys and registries can miss true cases and include people who do not have diabetes. We have, however, attempted to address this limitation in our analyses with the use of multiple data sources.²³ Based on historical trends^{5–8} and our estimate of 4.8% for 2011–2012, the current prevalence of diabetes in Australia may be approaching 5.0%. A strength of this study is the inclusion of the well characterised FDS2 cohort including a relatively rigorous assessment of diabetes type.

Conclusion

We have used data from FDS2 and other sources including the AHS and NDSS to estimate the prevalence of diabetes and distribution of its types in a relatively contemporary Australian community setting. The results suggest that around 1 in 20 Australians have diabetes and that the majority (86%) have type 2 diabetes. Patients diagnosed clinically with type 2 diabetes should, depending on features such as age at presentation and family history, be assessed for the possibility of LADA and MODY since these diagnoses may lead to a change to more appropriate blood glucose lowering therapies. We recommend that future estimates of diabetes prevalence and type in this country are informed by data from as many relevant sources as possible given the limitations inherent in most registers, databases and epidemiological studies.

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Figure 1.

Age distributions as at December 2012 in National Diabetes Services Scheme (NDSS) registrants (medium grey bars) versus residents of the Fremantle Diabetes Study (FDS) area identified with diabetes (lightest grey bars) and, FDS Phase II (FDS2) participants (black bars).

Figure 2.
Pie chart showing coverage of different methods of identifying Australians with known diabetes based on Fremantle Diabetes Study Phase II data: National Diabetes Services Scheme registrants and Australian Health Survey estimates in agreement (NDSS and AHS), NDSS only, AHS only, and missed prevalent cases (unlabelled white sector).

Table 1
Prevalence and type of known diabetes mellitus (DM) in Australia and the FDS2 study area 2007-2012.

Survey	Year	Method	Geographical area	Prevalence (%)	Numbers with DM* in FDS study area	Type 1 / Type 2 / Other or unknown type (%)
AHS	2007-08 ²⁰	Self-reported current	Australia	4.0	6280 [‡]	10.0 / 88.2 / 1.8
AHS	2011-12 ⁸	Self-reported current	Australia	4.0	6739 [‡]	12.4 / 85.3 / 2.2
NDSS	17/11/2010	Registered with DM in FDS area	FDS study area	4.4	7211	11.8 / 87.8 / 0.3
NDSS	7/7/2011	Registered with DM in FDS area	FDS study area	4.6	7774	11.6 / 88.4 / 0
NDSS	9/11/2012	Registered with DM in FDS area	FDS study area	4.2	7180	11.0 / 88.6 / 0.4
FDS2	2008-11	Identified with known DM from multiple sources in FDS area	FDS study area	2.9	4639	Clinical 7.9 / 89.9 / 2.2 Laboratory testing: 7.9 / 85.8 / 6.3

* Excluding GDM;

[‡] assuming national data apply to the FDS study area; AHS = Australian Health Survey; NDSS = National Diabetes Services Scheme

Table 2

Comparison of characteristics of participants and non-participants in the Fremantle Diabetes Study Phase II at the beginning of recruitment (18 February 2008).

	Participants	Non-participants	P-value
Number	1668	2971	
Age (years)	62.0 ± 13.8	61.3 ± 17.4	0.17
Male (%)	52.2	52.4	0.88
Region of birth (%):			<0.001
Australia/New Zealand	59.1	54.8	
United Kingdom/Ireland	15.8	12.7	
Southern Europe	12.9	20.7	
Other Europe	2.5	3.5	
Asia	5.3	4.5	
Other	4.4	3.8	
Aboriginal/Torres Strait Islander (%)	6.7	3.4	<0.001
Type of diabetes (%; excluding unknown):			0.35
Type 1	7.9	9.0	
LADA	0.7	0.5	
Type 2	90.1	89.5	
Secondary/other	1.3	1.0	