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RESEARCH ARTICLE

Low-density lipoprotein cholesterol in adolescents and young adults with type 1 diabetes: Data from the Australasian Diabetes Data Network registry

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Abstract

Aim: To determine low-density lipoprotein cholesterol (LDL-C) screening frequency and levels, and factors associated with elevated LDL-C, in Australasian youth with type 1 diabetes (T1D).

Methods: Data were extracted from the Australasian Diabetes Data Network (ADDN), a prospective clinical quality registry, on all T1D healthcare visits attended by young people aged 16–25 years (with T1D duration of >1 year) between January 2011 and December 2020. The primary outcomes were elevated LDL-C > 2.6 mmol/L (100 mg/dL) and threshold for treatment: >3.4 mmol/L (130 mg/dL), according to consensus guidelines. Multivariable Generalised Estimated Equations (GEE) were used to examine factors associated with elevated LDL-C across all visits.

Results: A cohort of 6338 young people (52.6% men) were identified, of whom 1603 (25.3%) had ≥1 LDL-C measurement documented. At last measurement, mean age, age at T1D diagnosis and T1D duration were 18.3 ± 2.4, 8.8 ± 4.5 and 8.9 ± 4.8 years, respectively. LDL-C was elevated in 737 (46.0%) and at the treatment threshold in 250 (15.6%). In multivariable GEE elevated LDL-C continuously was associated with older age (OR = 0.07; 0.01–0.13, $p = 0.02$), female sex (OR = 0.31; 0.18–0.43; $p < 0.001$), higher HbA1c (OR = 0.04; 0.01–0.08; $p = 0.01$) and having an elevated BMI (OR = 0.17, 0.06–0.39, $p < 0.001$).

Conclusions: LDL-C screening and levels are suboptimal in this cohort, increasing future cardiovascular complication risk. There is an urgent need to understand how healthcare services can support improved screening and management of dyslipidaemia in this population.

KEYWORDS

adolescents, cholesterol, LDL, type 1 diabetes, young adults

1 | INTRODUCTION

Cardiovascular disease (CVD) is a leading cause of morbidity and mortality in type 1 diabetes (T1D), with dyslipidaemia, defined as low levels of high-density lipoprotein cholesterol, or elevated total or low-density lipoprotein cholesterol (LDL-C) levels, being important contributing risk factors.[1–3] Evidence from genetic, epidemiologic and clinical studies consistently demonstrate that any mechanism of lowering plasma LDL-C particle concentration reduces the risk of atherosclerotic CVD. In people with familial hypercholesterolemia, the initiation of statin therapy during childhood slows the progression of carotid intima-media thickness and reduces the risk of CVD in adulthood.[4] With the age-adjusted relative risk for CVD in T1D at around 10 times that of the general population, an aggressive approach to screening and management of LDL-C in young people with T1D is warranted.[5,6]

Consensus guidelines from the International Society for Pediatric and Adolescent Diabetes (ISPAD) recommend screening for dyslipidaemia from age 11 years, with screening repeated every 5 years if normal results are obtained.[3] Similar screening frequency is recommended by the American Diabetes Association for individuals aged <40 years.[2] Both guidelines define elevated LDL-C as >2.6 mmol/L (100 mg/dL) and recommend dietary and lifestyle intervention as initial management, with statins commenced from age 11 years if LDL-C is >3.4 mmol/L (130 mg/dL).[2,3]

Worldwide, longitudinal data on LDL-C in young people with T1D have been limited by their retrospective nature and small sample sizes, and in Australasia, there is a paucity of published data.[7,8] We aimed to determine LDL-C screening frequency and levels, and to identify factors associated with elevated LDL-C in Australasian youth with T1D.

What's new

What is already known?

- Cardiovascular disease is a leading cause of type 1 diabetes (T1D) morbidity and mortality. Worldwide, longitudinal data on low-density lipoprotein (LDL-C) in young people with T1D are limited.

What this study has found?

- At last LDL-C measurement, LDL-C was elevated in 46.0% and at the treatment threshold in 15.6%. Across all T1D healthcare visits, elevated LDL-C was associated with older age, female sex, higher HbA1c and elevated body mass index.

What are the implications of the study?

- LDL-C screening and levels are suboptimal in this cohort, increasing future cardiovascular complication risk. Our findings can inform discussion around how T1D healthcare services can support improved screening and LDL-C in this population.

2 | METHODS

2.1 | Sample

Clinical data were collected from the Australasian Diabetes Data Network (ADDN), a collaboration between the Australian and New Zealand Society for Paediatric Endocrinology and Diabetes, the Australian Diabetes Society, the University of Melbourne, and the many diabetes centres represented by the ADDN Study Group. Established in 2012, the prospective clinical registry currently includes data on the majority (approximately 60%) of young people with diabetes attending tertiary centres in Australasia. Participating centres upload data to ADDN twice a year, and data quality is ensured by comprehensive data validation rules and error reports.

For this analysis, data derived from 24 participating centres across Australasia; 13 (54.2%) of which were paediatric centres. A systematic process for data extraction and cleansing was used, as explained elsewhere.[9,10] Summary data were extracted on all T1D healthcare visits attended by young people who were aged 16–25 years (with T1D duration of >1 year) at their last T1D healthcare contact during a 10-year period (1st January 2011–31st December 2020). This age range was chosen since older adolescence is widely recognised as a distinct phase of maturation and a period of particular vulnerability as youth transition to adulthood, generally complete by age 25 years.[11] Data extracted included socio-demographic and clinical variables, including age at T1D diagnosis, sex, T1D duration, insulin regimen, HbA1c, blood lipid profile, systolic and diastolic blood pressure (BP), and height and weight.

BP was defined as in the hypertensive range when participants aged <18 years had a systolic and/or diastolic BP ≥95th percentile, and for those aged ≥18 years, systolic BP ≥130 and/or diastolic BP ≥80 mmHg.[12] BMI was categorised as elevated when BMI standard deviation score (SDS) was ≥85th percentile for <18 years or BMI >25 kg/m^2 for

≥18 years.[13] All centres contributing data had Human Research or Health and Disability Ethics Committee approval for participation in ADDN, and the current analysis was approved by the University of the Sunshine Coast Human Research Ethics Committee, Australia (reference: E19113).

2.2 | Statistical methods

Analyses were performed for the last and all recorded LDL-C measurements. Descriptive statistics are reported as mean ± standard deviation (SD), and median and interquartile range (IQR 25–75th centile). The primary outcomes were elevated LDL-C (>2.6 mmol/L, 100 mg/dL) and the threshold for therapy (>3.4 mmol/L, 130 mg/dL), hereafter referred to as 'elevated' and 'threshold'. Univariate associations between categorical variables were examined using chi-square and continuous variables using *t*-tests.

Multivariable Generalised Estimated Equation (GEE) models were used to examine factors associated with elevated LDL-C across all T1D healthcare visits, analysed continuously and as two categorical variables (elevated and threshold). Potential explanatory variables included age, T1D duration, sex, year of consultation, HbA1c, insulin treatment (continuous subcutaneous insulin infusion [CSII] use vs. not), BP (in the hypertensive range vs. not) and BMI (elevated vs. not); variables chosen based on clinical knowledge and previous literature. Goodness of fit was assessed using the two extensions of Akaike's information criterion for model selection: quasi-likelihood under the independence model criterion (QIC) for choosing the best correlation structure and another QIC measure for choosing the best subset of predictors. Results are reported as odds ratios (OR) and 95% confidence intervals (95% CI), with $p < 0.05$ considered significant. Where case data were missing, all available data were included in analyses. Analyses were performed using SPSS version 27 (IBM).

3 | RESULTS

For this analysis, 6338 young people ($n = 3334$, 52.6% men) with T1D were identified; 4877 (77.0%) of this cohort attended paediatric diabetes centres. At last T1D healthcare visit, mean ± SD age was 18.4 ± 2.3 years, age at T1D diagnosis 9.2 ± 4.4 years and T1D duration 8.7 ± 4.7 years. Most young people were born in Australia or its territories (85.6%) or New Zealand (5.4%), with 56 (1.4%) identifying as Aboriginal and/or Torres Strait Islander and 63 (1.6%) as Māori. Treatment regimen was by multiple daily injection (MDI) in 53.8%, CSII in 37.6% and twice-daily (BD) injections in 8.5%.

3.1 | Last LDL-C measurement

Overall, LDL-C was documented in 1603 (53.7% men); 1107 (69.1%) attended paediatric centres (Table 1). Mean age, age at T1D diagnosis and T1D duration were 18.3 ± 2.4, 8.8 ± 4.5 and 8.9 ± 4.8 years, respectively, and for 1548/1603 mean HbA1c was 8.8% ± 1.8% (73.1 ± 20.1 mmol/mol); the measurement was ≥7.0% (53 mmol/mol) for 1384/1548 (89.4%). LDL-C was elevated in 737/1603 (46.0%) and above threshold in 250 (15.6%). Characteristics at last documented LDL-C measurement, stratified by HbA1c, are shown in Figure 1. As HbA1c increased, the percentage of young people with LDL-C at the level >2.6 mmol/L (100 mg/dL) increased.

3.2 | All T1D healthcare visits

Across 36,655 T1D healthcare visits, mean age was 17.8 ± 2.1 (min–max 15–25 years). Each young person ($n = 6338$) had a median of 5.0 (IQR 2.0, 8.0) occasions of service; the time difference between first and last visit for those with >1 visit ($n = 5229$) was 91.0 (51.0, 126.0) weeks. LDL-C was documented on 2457/36,655 (6.7%) occasions; 488/1603 (30.4%) young people had >1 measurement and 80/1603 (5.0%) had ≥3 LDL-C measurements documented, across a median 53.0 (26, 88) weeks; mean LDL-C across measurements was 2.9 ± 0.9 mmol/L.

In young people that had >1 LDL-C measurement, at first measurement mean age was 18.4 ± 2.3 years and LDL-C was 2.8 ± 0.9 mmol/L; elevated LDL-C was found in 248 (50.8%) at the level >2.6 mmol/L (100 mg/dL) and 101 (20.7%) at the level >3.4 mmol/L (130 mg/dL). When compared to the first measurement, at last measurement mean LDL-C was identical at 2.8 ± 0.9 mmol/L; elevated LDL-C was found in 257 (52.7%) at the level >2.6 mmol/L (100 mg/dL) and 105 (21.5%) at the level >3.4 mmol/L (130 mg/dL). Calendar year did not have a significant linear or quadratic impact on documented LDL-C values across T1D healthcare visits.

Using multivariable GEE modelling, elevated LDL-C was associated with older age, female sex, higher HbA1c and elevated BMI (Table 2). LDL-C above threshold was associated with female sex, whereas elevated LDL-C continuously was associated with older age (OR = 0.07; 0.01–0.13, $p = 0.02$), female sex (OR = 0.31; 0.18–0.43; $p < 0.001$), higher HbA1c (OR = 0.04; 0.01–0.08; $p = 0.01$) and having an elevated BMI (OR = 0.17, 0.06–0.39, $p < 0.001$).

Longitudinal data for ≥5 years were available for 222/1603 (13.9%) young people with T1D, across median 313.5 (290, 375) weeks; mean LDL-C across measurements was 2.8 ± 0.9 mmol/L. In this population, 71/222 (32.0%)

Variable	Overall, $n=1603$	<18 years, $n=870$	≥18 years, $n=733$	p Value
Age (years)	18.3 ± 2.4	16.4 ± 3.0	20.4 ± 2.0	**<0.001**
Women, *n* (%)	742 (46.3)	411 (47.2)	331 (45.2)	0.42
Age at T1D diagnosis (years)	8.8 ± 4.5	8.3 ± 4.1	9.5 ± 4.8	**<0.001**
T1D duration (years)	8.9 ± 4.8	7.7 ± 4.1	10.4 ± 5.1	**<0.001**
Attended paediatric centre, *n* (%)	1107 (69.1)	832 (95.6)	275 (37.5)	**<0.001**
Therapy, *n* (%)*				
BD/MDI	814 (56.6)	465 (56.0)	349 (57.5)	0.59
CSII	623 (43.4)	365 (44.0)	258 (42.5)	0.59
HbA1c (%)	8.8 ± 1.8	8.8 ± 1.8	8.9 ± 1.9	0.55
HbA1c (mmol/mol)	73.1 ± 20.1	72.3 ± 20.0	73.9 ± 20.3	—
BP hypertensive range, *n* (%)	264 (28.2)	108 (22.3)	156 (35.4)	**<0.001**
Overweight/obese, *n* (%)	381 (36.9)	291 (35.9)	90 (40.4)	0.24

TABLE 1 Characteristics of young people with T1D in the ADDN registry at last LDL-C measurement.

Note: Complete data were not always available on every participant. Mean ± SD unless stated. $p<0.05$ considered significant (in bold).

Abbreviations: ADDN, Australasian Diabetes Data Network; BD, twice-daily injections; BP, blood pressure; CSII, continuous subcutaneous insulin infusion; LDL-C, low-density lipoprotein; MDI, multiple daily injections; T1D, Type 1 diabetes.

*Data on other therapies have not been detailed.

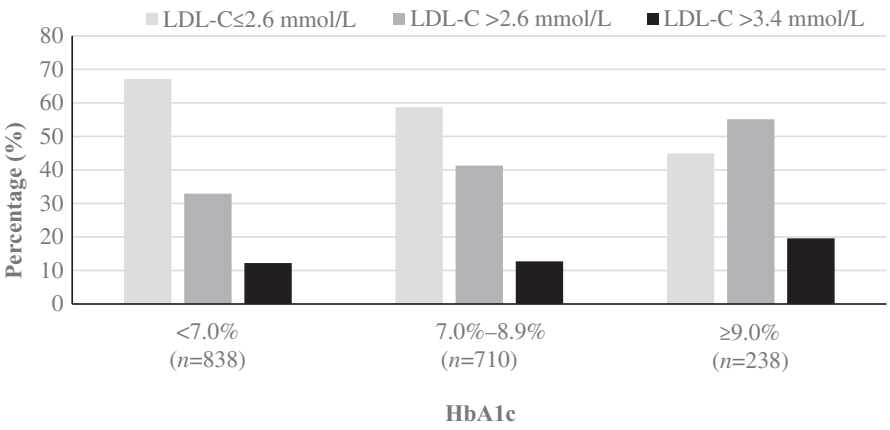

FIGURE 1 Characteristics of young people with T1D in the ADDN registry at last documented LDL-C measurement, stratified by HbA1c. 7.0% = 53 mmol/mol; 9.0% = 75 mmol/mol. 2.6 mmol/L = 100 mg/dL; 3.4 mmol/L = 130 mg/dL. ADDN, Australasian Diabetes Data Network; LDL-C, low density lipoprotein cholesterol; T1D, Type 1 diabetes.

young people had >1 LDL-C measurement documented. At first measurement, mean age was 19.7 ± 2.1 years and LDL-C was 2.9 ± 0.8 mmol/L; elevated LDL-C was found in 42 (59.2%) at the level >2.6 mmol/L (100 mg/dL) and 15 (21.1%) at the level >3.4 mmol/L (130 mg/dL). When compared to the first measurement, at last measurement mean LDL-C was improved at 2.7 ± 0.9 mmol/L ($p=0.006$); elevated LDL-C was found in 35 (49.3%) at the level >2.6 mmol/L (100 mg/dL) and 11 (15.5%) at the level >3.4 mmol/L (130 mg/dL).

4 | DISCUSSION

LDL-C levels are suboptimal in young people with T1D recorded in ADDN, increasing future cardiovascular complication risk. Overall, LDL-C was measured in 1603 (25.3%), and where documented, LDL-C was elevated; the proportion with T1D falling within consensus guideline targets[1–3] was low, with 46.0% having an elevated LDL-C, and 15.6% above the threshold for therapy at last measurement. Risk factors identified in multivariable analysis included older age, female sex, higher HbA1c and having an elevated BMI.

This is the largest study investigating the routine measurement of LDL-C in Australasian young people with T1D and one of few studies globally. When comparing our data on screening frequency, our findings are not consistent with a smaller study involving children with T1D elsewhere. In an online survey sent to members of the Association of Children's Diabetes Clinicians in the United Kingdom to assess cholesterol screening practice in young people with T1D, the majority (94%) of 87 respondents reported measuring cholesterol, with 33% stating

TABLE 2 Multivariable Generalised Estimating Equation modelling of LDL-C in young people with T1D in the ADDN registry, across all T1D healthcare visits.

	LDL-C > 2.6 mmol/L			LDL-C > 3.4 mmol/L		
Variable	**OR**	**95% CI**	***p* Value**	**OR**	**95% CI**	***p* Value**
Age	1.14	1.02–1.28	**0.02**	1.28	1.00–1.63	0.05
Women	2.00	1.57–2.55	**<0.001**	1.84	1.19–2.84	**0.006**
HbA1c	1.18	1.10–1.26	**<0.001**	0.93	0.78–1.12	0.49
Elevated BMI	1.57	0.49–0.72	**<0.001**	1.21	0.59–2.47	0.61

Note: $n = 1083$ young people with T1D and $n = 1397$ T1D healthcare visits/LDL-C measurements. $p < 0.05$ considered significant (in bold).

Abbreviations: ADDN, Australasian Diabetes Data Network, BMI, body mass index; LDL-C, low-density lipoprotein; T1D, Type 1 diabetes.

they do so annually. There was, however, marked variability in sampling for those screened.[14] As the characteristics of screened participant groups differed by clinician, it may be that applicable healthcare professionals apply their own judgements as to who would more benefit from an LDL-C measurement. These assumptions concur with our findings, as our Australasian data link similar characteristics with abnormal LDL-value state. Better understanding is needed of healthcare professional's decision making choices for LDL-C measurement; a review and implementation of protocols to ensure consistent LDL-C screening in paediatric and adult healthcare settings is warranted.

Our findings are, however, consistent with smaller studies involving LDL values in T1D populations internationally.[15–18] For example, in the SEARCH for Diabetes in Youth population, 47.0% of people with T1D aged >10 years had an LDL-C > 2.6 mmol/L (100 mg/dL), with 15% >3.4 mmol/L (130 mg/dL).[17] Similarly, in Egypt, 50% of children and adolescents with T1D were reported with elevated LDL-C levels.[16] Further, in the Pittsburgh Epidemiology of Diabetes Complications study, where participants were followed biennially for up to 25 years with surveys and/or examinations (median age 27 years), at the latest recorded follow-up, achievement of American Diabetes Association recommendations for LDL-C had decreased from 62.3% to 39.7%.[19]

We found associations between elevated LDL-C and female sex, and elevated BMI, which are consistent with other studies. For example, urban Asian Indian adolescent girls aged 14–18 years had higher mean LDL-C than boys at all ages,[20] and in Brazilian adolescents, the prevalence of dyslipidaemia was 72.5%, of whom 63.3% were women.[15] This is concerning since lipid abnormalities may promote atherogenesis in T1D. The pathophysiology is not totally explained, though hyperglycaemia and peripheral hyperinsulinaemia, due to the subcutaneous route of insulin administration, are considered to play a role.[21] When considering BMI, another study of adolescents from Brazil reported higher LDL-C in those with elevated body mass.[22] The potential impact of factors such as metabolic flexibility, namely the ability of an organism to rapidly shift substrate oxidation rates to accommodate changes in substrate availability, and adipose tissue dysfunction have also been well documented.[23]

The lack of improvement in LDL-C values amongst young people with >1 documented measurement, and limited improvement in those with longitudinal data for ≥5 years, was also of concern. Analysis of ADDN data has previously highlighted that physicians are circumspect about treating raised LDL-C.[24] Findings suggest that those caring for young people with T1D need to increase their awareness of dyslipidaemia and its management to optimise cardiovascular risk reduction. Pregnancy counselling may be required in those being considered for statins.

We did not report prescription and determine use of cholesterol lowering therapy in this population. The benefit of statin therapy in deferring or preventing cardiovascular morbidity and mortality has been well documented. Importantly in people with familial hypercholesterolaemia, the initiation of statin therapy during childhood has slowed the progression of carotid intima-media thickness and reduced the risk of cardiovascular disease in adulthood.[4] International guidelines provide differing recommendations around statin use, often not specific to childhood and based predominantly on limited evidence from trials involving type 2 diabetes.[2,3,25,26] Hence, additional risk-stratification methods may assist in decision making, including T1D-specific risk calculators, such as the Steno T1 risk engine.[27] ISPAD guidelines indicate that if interventions to improve glycaemia, dietary changes and increased exercise do not lower LDL-C to <3.4 mmol/L (130 mg/dL), statins should be commenced in children from age 11 years.[3] In the T1D Exchange, statin use was 2% in 10–17 year olds and 4% in 18–24 year olds.[28] Elsewhere, lack of treatment for dyslipidaemia in young people with T1D has been reported.[29,30] Australian data relating to the prescription of statins are required and will be determined as the ADDN database expands.

Registry data may be incomplete: some young adults with T1D in Australasia are managed in the private sector or by General Practitioners, and these data are not

recorded in ADDN. Further, those at risk of hyperlipidaemia may have been more likely to be tested. These considerations should be taken into account in future dyslipidaemia analyses. Finally, multivariable GEE modelling without use of extensions around any missing data may have yielded biased results.

In conclusion, we demonstrate that both the proportion of Australasian youth with T1D screened for LDL-C, and the numbers of those with levels above recommended targets are suboptimal.[3] It is crucial to focus attention on this young age group since it is around this time that adult self care practices are developed. Our findings can inform discussion around how T1D healthcare services can support improved screening and LDL-C in this population.

AUTHOR CONTRIBUTIONS

All authors contributed to the study design and the manuscript. Data were analysed by S. James and M. E. Craig.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

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APPENDIX A

AUSTRALASIAN DIABETES DATA NETWORK (ADDN) STUDY GROUP MEMBERS

Dr Kym Anderson, University Hospital Geelong, Geelong; A/Prof Sof Andrikopoulos, Australian Diabetes Society; Clinical Professor Geoff Ambler, The Children's Hospital at Westmead, Sydney; Dr Helen Barrett, The Mater Private Hospital, Brisbane; Professor Jenny Batch, Queensland Children's Hospital, Brisbane; A/Prof Philip Bergman, Monash Children's Hospital, Melbourne; Prof Fergus Cameron, Royal Children's Hospital, Melbourne; A/Prof Louise Conwell, Queensland Children's Hospital, Brisbane; A/Prof Andrew Cotterill, Queensland Children's Hospital, Brisbane; Dr Chris Cooper, University Hospital Geelong, Geelong; Prof Jennifer Couper, Women's and Children's Hospital, Adelaide; A/Prof Elizabeth Davis, Perth Children's Hospital, Perth; Dr Martin de Bock, Christchurch Hospital, New Zealand; Dr Jan Fairchild, Women's and Children's Hospital, Adelaide; Dr Gerry Fegan, Fiona Stanley Hospital, Perth; A/Prof Spiros Fourlanos, Royal Melbourne Hospital, Melbourne; A/Prof Sarah Glastras, Royal North Shore Hospital, Sydney; Dr Peter Goss, Geelong, Victoria; Dr Leonie Gray, Mater Medical Centre, Rockhampton; A/Prof Peter Shane Hamblin, Western Health, Melbourne; Prof Paul Hofman, University of Auckland, New Zealand; A/Prof Dianne Jane Holmes-Walker, Westmead Hospital, Sydney; A/Prof Tony Huynh, Queensland Children's Hospital, Brisbane; Sonia Isaacs, University of New South Wales, Kensington, A/Prof Craig Jefferies, Starship Children's Health, New Zealand; Dr Stephanie Johnson, Queensland Children's Hospital, Brisbane; Clinical Prof Tim Jones, Perth Children's Hospital, Perth; Dr Jeff Kao, Royal Children's Hospital, Melbourne; A/Prof Bruce R. King, John Hunter Children's Hospital, Newcastle; Dr Antony Lafferty, The Canberra Hospital, Canberra; Jane Makin, Perth Children's Hospital, Perth; Ms Michelle Martin, Illawarra Shoalhaven Diabetes Service, Wollongong; Dr Robert McCrossin, Gladstone Hospital, Gladstone; Dr Kris Neville, Sydney Children's Hospital, Sydney; Dr Mark Pascoe, Royal Hobart Hospital, Hobart; Dr Ryan Paul, Waikato Hospital, New Zealand; Dr Dorota Pawlak, Juvenile Diabetes Research Foundation Australia; Dr Alexia Peña, University of Adelaide, Adelaide; Dr Liza Phillips, The Mater Private Hospital, Brisbane; A/Prof Darrell Price, Pacific Private Clinic, Gold Coast; A/Prof Christine Rodda, Sunshine Hospital, Melbourne; Prof David Simmons, Campbelltown Hospital, Sydney; Prof Richard Sinnott, eResearch, University of Melbourne; Dr Carmel Smart, John Hunter Children's Hospital, Newcastle; Anthony

Stell, University of Melbourne, Dr Monique Stone, Royal North Shore Hospital, Sydney; A/Prof Steve Stranks, Australian Diabetes Society; Dr Elaine Tham, Women's and Children's Hospital, Adelaide; Barbara Waddell, University of New South Wales, Sydney, A/Prof Glenn Ward, St. Vincent's Hospital, Melbourne; A/Prof Ben Wheeler, Dunedin School of Medicine, New Zealand; Dr Helen Woodhead, Australasian Paediatric Endocrine Group; and Dr Anthony Zimmermann, Lyell McEwin Hospital, Elizabeth Vale.