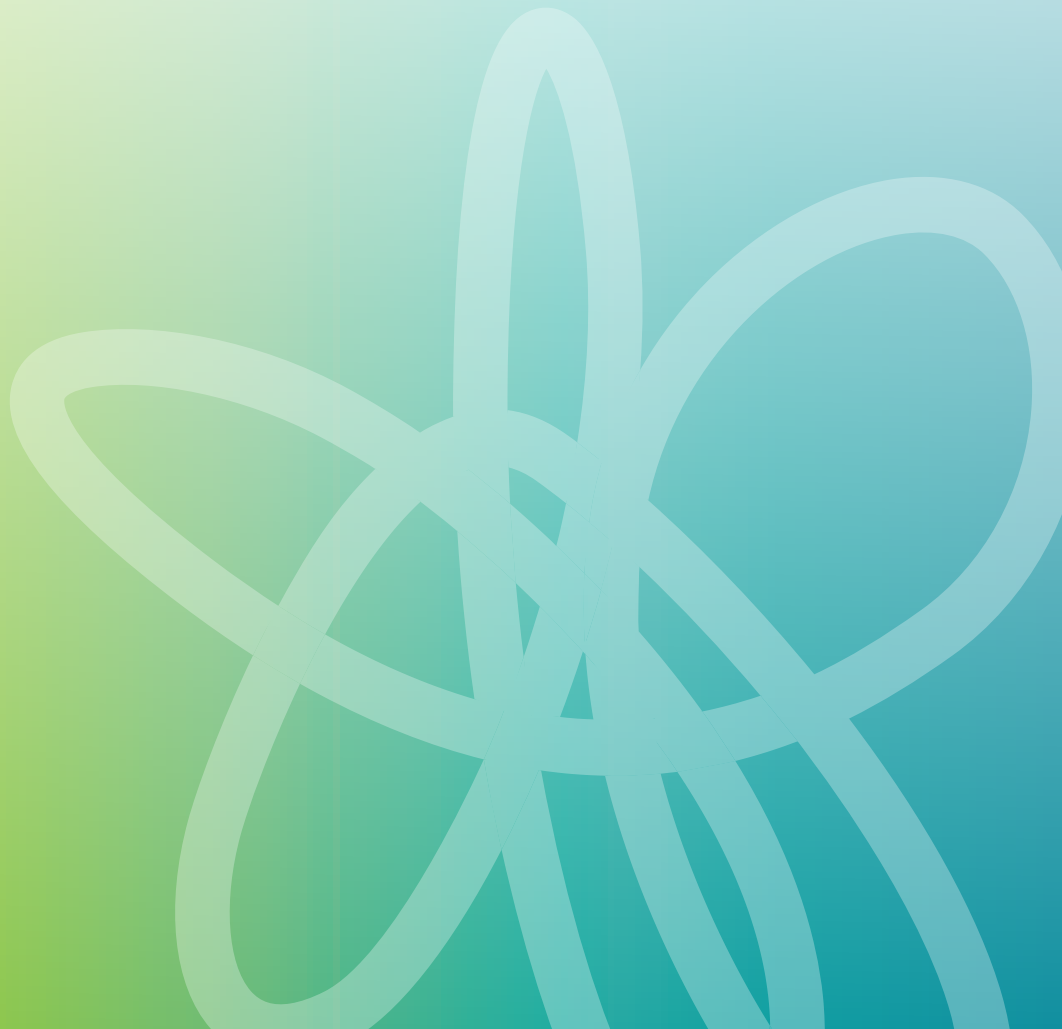The Australian Prevention Partnership Centre
Systems and solutions for better health

A rapid review of evidence

Prevention of type 2 diabetes and the implementation of large-scale prevention programs

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Prepared by: The Australian Prevention Partnership Centre and The Boden Institute of Obesity, Nutrition, Exercise & Eating Disorders

Contributing authors:

The Australian Prevention Partnership Centre
Professor Andrew Wilson
Dr Jo-An Atkinson
Ms Emily Morrice
Ms Eloise O'Donnell

The Boden Institute of Obesity, Nutrition, Exercise & Eating Disorders
Dr Crystal Lee
Professor Stephen Colagiuri

Editor: Ms Marge Overs

Disclaimer: This evidence review is not necessarily a comprehensive review of all literature relating to the topic area. It was current at the time of production (but not necessarily at the time of publication) and is based on sources believed to be reliable.

Contents

Glossary[1]

CI   Confidence interval

A measure of the uncertainty around the main finding of a statistical analysis. Estimates of unknown quantities, such as the odds ratio comparing an experimental intervention with a control, are usually presented as a point estimate and a 95% confidence interval. This means that if someone were to keep repeating a study in other samples from the same population, 95% of the point estimates from those studies would lie within the confidence limits.

HR   Hazard ratio

A measure of effect produced by a survival analysis. This represents the increased risk with which one group is likely to experience the outcome of interest. For example, if the hazard ratio for death for a treatment is 0.5, we can say that treated patients are likely to die at half the rate of untreated patients.

IRR   Incident rate ratio

The ratio of two incident rates, comparing the rate of an event occurring over time between two groups.

OR   Odds ratio

The ratio of the odds of an event in one group to the odds of an event in another group. In studies of treatment effect, the odds in the treatment group are usually divided by the odds in the control group. An odds ratio of one indicates no difference between comparison groups.

RR   Relative risk or risk ratio

The ratio of risks in two groups. In intervention studies, it is the ratio of the risk in the intervention group to the risk in the control group. A risk ratio of one indicates no difference between comparison groups. For undesirable outcomes, a risk ratio that is less than one indicates that the intervention was effective in reducing the risk of that outcome.

[1] These terms, with the exception of IRR, have been adapted from the Cochrane Community (beta) Glossary.

1. Executive summary

Diabetes is a crucial health issue for Australia. More than 10% of Australians have diabetes or are at high risk. The burden of diabetes is compounded because many complications of diabetes can be debilitating or even life threatening, such as cardiovascular disease, end-stage kidney disease, loss of vision, amputations and mental health issues. This review collates the evidence on diabetes prevention, highlights the key modifiable risk factors to target, and analyses what system-level changes are needed to successfully implement a population-wide diabetes prevention program.

Method

The scope of this review was determined through consultation with experts on areas of critical importance to inform an Australian national diabetes strategy. Searches were done in August and September 2014 from PubMed and other online databases. The searches were done iteratively and included hand searching of reference lists, and experts gave feedback of areas requiring further research. For the identification of evidence around diabetes risk factors, meta-analyses and systematic reviews were targeted during the searches, but in some cases individual controlled studies were included. Searches for past lifestyle interventions and the scaling up of interventions were broader and included grey literature such as program evaluation reports.

Risk factors for type 2 diabetes

Overweight and obesity are consistently demonstrated to be major risk factors for type 2 diabetes. While there is substantial evidence that physical activity is a protective factor against diabetes, it is less clear whether being physically inactive (sedentary behaviour) increases the risk of developing diabetes. The risk of type 2 diabetes increases with age and is more common in men than women. Other risk factors for type 2 diabetes include family history and early life factors such as high maternal weight, maternal smoking, and a high birthweight, while breastfeeding protects against diabetes.

Aboriginal and Torres Strait Islander people, people from the Pacific Islands and some other ethnic groups such as South Asians have a high prevalence of diabetes. Aboriginal and Torres Strait Islander people are over three times more likely to develop diabetes than non-Indigenous Australians.

There is suggestive but not conclusive evidence that depression and other mental health disorders may increase the risk of developing type 2 diabetes. This may be in addition to the adverse effect of some anti-psychotic medications on weight gain and glucose metabolism.

Progression from high risk to diabetes

There is evidence (NHMRC Evidence level B) that a person who has impaired glucose metabolism (pre-diabetes) is at risk of developing type 2 diabetes. The rate of progression to diabetes has been shown to vary according to the level of impairment of a person's glucose metabolism at initial examination but is also influenced by other factors such as weight, family history and ethnicity.

Diabetes can be prevented or delayed

There is a substantial body of evidence (NHMRC Evidence Level A) that diabetes can be prevented or delayed. Several large-scale diabetes prevention programs have demonstrated an ability to prevent or delay diabetes, including:

• The Diabetes Prevention Program in the United States found diabetes incidence reduced by 58% in the lifestyle intervention group and 31% in the metformin intervention group compared with the control group (Knowler et al. 2002).

• The Diabetes Prevention Program Outcomes Study in the United States found that, over a period of 10 years, people who achieved normal glucose regulation at least once, even if transient, during the intervention had a lower risk of developing diabetes than those who did not achieve normal glucose regulation (Knowler et al. 2009).

• The Finnish Diabetes Prevention Study found over a period of 13 years an absolute risk reduction of 19% in the lifestyle intervention group compared with the control group (Lindstrom et al. 2013).

People with impaired glucose tolerance may be more responsive to interventions

There is some evidence that people with impaired or abnormal glucose tolerance at baseline have the greatest improvement during interventions. However, available evidence does not suggest that the effect of a diabetes prevention intervention differs systematically according to a person's sex, race or ethnicity.

Little is known about what characteristics make an intervention successful

There is limited information directly evaluating the impacts of the intensity or duration of an intervention on diabetes risk. The available evidence suggests that the intensity of an intervention does not significantly determine the outcome. Further, prevention programs around the world have had success using various levels of intensity. Evaluations of population-wide diabetes prevention trials have not fully captured the characteristics of high- and low-level participants. However, being recruited from an occupational setting, and certain lifestyle attributes such as being married or living with a partner, were associated with a high level of participation, as was frequent contact with participants.

Initiatives can be scaled up, but key considerations affect success

If an intervention is to be successfully scaled up from trial size to population size, the following key considerations should be addressed:

• Effectiveness
• Potential reach and adoption
• Alignment with strategic content
• Acceptability and feasibility.

Existing models can be used for planning large-scale interventions

At least seven logic models have been used to plan and evaluate large-scale type 2 diabetes programs. While there are differences between the models, there were similarities in their identification of inputs and outcomes.

System-level factors need to be considered when implementing large-scale programs

The following factors need to be considered when implementing large-scale programs:

• Service delivery
• Workforce
• Training and education
• Governance and leadership
• Financing
• Infrastructure.

Conclusion

There is substantial evidence that type 2 diabetes can be prevented and delayed, particularly in certain high-risk groups, and this has been successfully demonstrated in prevention programs in a range of settings. There is also evidence of type 2 diabetes prevention interventions being successfully scaled up and implemented population-wide. The system-level factors needed to successfully scale up an intervention are known and should be considered when planning large-scale diabetes prevention programs. However, while it is clear that large-scale prevention interventions can be effective, further research is needed to determine which characteristics are needed to maximise the efficacy of an intervention.

2. Introduction

Diabetes is currently responsible for 2.31% of the burden of disease in Australia, of which more than 90% is attributable to type 2 diabetes (IHME 2013). The Australian Health Survey 2011–12 estimated that, based on haemoglobin A1c levels, 5.2% of people had diabetes and a further 5.3% of people were at high risk (Table 1.3, ABS 2013a).

Preventable diabetes complications include:

• Cardiovascular complications: three in five people with diabetes also have cardiovascular disease (AIHW 2014)

• Kidney damage: diabetes caused one in three new cases of end-stage kidney disease in 2011 (AIHW 2014)

• High blood pressure: is nearly four times more likely in people with diabetes (AIHW 2013)

• High cholesterol: is 4.6 times more likely in people with diabetes (AIHW 2013)

• Blindness: loss of sight is twice as likely in people with diabetes (AIHW 2013)

• Amputations: diabetes is the leading cause of lower limb amputations (AIHW 2013)

• Depression, anxiety and distress: people with diabetes are three times more likely to experience depression (AIHW 2013).

Preventing the development of diabetes, early diagnosis, optimal treatment and effective ongoing monitoring and self-management reduce the risk of diabetes-related complications.

This rapid review was prepared to summarise evidence on the prevention of type 2 diabetes particularly around reducing the risk of progression in individuals and groups at higher risk of diabetes. It is not a systematic review of the literature but draws together the best evidence available. The initial search was for systematic reviews or meta-analyses relevant to the specific questions. If none was identified, the most recent comprehensive review was sought. If no such reviews were identified, studies of the best quality evidence for the specific question were sought.

The structure of the review was guided by questions identified as important to inform a national diabetes strategy, and broadly follows the following structure:

• Risk factors for type 2 diabetes

• Natural history of progression from high risk to diabetes

• Evidence that progression can be prevented or delayed

• Attributes of successful prevention interventions

• Characteristics of individuals that predict response

• Relationship between duration and intensity of intervention and prevention

• Issues in scaling up of interventions from trial size to population size

• Examples of the logic models of large-scale interventions

• System-level factors that need to be considered in implementation of large-scale programs.

3. What do we know about the relative contributions of risk factors to type 2 diabetes risk?

Type 2 diabetes accounts for at least 85% of all diabetes mellitus. The risk factors for type 2 diabetes mellitus have been studied in many different countries, populations and time periods, and the main risk factors are fairly consistent in all settings.

4. Demography and diabetes risk

Age

In the Australian population and many others the risk of diabetes increases with adult age in both cross-sectional data and cohort studies. This was most recently shown in the Australian Diabetes, Obesity and Lifestyle Study (AusDiab), which followed 5842 participants without diabetes over five years. Multivariate adjusted odds ratio (OR) for incident diabetes was 1.82 (1.30, 2.52) for family history of diabetes; 1.12 (0.97, 1.29) for age per 10 years; and 1.22 (0.89, 1.68) for female sex (Magliano et al. 2008).

The effect of age has been further quantified in the broader AusDiab study, which followed 11,247 participants aged ≥25 years at baseline in 2000. It showed an increasing prevalence of known and undiagnosed diabetes (and prevalence of impaired glucose tolerance, IGT) with increasing age. For impaired fasting glucose (IFG), the prevalence peaked in the 55–64 years age group. The Busselton study comprises a cohort from a single community in WA followed since the mid-1960s. Using the combined Busselton (1981) and AusDiab data, the odds of diabetes (known and newly diagnosed diabetes) adjusted for other factors increased by 1.07 (1.06, 1.07) per life year (Dunstan et al. 2002).

Gender

In the 2011–12 Australian Health Survey, the prevalence of diabetes was higher in men than in women (6.3% compared with 3.9%) (ABS 2013a). This was the case for both known diabetes (4.9% compared with 3.4%) and newly diagnosed diabetes (1.4% compared with 0.4%). Findings in other studies in relation to gender differences in the prevalence of diabetes within populations have been variable, but less so once adjustment is made for age and body mass index (BMI) (Minges et al. 2011; Li et al. 2012; Coppell et al. 2013).

High-risk groups

Diabetes is more prevalent in certain non-Caucasian ethnicities and some geographically defined populations such as among urban residents (although this may be changing). According to the Diabetes Atlas, the only region with a lower diabetes prevalence than Europe (7.9%) is Africa (5.1%) (IDF 2014).

Aboriginal and Torres Strait Islanders

Indigenous populations have been reported to have higher prevalence of diabetes than their non-Indigenous counterparts in countries such as Australia, Canada, New Zealand and the United States.

In Australia, a systematic review on diabetes prevalence in Indigenous populations showed that most studies reported higher prevalence than AusDiab reported for the general Australian population (Minges et al. 2011). Furthermore, studies that have included both Aborigines and Torres Strait Islanders, two ethnically distinct Indigenous groups in Australia, reported higher diabetes prevalence in Torres Strait Islanders. The Australian Aboriginal and Torres Strait Islander Health Survey: Biomedical Results 2012-13 found that more than one in 10 (11.1%) Aboriginal and Torres Strait Islander adults had diagnosed diabetes, and a further 4.7% were found to have a high risk of developing diabetes (ABS 2014). The prevalence of diabetes increased with age and was twice as high among those living in remote compared with urban areas. After adjusting for differences in age structure between the two populations, Aboriginal and Torres Strait Islander people were more than three times as likely as non-Indigenous people to have diabetes/high sugar levels (rate ratio of 3.3). The 2012–13 Australian Aboriginal and Torres Strait Islander Health Survey: First Results (ABS 2013b) found that females were significantly more likely than males to have diabetes/high sugar levels (10% compared with 7%). Prevalence of diabetes in both males and females had increased significantly from 6% to 8% between the 2001 and 2012–13 surveys, and there were statistically significant differences between the age-standardised rates for females (rate ratio of 4.3) and males (rate ratio of 2.6).

The finding of higher prevalence of type 2 diabetes among Indigenous populations is common across developed countries. Age-standardised prevalence differed between the various native groups in Canada; 17.2% for First Nations people living on reserve, 10.3% for First Nations people living off reserve and 7.3%

for the Metis Indigeous people compared with 5.0% for non-Indigenous people in 2008–09 (Pelletier et al. 2012). For New Zealand, the prevalence of diabetes for people aged ≥15 years in 2008–09 was 6.1% and 9.8% respectively for New Zealand European and other ethnic groups except for Pacific Islander and Maori (Indigenous people of New Zealand) (Coppell et al. 2013).

Globally, the highest recorded prevalence was the Pima Indians, an Indigenous group in the United States. A study conducted between 1965 and 1969 reported the prevalence of diabetes based on a two-hour glucose level ≥11.1 mmol/L or previously diagnosed diabetes was 14.7% in Pima Indians aged ≥5 years and 41.0% in those aged ≥35 years (Bennett et al. 1971). In contrast, the national prevalence estimated by the International Diabetes Federation for the United States in 2011 was only 10.9% for those aged 20–79 years. Although the incidence of type 2 diabetes has remained stable between 1965 and 2003 for the Pima Indians, both incidence and prevalence of type 2 diabetes have increased in children aged 5–14 years but decreased or stabilised in those 25 years and older (Pavkov et al. 2007).

Pacific Islanders

Diabetes prevalence is high in some Pacific Islander populations. The latest Diabetes Atlas reported that Tokelau, a Pacific Island nation, had the highest prevalence at 37.5% (IDF 2013). Other Pacific Island countries among the top 10 highest prevalence were Federated States of Micronesia (ranked 2nd), Marshall Islands (3rd), Kiribati (4th), Cook Islands (5th), Vanuatu (6th) and Nauru (8th). Nauru has long been known as a country with a very high prevalence of diabetes. The Diabetes Atlas 2012 update estimated Nauru to have the second highest diabetes prevalence in the world (IDF 2012). A study conducted in 1975 in Nauru reported similar prevalence to the Pima Indians (Zimmet et al. 1977). More recent data on Nauru suggested a declining trend in diabetes, but the proportion of undiagnosed diabetes has increased in comparison with the 1975 study (Khambalia et al. 2011).

In New Zealand, diabetes prevalence for Pacific Islanders in 2008–09 was 15.4%, which is 2.5 times the prevalence for New Zealand European and other ethnic groups (Coppell et al. 2013). Since Torres Strait Islanders have ancestral links to Pacific Islanders, it may explain the high diabetes prevalence compared with the general Australian and Aboriginal populations.

South Asians

The risk of diabetes is higher in South Asians compared with Caucasians of the same weight status. Diabetes also occurs at a much younger age in this population. South Asia has three out of 10 most populous countries in the world (India, Pakistan and Bangladesh), leading it to have the second highest number of people with diabetes in the world after the Western Pacific region. This is despite the actual prevalence of diabetes in South Asia being not as high as in North America and Caribbean, or the Middle East and North Africa (IDF 2013).

A systematic review on the prevalence of diabetes in South Asia reported a lack of national data for most South Asian countries (Jayawardena et al. 2012). However, studies on urban and rural populations in the region have all shown an increase in diabetes prevalence over time. The prevalence of diabetes ranged from 2.2% in 1989 to 9.2% in 2005 for rural India and from 8.3% to 18.6% for urban India over the same period. A steeper increase, from 2.3% in 1999 to 6.8% in 2004, was observed for urban Bangladesh (Rahim et al. 2007; Jayawardena et al. 2012).

Moreover, South Asians are reported to have higher diabetes prevalence in multi-ethnic populations such as Malaysia, where the three major ethnicities are Malays, Chinese and Indians, and England, where South Asians are the largest ethnic group after Whites (Office for National Statistics 2012). In Malaysia, a 2006 national survey reported the prevalence of diabetes for adults aged ≥18 years was 19.9% for Indians, 11.9% for Malays and 11.4% for Chinese (Letchuman et al. 2010). In England, diabetes is six times more common in people with South Asian background compared with the general population (Diabetes UK 2012).

5. Family and genetic history

While it is evident that there are genetic markers associated with increased risk of type 2 diabetes, these appear to account for only a small amount of the observed variance in risk and at this time serve little value in risk prediction.

A systematic review on the predictive performance of genetic risk models for predicting risk of type 2 diabetes reported that adding genetic markers into conventional risk factor-based models did not appreciably improve the predictive performance. These findings occurred even though genetic markers have been widely demonstrated to have an association with type 2 diabetes risk (Bao et al. 2013). One of the probable reasons for this is that it is difficult to disentangle the impacts of genetics from that of shared family environments, pre-conception and in-utero exposures. Consequently, prevention research has focused more on these latter factors because they may be more amenable to change.

Early life factors

There is a strong and growing body of research indicating that early life factors influence the risk of adult diabetes. Some of the more important factors include:

Maternal BMI

The body mass of the biological mother has been shown to be a strong risk factor for the risk of diabetes in their children in several studies. The Cardiovascular Risk in Young Finns Study followed 1935 participants aged 3–18 years at baseline for a mean of 23.8 years. Mother's BMI and offspring's systolic blood pressure at baseline were independent predictors of adult type 2 diabetes. The adjusted OR for 1 standard deviation (SD) increase in mother's BMI was 1.54 (1.12, 2.11) and in the offspring's systolic blood pressure was 1.54 (1.01, 2.35) (Juonala et al. 2013).

Maternal smoking

Another maternal exposure associated with increased risk of diabetes in offspring is tobacco smoking. In the Nurses' Health Study II, of 34,453 participants, 25.9% reported that they smoked during the pregnancy. Daughters of mothers who smoked during pregnancy were born with lower birthweight, were less frequently breastfed, were more often smokers in adulthood, and had a higher alcohol intake in adulthood when compared with daughters of mothers who did not smoke during pregnancy. The hazard ratio (HR) for smoking during the first trimester only of pregnancy was 1.34 (1.01, 1.76) when compared with no smoking during pregnancy, after being adjusted for variables (age, perinatal variables, adult life variables, birth weight, BMI at age 18, current BMI). The associations between type 2 diabetes risk and maternal continued smoking of <15 cigarettes per day, maternal continued smoking of ≥15 cigarettes per day, paternal smoking <15 cigarettes per day and paternal smoking ≥15 cigarettes per day were not significant (Jaddoe et al. 2014).

Birthweight

The relationship between birthweight and risk of diabetes is complicated. A study identified participants born in 1973–1982 through the Swedish Medical Birth Register (n=759,999) and followed to ages 27.5–37.5 years. It reported the adjusted risk (measured as HR) for diabetes was 1.91 (1.25, 2.90) for men with moderately high birthweight and 5.44 (2.70,10.96) for men with very high birthweight compared with men whose birthweight was appropriate for gestational age. After adjusting for obesity, the HR reduced to 1.38 (0.69, 2.78) and 4.62 (2.28, 9.33), respectively. For women, a moderately high birthweight had a reduced risk of diabetes (0.60 (0.40, 0.91)) and a very high birthweight had a statistically non-significant association (1.71 (0.85, 3.43)) (Johnsson et al. 2014).

In the Women's Health Initiative Observational Study (n=75,993), multivariate adjusted OR for type 2 diabetes was 1.16 (1.01, 1.33) for those with birthweight <6lbs, and 0.72 (0.57, 0.92) for those with birthweight ≥10lbs (4.5kg) compared with those with birthweight 7–8lbs (3.2–3.6kg). When stratified by ethnicity, age- and BMI-adjusted OR was significant for White and Black ethnicity with birthweight <6lbs (2.7kg). When OR was further adjusted for neighbourhood socioeconomic status, only White ethnicity with birthweight <6lbs remained significant (1.35 (1.17, 1.55)) (Ryckman et al. 2014).

A systematic review on birthweight and type 2 diabetes, which included 31 populations (n=152,084), reported pooled age- and sex-adjusted OR of type 2 diabetes per 1kg increase in birthweight of 0.80 (0.72, 0.89). However, there was heterogeneity between populations (I2=66%). Heterogeneity was reduced when two native North American studies (in Arizona and Saskatchewan), and one young adults study (from Saskatchewan general population), were removed (I2=20% age- and sex-adjusted OR=0.75 (0.70, 0.81)). The three removed studies showed associations with positive birthweight and type 2 diabetes. Analysis of 16 studies with individual participant data showed a graded inverse association between birthweight and type 2 diabetes risk. For birthweights >3.5kg, however, the ORs for diabetes were not significant (Whincup et al. 2008).

Breastfeeding

Mothers who breastfeed appear to have a reduced risk of subsequent diabetes. The EPIC-Potsdam study included 1262 female participants aged 35–64 years at baseline. Multivariate adjusted (lifestyle and reproductive factors, BMI, WC) HR for type 2 diabetes was 0.47 (95%CI 0.25, 0.89) for women who had breastfed for ≥6 months compared with those who had never breastfed. When including three other prospective studies (total n=220,360) in the meta-analysis, the pooled HR for lifetime breastfeeding duration of 6–11 months was 0.89 (0.82, 0.97) and for >11–23 months was 0.88 (0.81, 0.96) compared with no breastfeeding (Jager et al. 2014).

Childhood determinants of adult overweight and obesity

Studies of early determinants of adult obesity are relevant because increased adult BMI is a strong risk factor for type 2 diabetes. A review of systematic reviews on the early determinants of overweight and obesity found that the factors included maternal factors, birthweight, infant size and growth, infant feeding, sleep duration, family, physical activity and sedentary behaviour, and society and built environment. The authors concluded that having been breastfed may reduce the risk of becoming overweight and obese later in life.

Obesity in infancy, rapid infant growth, maternal diabetes, maternal smoking, short sleep duration, less than 30 minutes of daily physical activity, and consumption of sugar-sweetened beverages may be considered as risk factors. Furthermore, parental obesity, restricted or excessive intrauterine growth, small or large size at birth, TV viewing, food insecurity and low socioeconomic status may also be included as risk factors (Monasta et al. 2010).

6. Later modifiable lifestyle and environmental factors

Overweight and obesity

Increased BMI and other measures of obesity have consistently been shown to be associated with increased risk of type 2 diabetes. A meta-analysis of 18 prospective studies (n=590,251) reported the pooled risk (measured as relative risk (RR)) of type 2 diabetes in those who were obese compared with those who were normal weight was 7.19 (5.74, 9.00).

However, there was heterogeneity between studies (I2=89%). After removing six poor-quality studies, which accounted for the heterogeneity, the pooled RR was 7.28 (6.47, 8.28). The pooled RRs for those who were overweight compared with those who were normal weight were 2.99 (2.42, 3.71) for all studies and 2.92 (2.57, 3.32) after excluding poor-quality studies (Abdullah et al. 2010). A meta-analysis of 15 cohorts from 10 studies reported the pooled OR for abdominal obesity and type 2 diabetes was 2.14 (1.70, 2.71).

The measures used for abdominal obesity included waist circumference, waist-to-hip ratio, iliac circumference and intra-abdominal fat area (Freemantle et al. 2008).

Physical activity and sedentary behaviour

Increased physical activity has consistently been shown to be associated with decreased risk of type 2 diabetes, but the evidence is less clear in relation to sedentary activity. A meta-analysis of 10 cohort studies (n=301,221) suggested that the pooled BMI-adjusted RR for type 2 diabetes was 0.83 (0.76, 0.90) for the highest compared with the lowest category of moderate-intensity physical activity (Jeon et al. 2007). A meta-analysis of 10 studies (n=482,117) reported the pooled baseline event rate adjusted RR for diabetes was 2.12 (1.61, 2.78) for the highest level of sedentary behaviour compared with the lowest level of sedentary behaviour. However, there was publication bias (p≤0.001). The result remained unchanged after further adjustment to account for publication bias (RR 2.12 (1.61, 2.78)) (Wilmot et al. 2012).

A systematic review of prospective studies on sedentary behaviour and health outcomes concluded that "there is moderate evidence for a significant positive relationship between the time spent sitting and the risk for type 2 diabetes". However, this conclusion is based on two studies with less reliable measures of sedentary behaviour: the Health Professionals' Follow-up Study and the Nurses' Health Study (Proper et al. 2011).

Nutrition

The relationship of food and nutrition to risk of diabetes is complex and difficult to disentangle from the effects of over-nutrition (ie. energy intake in excess of energy expenditure). Ley et al. (2014) conducted a review on prevention and management of type 2 diabetes focusing on dietary components and nutritional strategies. The summary results of the meta-analyses of prospective cohort studies of type 2 diabetes and nutrient intake, glycaemic variables and food and beverage intake are shown in Figure 1. Interpretation of the actual dietary levels is difficult as studies used different measures and cut-points when estimating risk. Where cut-points are defined by highest and lowest quintiles (or similar); these can vary depending on the population.

Figure 1: Summary of nutrient intake and glycaemic variables, and food and beverage intake, and type 2 diabetes

Nutrients	Number of cohorts	Relative risk (95% CI)
Haeme-iron	4	
Glycaemic index	13	
Glycaemic load	17	
DHA/EPA	16	
Vegetable fibre	7	
Fruit fibre	8	
Alpha-linolenic acid	7	
Magnesium	13	
Cereal fibre	8	
Vitamin D	21	
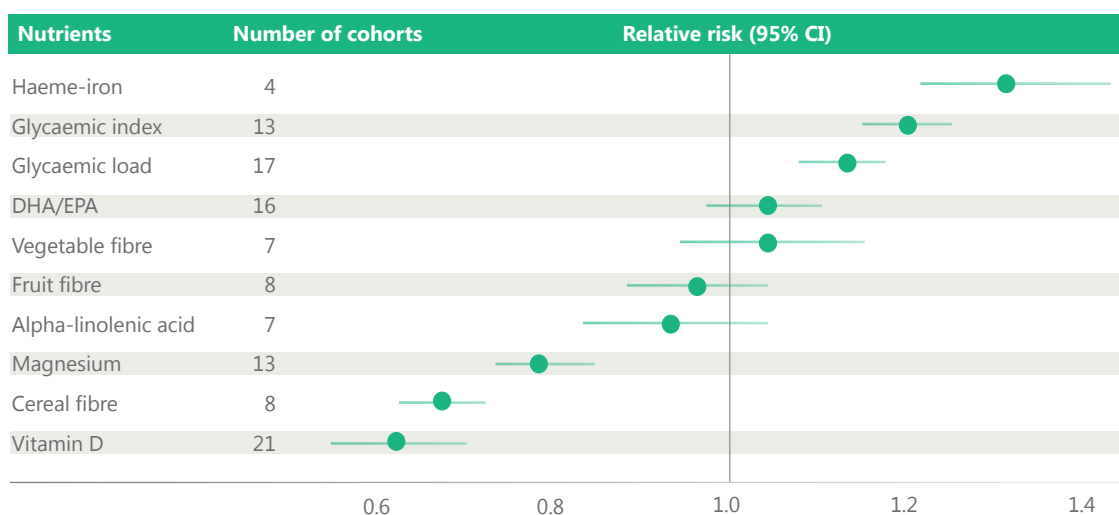
Figure 1: Summary of meta-analyses of prospective cohort studies of nutrient intake and glycaemic variables and type 2 diabetes (Ley et al. 2014)

DHA=docosahexaenoic acid. EPA=eicosapentaenoic acid. Relative risks are a comparison of extreme categories, except for DHA/EPA (per 250mg per day increase) and alpha-linolenic acid (per 0.5g per day). All nutrients and glycaemic variables were assessed from dietary intake, except vitamin D for which blood 25-hydroxyvitamin D was used.

Figure 2: Summary of meta-analyses of prospective cohort studies on food and beverage intake and type 2 diabetes

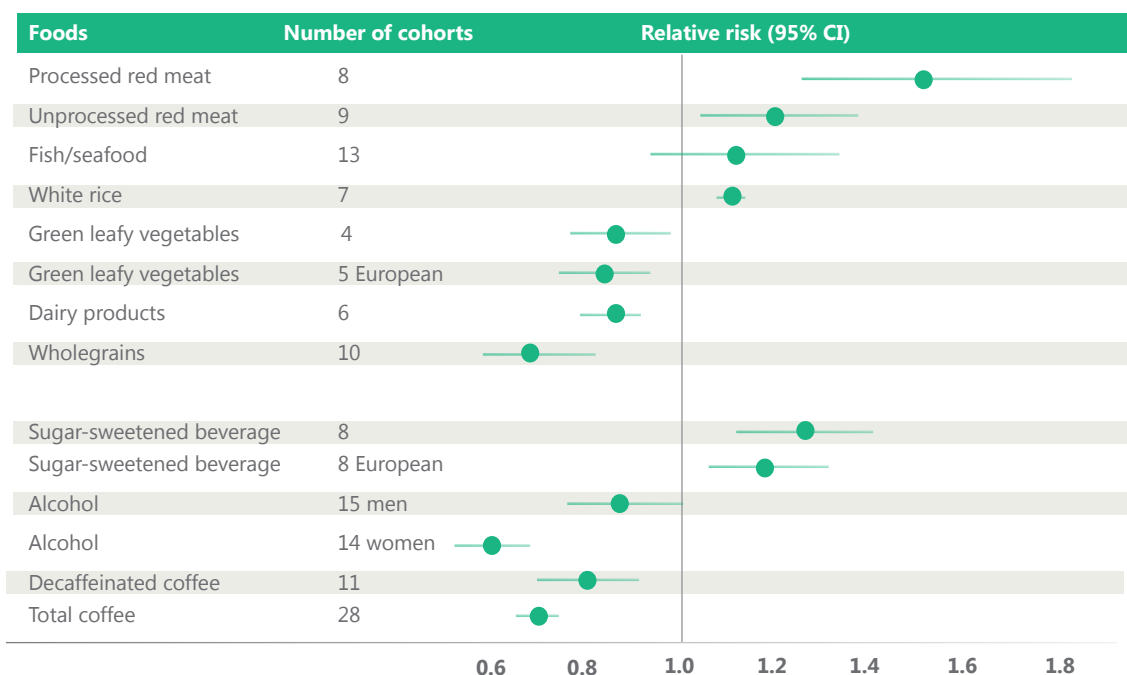| Foods	Number of cohorts	Relative risk (95% CI)
Processed red meat	8	
Unprocessed red meat	9	
Fish/seafood	13	
White rice	7	
Green leafy vegetables	4	
Green leafy vegetables	5 European	
Dairy products	6	
Wholegrains	10	
Sugar-sweetened beverage	8	
Sugar-sweetened beverage	8 European	
Alcohol	15 men	
Alcohol	14 women	
Decaffeinated coffee	11	
Total coffee	28	

0.6 0.8 1.0 1.2 1.4 1.6 1.8

Figure 2: Summary of meta-analyses of prospective cohort studies on food and beverage intake and type 2 diabetes (Ley et al. 2014)

Relative risks are a comparison of extreme categories, except for processed meat (per 50p per day increase), unprocessed red meat and fish or seafood (per 100g per day), white rice (per each serving per day), wholegrains (per three servings per day), sugar-sweetened beverages in European cohorts (per 336g per day), and alcohol (22g per day for men or 24g per day for women with abstainers).

Micronutrient deficiencies

There have been many studies of different micronutrients and risk of diabetes with inconsistent results even within the same nutrients. These inconsistent results may reflect variation in assessment methods and the difficulty of assessing micronutrients. The most consistent finding to date has been a relationship between increased risk and lower vitamin D levels. A meta-analysis of 14 prospective studies (n=190,626) suggested that the pooled RR for type 2 diabetes was 0.81 (0.71, 0.92) for top-third baseline vitamin D level compared with bottom-third baseline vitamin D level. However, there was heterogeneity (I2=67%) and publication bias (p=0.006) (Khan et al. 2013). The systematic review of vitamin B12 and cardiovascular disease and diabetes found that only one study (Pima Indian study) reported the association between each 100 pg/mL difference in vitamin B12 and diabetes/nephropathy mortality (HR: 1.28 (1.12, 1.53)) (Rafnsson et al. 2011).

Tobacco and alcohol

Tobacco smoking and tobacco smoke exposure is associated with a higher risk of diabetes. The relationship of alcohol intake with risk of diabetes appears to be similar to the relationship with cardiovascular disease, that is, non-drinking and high levels of alcohol consumption are associated with an increased risk.

A meta-analysis of 25 prospective cohort studies (n=1.2 million) reported the pooled RR for type 2 diabetes was 1.44 (1.31, 1.58) for current smokers compared with non-smokers. There was a greater risk of diabetes for heavy smokers (≥20 cigarettes per day; RR 1.61 (1.43, 1.80)) than for light smokers (<20 cigarettes per day; 1.29 (1.13, 1.48)); and a lower risk of diabetes for former smokers (1.23 (1.14, 1.33)) compared with active smokers (1.44 (1.31, 1.58)) (Willi et al. 2007).

A meta-analysis of four prospective cohort studies (n=112,351) reported the pooled RR for type 2 diabetes was 1.28 (1.14, 1.44) for those exposed to passive smoking compared with those not exposed to passive smoking (Wang et al. 2013b).

A review of review articles (including meta-analyses, reviews and observational studies) reported a J-shaped association between alcohol intake and type 2 diabetes incidence. (In a J-shaped association, risk levels are lower for low consumption than for no consumption, and are highest for high consumption.) The review also reported an association between binge drinking and type 2 diabetes (Pietraszek et al. 2010).

Sleep quality and quantity, sleeping disorders

There is a growing body of research finding causal relationships between the length and quality of sleep and a number of chronic diseases including type 2 diabetes. This relationship is strongest for sleep disorders such as obstructive sleep apnoea. A meta-analysis of six prospective cohort studies (n=5953) reported the pooled RR for type 2 diabetes was 1.63 (1.09, 2.45) for those with moderate-severe obstructive sleep apnoea compared with those without obstructive sleep apnoea. For those with mild obstructive sleep apnoea, the pooled RR was 1.22 (0.91, 1.63) compared with those without obstructive sleep apnoea (Wang et al. 2013a).

Depression and other mental health disorders

A systematic review on depression and diabetes concluded that there are not enough prospective studies to definitively rule in a relationship between depression and diabetes (Roy and Lloyd 2012).

The Whitehall II Cohort Study, which included 5932 diabetes-free participants with three five-year follow-ups (total 13,207 person-observations), reported no significant association between psychological distress and type 2 diabetes after adjusting for age, sex, socioeconomic status and ethnicity (OR 1.16 (0.94, 1.42)). Among participants at high risk of developing type 2 diabetes and high Framingham Risk Score (>19) at baseline, significantly more of those with psychological distress developed diabetes at follow-up compared with those without psychological distress (40.9% vs. 28.5%; multivariate adjusted OR 2.07 (1.19, 3.62)) (Virtanen et al. 2014).

There is a strong body of evidence that some major and commonly used anti-psychotic drugs are associated with significant weight gains, metabolic syndrome and early onset type 2 diabetes. Depression (Kan et al. 2013) and anti-depressant medications (Barnard et al. 2013) have been associated with changes in insulin resistance and possibly higher risk of diabetes, but a causal association is not clear on the basis of current evidence.

Environmental chemicals

An updated systematic review reported insufficient evidence to infer a causal association between some environmental chemicals and diabetes outcomes. The current evidence available is only suggestive of an association. It was noted that there were few high-quality prospective studies (Kuo et al. 2013).

Other factors

Other factors that have been reported as associated with higher risk of diabetes but where the evidence is still exploratory include the presence of spousal diabetes (Leong et al. 2014), job strain (Huth et al. 2014) and periodontal disease (Bascones-Martinez et al. 2014).

7. What do we know about the natural history of progression, from having a high risk of developing diabetes to diabetes?

It is generally agreed that without intervention most people at high risk of developing type 2 diabetes (as defined by abnormal but not diabetic levels of fasting plasma glucose or equivalent) will progress to diabetes in the long term, although the number of long-term prospective studies (the only way to adequately assess this) are limited (NHMRC Evidence level B).

The available research suggests that the rate of progression is variable and depends on the level of glucose metabolism disturbance at the time of evaluation. Moreover other factors, in particular ethnicity and increasing weight, can modify the rate of progression.

In AusDiab, among the 5842 participants without diabetes at baseline, the age- and sex-standardised incidence of diabetes was 0.2% per year for men with normal glucose tolerance (NGT) at baseline, 2.0% per year for men with impaired fasting glucose (IFG) at baseline and 4.4% per year for men with impaired glucose tolerance (IGT) at baseline. For women, the incidence of diabetes was 0.2% per year, 4.0% per year and 2.9% per year respectively.

Notably there appears to be a higher incidence for men with IGT but a higher incidence for women with IFG at baseline. This apparent difference is explained in the discussion: "It appears that although more women had IGT (at baseline), men with IGT were more likely to convert to diabetes. In contrast, while more men had IFG, women with IFG were more likely to convert to diabetes. While this may appear counterintuitive, it is explained by sex differences in glucose levels. Among those with IGT, men have a higher fasting plasma glucose, leading to a higher risk of progressing to diabetes. Among those with IFG, women have a higher two-hour post-load plasma glucose, leading to a higher risk of progression to diabetes." (Magliano et al. 2008).

In Mauritius, three cross-sectional surveys on adults aged 25–79 years were conducted in 1987, 1992 and 1998. For those with IFG followed over 11 years, 38% reverted to NGT, 7% remained in the IFG category, 17% progressed to IGT and 38% progressed to diabetes. For those with IGT followed over 11 years, 24% reverted to NGT, 4% reverted to IFG, 26% remained IGT and 46% progressed to diabetes. For further results on diabetes incidence, see Table 1 below.

Table 1. Diabetes incidence per 1000 person years

Category	Cohort	Men	Women
NGT at baseline	1987-1992*	14.6	5.8
	1992-1998**	12.9	10.3
IFG at baseline	1987-1992	54.1	35.1
	1992-1998	60.5	74.7
IGT at baseline	1987-1992	60.7	47.9
	1992-1998	119.6	81.0

Soderberg et al. 2004

* Cohort 1987-1992 (n=3680), five-year standardised diabetes incidence

** Cohort 1992-1998 (n=4178), six-year standardised diabetes incidence

Stratified analysis by ethnic group showed that diabetes incidence increased in Asian Indians (incidence rate ratio (IRR) 1.15 (1.01, 1.32) and Creoles (IRR 1.41 (1.23, 1.62)) between the five- and six-year periods. Compared with Asian Indians, Creole participants had a lower diabetes incidence during the first period, but this difference disappeared in the second period. Chinese participants also had a lower diabetes incidence during the first period, but the sample size was too small for comparison in the second period. For IFG and IGT, Asian Indians developed more IGT, but less IFG, than Creoles during the first period. IFG incidence remained higher in Creoles in the second period (Soderberg et al. 2004).

In the San Antonio Heart Study, 3015 participants aged 25–64 years were followed for a median 7.8 years. The age- and sex-adjusted OR for incident diabetes was 2.33 (1.74, 3.11) for Mexican Americans compared with non-Hispanic whites, which was reduced to 1.53 (1.09, 2.14) after further adjustment. Independent predictors of incident diabetes included IGT (OR 4.69 (3.49, 6.31)), IFG (4.07, 2.98, 5.57)), family history of diabetes (1.73 (1.29, 2.31), and BMI per 1 unit increase (1.10 (1.07, 1.13)). Mexican Americans with normal two-hour glucose at baseline had greater odds of developing IGT or diabetes, and Mexican Americans with IGT at baseline had greater odds of developing diabetes than non-Hispanic whites. The excess risk of incident IGT and diabetes in Mexican Americans was attenuated after further adjustment. Furthermore, compared with non-Hispanic whites, Mexican Americans have a greater age- and sex-adjusted risk of IGT or diabetes among the non-obese (OR 1.73 (1.36, 2.21)) but a comparable risk among the obese (1.08 (0.75, 1.56). For those who had IGT at baseline, age- and sex-adjusted OR for incident diabetes were 2.01 (1.04, 3.87) and 1.07 (0.52, 2.20), respectively, for non-obese and obese Mexican Americans when compared with their non-Hispanic whites counterparts.

The authors concluded that: "Ethnic differences can be detected at both the early and later stages of the diabetes disease process. However, non-Hispanic whites lose much of the ethnic advantage once they have developed obesity." (Lorenzo et al. 2012).

In a review using data from October 1994 onwards of participants with IGT from six prospective studies (n=2389 with IGT, followed for 16,775 person-years), IGT to diabetes progression ranged from 23% to 62%. Fasting and two-hour glucose were associated with diabetes incidence. BMI was associated with diabetes incidence even after adjusting for age, fasting and two-hour glucose (adjusted relative hazard per 4 unit increase in BMI 1.13 (1.08, 1.19)) (Edelstein et al. 1997).

In the KORA S4/F4 cohort study of 887 people aged 55–74 years without diabetes at baseline and followed for seven years, the standardised incidence rate was 15.5 per 1000 person-years. Predictors of diabetes incidence included age per 1 SD increase (adjusted OR 1.6 (1.2, 2.1)), parental diabetes (1.8 (1.0, 3.3)), BMI per 1 SD (1.4 (1.1, 1.9)), fasting glucose per 1 SD (1.6 (1.2, 2.2)), 2 hour glucose per 1 SD (2.5 (1.9, 3.3)), HbA1c per 1 SD (1.6 (1.2, 2.2)), uric acid per 1 SD (1.7 (1.3, 2.3)), and current smoker (3.6 (1.5, 8.9)). The age- and sex-adjusted OR for diabetes incidence was 21.2 (10.4, 43.3) for those with combined IFG and IGT. The adjusted OR for diabetes was not statistically different between those with isolated IGT (9.9 (5.0, 15.6)) and those with isolated IFG (4.7 (2.2, 10.0)) (Rathmann et al. 2009).

The Epidemiological Study on the Insulin Resistance Syndrome (DESIR) study followed 979 participants aged 30–64 years with IFG at baseline for nine years. Adjusted OR for nine-year diabetes incidence was 1.79 (1.45, 2.21) for 1 SD increase in waist circumference and 1.86 (1.51, 2.30) for 1 SD increase in weight. For those with BMI <25 kg/m^2 and those with BMI ≥25 kg/m^2, the adjusted OR for 1 SD increase in waist circumference were 2.40 (1.63, 3.52) and 1.66 (1.28, 2.16), respectively. The adjusted OR for 1 SD change in weight for those with BMI <25 kg/m^2 and BMI ≥25 kg/m^2 were 1.92 (1.32, 2.79) and 1.96 (1.50, 2.55), respectively (Gautier et al. 2010).

The Pathobiology of Prediabetes in a Biracial Cohort (POP-ABC) study included 343 non-diabetic offspring of parents with type 2 diabetes followed for 5.5 years. After a mean follow-up of 2.6 years, 101 participants had a level of IFG and/or IGT defined as prediabetes and 10 developed diabetes.

No significant racial (black/white) difference was observed for cumulative incidence of prediabetes or combined high risk of prediabetes/diabetes. Age- and sex-adjusted HR for having prediabetes and diabetes were 2.9 (1.74, 4.82) for 90th vs 10th percentile trunk fat mass, and 2.54 (1.46, 4.40) for 90th vs 10th percentile 2hPG (Dagogo-Jack et al. 2014).

8. To what extent can progression to diabetes be successfully delayed or permanently reversed with lifestyle modification?

There is substantial evidence that progression to diabetes in people at high risk of type 2 diabetes can be prevented or at least delayed (NHMRC Evidence Level A). There are remaining questions about whether any one intervention approach is more effective than any other, the intensity of treatment required for optimal effect, whether there are individual or group characteristics that identify better responses to specific interventions, and the overall cost-effectiveness.

The Diabetes Prevention Program (DPP) is a key study in the evidence base for prevention of progression. This was a randomised controlled trial of intensive lifestyle intervention, metformin or placebo to prevent progression among people screened as at high risk of diabetes. The Diabetes Prevention Program Outcomes Study (DPPOS) was the long-term follow-up (median of 5.7 years) of 2766 of 3150 original participants from the DPP. When compared with the placebo group, diabetes incidence in the 10 years since randomisation was reduced by 34% in the lifestyle group and 18% in the metformin group. For the intensive lifestyle intervention group, the incidence was 4.8 (4.1, 5.7) cases per 100 person-years during DPP and 5.9 (5.1, 6.8) per 100 person-years during DPPOS (Knowler et al. 2009).

In the DPPOS study, at entry all participants were at high risk of diabetes based on the screening blood test. However, on subsequent testing not all had a persistent abnormal test and were therefore not considered as having responded to the specific intervention. Compared with the placebo group, participants in the intensive lifestyle intervention group who consistently had a high risk of developing type 2 diabetes during DPP had a higher risk of diabetes (HR = 1.31 (1.03, 1.68)) and lower odds of normal glucose regulation (OR = 0.59 (0.42, 0.82)) during DPPOS. No such association was observed for the metformin group (Perreault et al. 2012).

More recently, 15-year follow-up diabetes incidence has been reported. It showed that incidence reduced by 27% in the lifestyle group and 17% in the metformin group compared with the placebo group. The press release stated that: "After the initial randomised treatment phase in DPP, all participants were offered lifestyle intervention and the rates of diabetes development fell in the metformin and former placebo groups, leading to a reduction in the treatment group differences over time. However, the lifestyle intervention and metformin are still quite effective at delaying, if not preventing, type 2 diabetes." (American Diabetes Association 2014).

The Finnish Diabetes Prevention Study (DPS) randomised 522 participants aged 40–64 years to either intensive lifestyle intervention or control. Participants (n=366) still free of diabetes after a median of four years intervention (200 in intervention, 166 in control) were followed up for a median of nine years (total median 13 years from baseline). The incidence of diabetes from randomisation was 4.5 per 100 person-years for the intervention group and 7.2 per 100 person-years for the control group. The adjusted HR was 0.61 (0.48, 0.79) and the absolute risk reduction was 19%. The incidence of diabetes during the post-intervention follow-up was 4.9 per 100 person-years for the intervention group and 7.0 per 100 person-years for the control group. The adjusted HR was 0.67 (0.48, 0.95) and the absolute risk reduction was 15% during the post-intervention follow-up period. For participants who developed diabetes, the median time to onset of diabetes was 15 years and 10 years, respectively, in the intervention and control groups; i.e. a median delay of five years with intensive lifestyle intervention. The magnitude of the risk reduction was not influenced by the duration of the invention (Lindstrom et al. 2013).

Another Finnish diabetes prevention program, the FIN-D2D, aimed to prevent diabetes in 2798 high-risk adults using lifestyle interventions delivered in primary health care settings. Compared with the group who maintained their weight, the RR of developing diabetes was 0.31 (95%CI 0.16, 0.59) in the group that lost more than 5% of their weight, 0.72 (95%CI 0.46, 1.13) in the group who lost 2.5–4.9% of their weight, and 1.10 (95%CI 0.77, 1.58) in the group who gained more than 2.5% of their weight. In total, 17.5% of participants lost more than 5% of their baseline weight, which translated to a reduction of 8.5kg in weight and 6.6cm in waist circumference. Of the remaining participants, 16.8% of participants lost 2.5–4.9% of their weight, 46.1% maintained weight, and 19.6% gained more than 2.5% of their weight (Saaristo et al. 2010).

A meta-analysis of intervention strategies to prevent type 2 diabetes reported the order of effectiveness in prevention of new cases of diabetes was bariatric surgery (OR 0.16), followed by glitazones (0.37), diet and physical activity (OR 0.43 (0.35, 0.52); I2=0%), diet (0.51 (0.39, 0.68); I2=29%), physical activity or education (0.53 (0.40, 0.70); I2=93%), alfa-glucosidase inhibitors (0.54), metformin (0.65), lipid-affecting drugs (0.66) and cardiovascular drugs (0.74–0.76). Since the intervention strategies were not always directly compared, the effectiveness order is not fully reliable (Merlotti et al. 2014a).

A further meta-analysis from the same authors of intervention strategies to prevent diabetes specifically in obese people reported the relative effectiveness in prevention of new cases of diabetes was bariatric surgery (OR 0.10), physical activity and diet (OR 0.44 (0.36, 0.52)), weight loss-promoting drugs and lipid-lowering drugs (0.52), anti-diabetic drugs (0.53), and antihypertensive drugs (0.86) (Merlotti et al. 2014b).

A systematic review on lifestyle intervention for people at risk of type 2 diabetes reported pooled relative risk (RR) for four studies at the end of intervention (0.35 (0.14, 0.85); I2=68%), two studies at four-year follow-up (0.56 (0.48, 0.64); I2=0%), and three studies at six-year follow-up (0.47 (0.34, 0.65); I2=0%). It also reported RR from DPP for 10-year follow-up (0.80 (0.74, 0.88)) (Schellenberg et al. 2013).

Another systematic review on lifestyle intervention for people with IGT reported that of the seven randomised clinical trials identified, five showed significant reduction in diabetes incidence by lifestyle intervention, one showed significant reduction only in the first year of follow-up, and one showed significant reduction only in the per-protocol analysis (Yoon et al. 2013).

A number of lifestyle intervention programs designed to prevent diabetes reported weight change or other clinical measurements as primary outcomes rather than diabetes incidence:

• A recent systematic review and meta-analysis examined the efficacy of lifestyle interventions that included diet, aerobic and resistance training components for the prevention of type 2 diabetes in at-risk adults. Of the eight included studies, only two studies reported diabetes incidence and found diabetes incidence had reduced by 58% and 56%. The meta-analysis reported that average weight was reduced by 3.79kg (95%CI -6.13, -1.46) and fasting plasma glucose was reduced by 0.13 mmol.L-1 (95%CI -0.24, -0.02) (Aguiar et al. 2014).

• Another recent systematic review and meta-analysis of the effectiveness of lifestyle interventions for the prevention of type 2 diabetes reviewed 25 studies and reported a mean weight loss of 2.32 kg (95%CI -2.92, -1.72). A pooled diabetes incidence rate of 34 cases per 1000 person-years was reported for the eight included studies that measured diabetes incidence (Dunkley et al. 2014).

• A systematic review and meta-analysis of 2916 participants from 28 studies that conducted lifestyle interventions based on the DPP in the United States found that the average weight change at one year was -4% (95%CI -5.16, -2.83) of participants' baseline weight (Ali et al. 2012).

• A systematic review of multi-factorial lifestyle interventions found mean differences (MD) for BMI between intervention groups and controlled groups of -.10 and -1.01 for interventions that went for six months, and MD -0.70 for an intervention that went for 8–12 months (Angermayr et al. 2010).

9. Is the effect of lifestyle modification on diabetes risk equal across different groups? Are there some people who benefit more or others who do not benefit even with good adherence to a diet and exercise regime?

There is little evidence that the impact of lifestyle changes on the risk of diabetes varies systematically by any attributes other than the level of risk. The DPP randomly assigned 3234 participants to intensive lifestyle intervention, metformin and placebo. Participants were recruited between 1996 and 1999, and followed for an average of 2.8 years (range, 1.8 to 4.6). Diabetes incidence was 58% lower in the lifestyle intervention group and 31% lower in the metformin group compared with the placebo group. There was no significant difference in treatment effects according to sex or to race and ethnicity.

When compared with placebo, the effect of lifestyle intervention was significantly greater among participants with lower baseline two-hour glucose than those with higher baseline levels (reduction in incidence were 76% for 2hPG 140–153 mg/dL, 60% for 2hPG 154–172 mg/dL and 50% for 2hPG 173–199 mg/dL).

When compared with metformin, the advantage of lifestyle intervention was greater in older persons (reduction in incidence were 8% for aged 25–44 years, 41% for aged 45–59 years and 69% for aged ≥60 years) and those with a lower BMI (reduction in incidence was 63% for BMI 22-<30, 53% for BMI 30– <35, and -4% for BMI ≥35) (Knowler et al. 2002).

An add-on study of the DPP sample examined the effect of Q risk allele at ENPP1 K121Q loci. The authors reported: "This study demonstrates that carriers of the Q risk allele at ENPP1 K121Q have an increased incidence of diabetes and that the lifestyle or metformin intervention arms of the DPP abolished this effect … Lifestyle modification eliminated and metformin reduced the increased risk imparted by ENPP1 K121Q in the placebo arm through a mechanism at least partially mediated by a reduction in BMI. These results suggest that carriers of the ENPP1 Q risk allele may benefit disproportionately from lifestyle modification or metformin therapy compared with K allele carriers."

However, this claim is questionable as on examination of the results, the hazard ratios (HR) for lifestyle and metformin were not significantly different. The multivariate adjusted (age, sex, ethnicity, BMI) HR between Q allele carriers and diabetes incidence were 1.36 (1.02, 1.80) for placebo, 1.09 (0.78, 1.53) for metformin and 0.83 (0.56, 1.24) for lifestyle (Moore et al. 2009).

The Zensharen Study for Prevention of Lifestyle Diseases randomised 641 overweight participants (311 in frequent intervention and 330 in control) aged 30–60 years with IFG. With mean follow-up of 32 months, adjusted HR for diabetes incidence was 0.56 (0.36, 0.87) for the frequent intervention group compared with the control group. In subgroup analysis by baseline glycaemic status, the adjusted HR was 0.41 (0.24, 0.69) for those with IFG and IGT and 0.24 (0.12, 0.48) for those with HbA1c ≥5.6%. The authors concluded that: "Identifying individuals with more deteriorated glycaemic status by using 75-g OGTT findings or, especially, measurement of HbA1c levels, could enhance the efficacy of lifestyle modification" (Saito et al. 2011).

What do we know about participants who have particularly high or low levels of participation during large-scale diabetes prevention programs?

The characteristics of high- and low-level participants in diabetes prevention trials are not well documented. During the DE-PLAN diabetes prevention program in Greece, participants who had the highest participation rates were significantly more likely to be male and to have been recruited from an occupational rather than primary health care setting. Attendance at the one-year follow-up oral glucose tolerance test was higher at occupational centres than primary health care settings (Schwarz and Reddy 2013).

During the GOAL-LIT program in Finland, participants who had high attendance rates were more likely to be married or live with a partner than those who dropped out. Attendance rates did not differ significantly according to socioeconomic status (Absetz et al. 2007).

Results from the large-scale diabetes prevention programs in Australia indicate that older women are likely to have high participation rates, and smokers are more likely to drop out (Schwarz and Reddy 2013). Frequent contact with the program providers may make participants less likely to drop out of interventions. The Life! Course Victorian prevention program had the highest attrition rate between visits 5 and 6, where there was a six-month gap. The use of health workers from within the study community, and financial incentives, are also factors that may help to keep participants in programs (Schwarz and Reddy 2013).

Are there predictors of success for lifestyle interventions?

A study by Rautio et al. (2013) evaluated individuals at high risk of type 2 diabetes (n=3880) in the one-year follow-up of the Finnish National Diabetes Prevention Program to assess predictors of ≥5% weight loss and improved glucose tolerance. Abnormal glucose tolerance was the strongest predictor of both ≥5% weight loss and improvement in glucose tolerance. A high attendance at lifestyle intervention visits, unemployment, screen-detected T2D, and high BMI were factors that made participants more likely to achieve ≥5% weight loss. Having a high level of education was related to improved glucose tolerance.

10. What is the impact of the intensity of an intervention on reduction in risk of progression to diabetes?

Are there any studies that directly evaluate the impact of the intensity of an intervention on type 2 diabetes prevention?

The direct evidence for the impact of the intensity of diabetes prevention programs is very limited, with small cohort studies often including an extremely low number of subjects (Hansen et al. 2010). While many prevention programs provide details of the intensity of their interventions, they have not been directly compared with lower or higher 'doses' of interventions to examine effectiveness.

A systematic review evaluating multifactorial lifestyle interventions in both primary and secondary prevention of CVD and type 2 diabetes identified 15 randomised controlled trials (Angermayr et al. 2010). The authors found that interventions varied greatly in their intensity, for example contact hours ranged from one to 596 hours. Some interventions were delivered intensively between 2.5 and 45 days while participants stayed at a retreat, while other interventions were provided over months. The intensity of an intervention was not found to significantly impact on outcome.

What can we learn from the differing intensities of large-scale diabetes prevention programs from around the world?

Population-level lifestyle interventions from around the world have delivered type 2 diabetes prevention programs successfully using different levels of intensity. See Table 2 on the next page.

Table 2. Large-scale[2] type 2 diabetes prevention programs using lifestyle interventions

Program (reference)	Intervention	Intervention administrator	Dose	Target population	Main outcomes	Evidence of effectiveness?	Results
FIN-D2D, Finland (Saaristo et al. 2007; Saaristo et al. 2010; Salopuro et al. 2011; Schwarz 2011; Rautio et al. 2013)	Either group meetings (content included lectures, weight maintenance, exercise sessions) or individual counselling	Primary and occupational health care providers	Frequency varied according to local circumstances. Both 1–3 and 4–8 sessions have been reported	10,149 patients aged 18–87 years from 5 hospital districts, deemed high risk (Finish Diabetes Risk Score ≥15; or history of elevated blood glucose, coronary heart disease or gestational diabetes)	• Diabetes • Weight • Waist circumference • Blood pressure • Lipids	Yes	Mean body weight and obesity decreased in both the intervention and control areas. Mean waist circumference and morbid obesity was unchanged in intervention area, but increased in control area
GOAL-LIT, Finland (Absetz et al. 2007)	Group-based, task-oriented counselling lifestyle change program	Occupational nurses	5 sessions of 2–hour duration, and 1 booster session	352 middle-aged people at risk (Finish Diabetes Risk Score >12)	• Diabetes • Weight • Waist circumference • Pre-defined healthy behaviour goals • BMI • Lipids • Blood pressure • Plasma glucose	Yes	20% of participants at 12 months achieved ≥4 of the following: (1) <30% of total energy intake from fat (2) <10% of total energy intake from saturated fat (3) ≥15 g of fibre/1,000 kcal (4) ≥4 h/week moderate level physical activity (5) >5% weight reduction. Physical activity and weight loss goals occurred significantly less frequently
DPP Lifestyle Balance, United States (Diabetes Prevention Program Research Group 2002; Knowler et al. 2002)	Individual consultations and physical activity sessions; follow-up used group or individual sessions, telephone contact	Trained lifestyle coaches of which majority were dietitians	16 sessions, 24 weeks, 30–60 mins, 2 voluntary physical activity sessions per week. Follow-up over 2+ years: session every 2 months, 15–45 mins, telephone contact	1075 participants over 25 years of age, BMI ≥24 (22 in Asians), elevated plasma glucose	• Diabetes • Fasting plasma glucose • Glycosylated haemoglobin • Physical activity levels • Diet	Yes	Diabetes incidence was 58% lower in the lifestyle intervention group than the control group

[2] Programs have more than 350 participants

Table 2. Large-scale[2] type 2 diabetes prevention programs using lifestyle interventions

Program (reference)	Intervention	Intervention administrator	Dose	Target population	Outcome evaluation	Evidence of effectiveness?	Results
Life! Taking Action on Diabetes, Australia (Schwarz 2011; Schwarz and Reddy 2013; Dunbar et al. 2014)	Group-based intervention	Health professional	6 sessions, 1.5–2 hour duration, fortnightly but final session conducted 6 months after previous	8412 people deemed at risk (AUSDRISK >12)	• Weight • Waist circumference • Pre-defined healthy behaviour goals	Yes	Weight loss: those who completed sessions 1–5 lost an average 1.4 kg, those who completed six sessions lost 2.4 kg (2.7% body weight). Waist circumference: those who completed sessions 1–5 lost an average of 2.5 cm, those who completed six sessions lost 3.8 cm
Zensharen, Japan (Saito et al. 2011)	Individual lifestyle modification instructions, follow-up support	Trained medical staff	At least 9 individual sessions at baseline and 1, 3, 6, 12, 18, 24, 30 and 36 month marks, with two extra sessions available	641 adults aged 30–60 years, fasting plasma glucose level 100–125 mg/dL, BMI at least 24	• Diabetes	Yes	The cumulative incidence of diabetes was estimated to be 12.2% in the intervention group and 16.6% in the control group.
IDPP-I, India (Ramachandran et al. 2006)	Lifestyle modification advice, or metformin, or lifestyle modification advice and metformin	Unknown	Initial interview, monthly telephone contact, 6-month face-to-face visit, progress reviewed every 6 months	531 adults with impaired glucose tolerance, working in service organisations, deemed to be middle-class	• Diabetes	Yes	The 3-year cumulative incidence of diabetes was 39.3% in the lifestyle modification group, 40.5% in the metformin group, 39.5% in the lifestyle plus metformin group, compared with 55.0% in the control group.
Da Qing, China (Pan et al. 1997; Li et al. 2008)	A prescribed diet, or exercise targets, or prescribed diet + exercise targets	Local physicians, nurses and technicians	Individual counselling sessions weekly for 1 month, monthly for next 3 months, then once every 3 months.	577 adults with impaired glucose tolerance, of which 568 participated in the 20 year follow-up	• Diabetes • Cardiovascular disease • Mortality • Diabetes complications	Yes	Participants in combined lifestyle interventions had 51% lower incidence of diabetes compared with the control group during the active intervention, and 43% lower for the 20-year follow-up period. Intervention participants had 3.6 fewer years with diabetes than control groups.

Table 2. Large-scale[2] type 2 diabetes prevention programs using lifestyle interventions

Program (reference)	Intervention	Intervention administrator	Dose	Target population	Outcome evaluation	Evidence of effectiveness?	Results
Occupational setting study, Finland (Viitasalo et al. 2015)	Face-to-face lifestyle counselling sessions, access to a diabetes prevention information website	Dietitian and/or diabetes nurse	1–3 sessions, 1 hour duration, 6 months interval between session 1 and 2	1347 employees of an airline company, at risk	• Weight • Waist circumference • BMI • FINDRISC score • Blood glucose • Cholesterol • Triglycerides	Yes	Modest health benefits were found for men at risk, but not for all women or men with low baseline risk. The authors concluded the study needed to be more intensive to improve health outcomes.
SDPP, Australia (Colagiuri et al. 2010; Schwarz 2011; Schwarz and Reddy 2013)	Face-to-face group or individual consultations, telephone health coaching, GP consultations	Health professionals (psychologists, nurses, dietitian, diabetes educators, exercise physiologists)	1 initial individual session, 3 group or individual telephone sessions, 3 telephone follow up calls, 2 GP consultations, over 12 months, sessions ranged from 20–120 mins	1238 English-speaking persons aged 50–65 years deemed at risk (AUSDRISK >15)	• Weight • Physical activity levels • Diet	Pending published results	Pending published results
SDPP, Germany (Schwarz 2011)	Group consultations	Health professional	8 sessions, 8 hours total, telephone and email support and annual follow-up	Persons at risk (FINDRISK >11)	• Weight • Waist circumference • Blood pressure • Other unspecified clinical measurements	Pending published results	Pending published results
Let's Prevent Diabetes, United Kingdom (Schwarz 2011; Gray et al. 2012)	Structured education program and telephone support	Registered health care professional	6-hour program followed by annual maintenance session Telephone contact every 3 months	Persons at risk (Leicester Risk Score and confirmed with OGTT)	• Diabetes • Physical activity levels • Diet • Lipids	Pending published results	Pending published results

[2] Programs have more than 350 participants

11. Is there any evidence that the effects of large-scale diabetes prevention programs continue after the active period of intervention?

Li et al. (2008) conducted a 20-year follow-up study of the Da Qing Prevention Study in China. The study found that, over the 20-year period, those in combined lifestyle intervention groups had 43% lower incidence of diabetes compared with the control groups. During the active period of intervention, the intervention groups had 51% lower incidence of diabetes compared with control groups. The authors concluded that group-based lifestyle interventions over a six-year period can prevent or delay diabetes for up to 14 years after the active intervention. A recent systematic review and meta-analysis found that more research is needed to understand the longer-term effects (i.e. beyond 12 months) of weight loss and diabetes prevention interventions (Dunkley et al. 2014).

12. What strategies are required to support scaled implementation of programs to promote individual lifestyle change for diabetes prevention in Australia?

To achieve population-wide benefits of potentially effective diabetes interventions in Australia, three key questions require consideration:

• Are the interventions scalable?

• What existing models for diabetes prevention in Australia and internationally are available to inform program planning for diabetes prevention?

• What system-level factors need to be considered to create an enabling environment to support the delivery of programs for diabetes prevention?

Scalability of interventions

"Scalability refers to the ability of a health intervention shown to be efficacious on a small scale and/or under controlled conditions to be expanded under real world conditions to reach a greater proportion of the eligible population while retaining effectiveness" (Milat et al. 2013). The scalability of an intervention is determined by its likely reach and adoption, the costs of operating at scale, and its suitability for adaption to improve local acceptability and fit while retaining core fidelity.

Program planners and implementers often lack the skills and knowledge of scaling-up methods (World Health Organization 2010), and there is a paucity of published literature to provide guidance (Kohl and Cooley 2003; World Health Organization 2010; Yamey 2011), contributing to under-utilisation of available evidence-based public health interventions (Milat et al. 2014b).

Based on a synthesis of this existing literature and recent research (Milat et al. 2013; Milat et al. 2014b), the NSW Ministry of Health developed a guide to assessing the scalability of an intervention and steps to developing a scaling-up strategy for public health interventions (Milat et al. 2014b). This document has recently been released and can guide policy makers, program planners and implementers in Australia.

The key considerations for assessing the scalability of interventions (Milat et al. 2014b) are:

Effectiveness

Potential reach and adoption

a) Determining the likely reach of the intervention per eligible population when scaled up

b) Determining the likely adoption rate by local setting and organisational level

c) Considering potential likelihood of differential rates of reach and adoption among different risk groups.

Alignment with the strategic content

a) Consider consistency of the intervention with national, state and regional policy directions

b) Consider compatibility of intervention with roles, skills, practices and existing interventions in the contexts where the intervention is to be scaled.

Acceptability and feasibility

a) Consider the capability of the current system to accommodate the requirements of delivering the intervention at scale

b) Determine whether the intervention is likely to be acceptable to target groups and stakeholders when scaled up

c) The potential costs of going to scale should fit within the budget available.

13. What existing models for diabetes prevention in Australia and internationally are available to inform program planning for diabetes prevention?

Models can be an important tool for planning population-level prevention programs. A model can provide clarification of the linkages between activities and expected outcomes, as well as identify stakeholders and priority areas (Greenfield et al. 2006; United States Department of Health and Human Services 2006). Models can also be used to guide the evaluation of the impact and effectiveness of an intervention, and guide quality improvement, since expected outcomes and achievements can be compared with empirical observations (Greenfield et al. 2006).

We conducted a search for the application of models for planning and evaluating large-scale type 2 diabetes programs in both the grey and peer-reviewed literature. The search yielded seven models for type 2 diabetes prevention programs: one from Australia; four from the United States; and two from Canada. It is likely that there are additional models to guide large-scale type 2 diabetes programs but have not been made publicly available. It is therefore probable that our search did not capture all relevant existing models.

The model used to plan the Life! Taking action on diabetes program in Australia did not identify inputs and had significantly less detail. While each model from the United States varied, they all identified similar relationships between inputs and outcomes, and had a similar level of detail. Only one model, also from the United States, was designed specifically for community engagement. The two models from Canada were not of sufficient picture resolution to be compared with the other models. Our search also identified two models for state-wide health promotion programs that did not exclusively focus on diabetes prevention, but included the prevention and reduction of diabetes as an impact (State Government of Victoria; Rosenberger and Lawrence 2000). This included a model for the evaluation of the Healthy Together Victoria intervention in Australia (State Government of Victoria). Available logic models:

Australia

1. Life! Taking action on diabetes, evaluation framework (Holt 2009)

United States

1. Logic model for diabetes prevention and control program grantees (Centers for Disease Control and Prevention 2013, p. 114)
2. National Diabetes Education Program conceptual framework (Gallivan et al. 2008, Figure 1, p. 4)
3. Draft logic model for community engaged type 2 diabetes intervention (West 2014, Figure 1)
4. Nebraska Diabetes Prevention and Control Strategic Plan logic model (Nebraska Department of Health and Human Services Diabetes Prevention and Control Program 2010, p. 9)

Canada

1. ISHLCD community-based programming and community capacity building functional component logic model (Public Health Agency of Canada 2009, Figure 1)

2. Renewed Canadian diabetes strategy logic model (Public Health Agency of Canada 2007, Annex B.)

14. What system-level factors need to be considered to create an enabling environment to support the delivery of diabetes prevention programs?

Ensuring the effective implementation of a program is often the responsibility of program implementers, with emphasis placed on intervention-level factors such as the intervention's adaption and fidelity, dose (delivered and received), quality and reach. However, focusing on intervention-level factors alone neglects barriers that arise from the broader system within which the program is operating. These broader system-level factors are often beyond the ability of program implementers to influence, but it is crucial to recognise their influence on program implementation.

A systems-orientated framework for the implementation of large-scale programs can help to remove system-level barriers to large-scale implementation and help prevent adverse unintended consequences. Two such frameworks are structurally similar but highlight different elements as the key to effective implementation (Durlak and DuPre 2008; Wandersman et al. 2008). These frameworks conceptualise three interacting systems that support effective implementation of public health initiatives. These are:

- The Prevention Synthesis and Translation System – the key function is to synthesise diverse evidence sources regarding the effectiveness of interventions or innovations and prepare them for implementation
- The Prevention Support System – provides intervention-specific capacity building and general organisational-level capacity building to support the work of those that will deliver the interventions
- The Prevention Delivery System – supports the delivery of programs with a focus on the following factors that influence implementation success:
    - Individual-level practitioner characteristics – including education, experience with a similar intervention, and motivation to use it
    - Organisational-level factors – including leadership, alignment of program with organisational goals, commitment, skills for planning, implementation and evaluation, organisational structure and culture
    - Community factors – including community readiness, health literacy, empowerment and collective efficacy.

While these frameworks conceptualise a preventive health system and the multi-level factors that might influence its performance, they don't make explicit the system-level factors that need to be considered when designing an implementation strategy for a national public health program. Table 3 outlines system-level considerations that may help to lay the necessary foundations for optimal impact of diabetes prevention programs in Australia. These considerations have been drawn primarily from 'lessons learned' from past Australian and international diabetes prevention programs as well as from reviews of research and evaluation of large-scale programs to promote lifestyle change (with a focus on diet and physical activity).

In addition, a key report discusses the role of Australian primary health care in the prevention of chronic disease (Harris and Lloyd 2012). This report provides some recommendations specific to the Australian context regarding:

- The organisation of preventive care at the practice level
- The potential and actual roles of primary care organisations
- Improving integration between clinical and community-based programs
- Improving equity of access to preventive care
- A number of system-level strategies (Harris and Lloyd 2012).

Table 3. System-level factors and considerations to support implementation of diabetes prevention programs in Australia

Service delivery	Considerations
	• Consider staged roll-out to ensure supply of facilitators does not outstrip demand for lifestyle modification programs (Dunbar et al. 2010).
	• Consider delivery mechanisms – programs in clinics were better able to refer, track and retain participants (Centers for Disease Control and Prevention 2013).
	• Consider participatory approaches to engage target populations in the planning, delivery or evaluation of programs to effectively reach communities of interest and delivering appropriate messages and activities (Durlak and DuPre 2008; Public Health Agency of Canada 2010).
	• Early use of multi-media approaches and specific targeted social marketing techniques to raise awareness of seriousness of diabetes, risk assessment and prevention. Use local marketing for program promotion – the most successful strategies for recruiting participants in prevention programs have been generated by local programs because these activities are better tailored to the community context (Dunbar et al. 2010; Schwarz et al. 2012; Centers for Disease Control and Prevention 2013).
	• Design strategies for achieving lifestyle change program credibility among primary care physicians as well as the public to improve acceptability and engagement (US Department of Health and Human Services 2006; Reddy et al. 2011).
	• Establish strategies to optimise recruitment (may include organisational support, remuneration, workforce development, incentives for GPs etc.) (Dunbar et al. 2010).
Workforce	**Considerations**
	• Use respected health care professionals including registered nurses, registered dietitians, exercise specialists and other health care professionals with expertise and experience in nutrition and physical activity as well as counselling and motivational interviewing techniques (US Department of Health and Human Services 2006; Centers for Disease Control and Prevention 2013).
	• Ensure adequate engagement of service providers, particularly GPs, and their professional organisations (Durlak and DuPre 2008; Dunbar et al. 2010).
	• Assist primary care practitioners to be systematically and proactively orientated towards prevention programs (Dunbar et al. 2010).
	• Consider establishment of an accreditation system to deliver program components; with annual review workshop to maintain fidelity of intervention consistency in program delivery; and provide avenue for peer learning and support (Dunbar et al. 2010; Centers for Disease Control and Prevention 2013).
	• Consider workforce challenges in rural and remote areas. A lack of appropriate and adequately trained health professionals to manage, implement and sustain interventions is likely to be an obstacle in rural and remote areas. A separate model for workforce, training, program delivery, governance and financing mechanisms may be required (Reddy et al. 2011).
	• To better understand the nature of the existing preventive health system, workforce and labour market context in which the program will be operating, a framework and methodology for data collection has recently been developed (Harris and Lloyd 2012).

Training and education	**Considerations** • Investment in high-quality training is considered essential for the implementation of a successful diabetes prevention program (Schwarz et al. 2012). Consider training approaches that ensure provider proficiencies in the skills required, and enhance providers' self-efficacy (Durlak and DuPre 2008). • Establish a robust induction process, a system for ongoing training and technical support, and a structure for interaction between primary health care professionals and facilitators of lifestyle change programs (US Department of Health and Human Services 2006; Goodman et al. 2008). • Establish networks of practice as a forum for exchanging knowledge and locally adapted intervention materials, educational standards and recommendations for best prevention practice (Schwarz et al. 2012). • Develop adequate resources for professionals including guidelines for diabetes prevention practice. The availability of structured material is considered vital to successful large-scale implementation (Department of Health 2010; Schwarz 2011; Schwarz et al. 2012). • The barriers to healthy behaviours in at-risk populations often go beyond the health sector (i.e. settlement, housing and employment issues). Provide information in the resource manuals for frontline staff to help address these barriers (Schwarz et al. 2012; Centers for Disease Control and Prevention 2013).
Governance and leadership	**Considerations** • Program governance – consider inclusive involvement of senior policy makers, health professionals and academics on the advisory board (Dunbar et al. 2010). • Establish a leadership structure appropriate for the intended scale of the initiative including overarching and local management structures (Goodman et al. 2008). • Establish a legitimation strategy – to achieve agreement among relevant decision makers, funders and opinion leaders that the new solutions are necessary and desirable, and that they are cost-effective and feasible (Kohl and Cooley 2003). • Constituency building – develop and mobilise constituencies for change among key decision makers and stakeholders within the implementing organisations and among the broader public (Kohl and Cooley 2003). • Build a national coalition of partners, including primary care, commercial sector, non-governmental organisations, community groups, media, workplace health agencies and other government departments (Durlak and DuPre 2008; Goodman et al. 2008; Department of Health 2010; Centers for Disease Control and Prevention 2013). Utilise existing partnerships where possible (Porterfield et al. 2010). Intervention champions, particularly physicians, have been found to be particularly valuable partners (US Department of Health and Human Services 2006; Durlak and DuPre 2008; Centers for Disease Control and Prevention 2013). • Develop an engagement strategy targeted to relevant stakeholders (political, private sector, non-government organisations, and target population) (Schwarz 2011; Schwarz et al. 2012). Ensure engagement is an ongoing process rather than a one-off exercise (Goodman et al. 2008). • Foster a culture of accountability at the local level through strategies including the promotion of local ownership of the program (Goodman et al. 2008). • Establish service level agreements with service providers to ensure standards and accountability (Dunbar et al. 2010).

Financing	Considerations
	• Develop an acceptable reimbursement model (including Medicare and private health insurance) to adequately fund diabetes prevention programs delivered through primary care (Katula et al. 2011). Ensure reimbursement model adequately provides for patient screening, recruitment, delivery of lifestyle modification programs, as well as multiple follow-up visits and feedback of results (Dunbar et al. 2010). Consider quality-dependent reimbursement and payment structures (Schwarz et al. 2012). Consider a tax incentive for private sector screening (Schwarz 2011).
	• Consider an incentive model for consumers – e.g. partner with state and local governments and the private sector to recommend that recognised/accredited lifestyle modification programs be offered as a covered benefit for employees or develop worksite policies that promote increased physical activity (Centers for Disease Control and Prevention 2013).
Infrastructure (including tools/products/ technology)	Considerations
	• Leverage existing infrastructure and networks by integrating the national diabetes strategy with other relevant national actions to prevent related chronic disease (Durlak and DuPre 2008; Department of Health 2010; Schwarz et al. 2012).
	• Consider separately the infrastructure, resources, partnerships, and mechanisms needed to support the process of 'going to scale' and those that will be needed for 'operating at scale' (Kohl and Cooley 2003).
	• Ensure sufficient organisational capacity for local level implementation and provide adequate managerial/supervisory/administrative support (Durlak and DuPre 2008).
	• Monitor and ensure that there is adequate access, availability and affordability of referral sources/services (Centers for Disease Control and Prevention 2013).
	• Ensure availability and acceptability of tools for screening for a high risk of developing diabetes or diabetes risk assessment (Centers for Disease Control and Prevention 2013).

Information systems/ monitoring and evaluation	Considerations
	• Establish an evaluation framework at the outset and strengthen capacity and resources for evaluation (Goodman et al. 2008; Porterfield et al. 2010; Public Health Agency of Canada 2010).
	• Ensure any newly established data collection systems are carefully planned and easy to use, and are piloted, refined and put in place before roll-out of the initiative (Goodman et al. 2008).
	• Strengthen local, regional and national monitoring systems for the risk factors of diabetes and other chronic disease (National Institute for Health and Care Excellence 2011).
	• Establish high-quality management systems and allocate sufficient staff and resources to the operation of data collection systems to track outcomes, monitor fidelity of the program, and provide continuous bench marking and monitoring of progress. Ensure information is fed back to the local level and is used to encourage participation and make improvements to program delivery (Kohl and Cooley 2003; Dunbar et al. 2010; Porterfield et al. 2010; Schwarz et al. 2012).
	• Poor data collection processes and difficulties in obtaining complete and timely data from participants and providers are obstacles to program monitoring and evaluation (Reddy et al. 2011). Consider linking payments/incentives to provision of data.
	• Ensure adequate performance indicators are established at the macro, meso and micro levels: - Macro-level indicators (national level) might include the prevalence of diabetes in the population and percentage of the population that are physically inactive – Meso-level indicators (health districts) might include proportion of the population screened by health care providers per year and the percentage of identified high-risk individuals directed to diagnostic procedures – Micro-level indicators (healthcare personnel) might include changes in waist circumference and quality of nutrition over one year (Schwarz 2011).
	• Publish to describe and evaluate the program, disseminate evidence of its success, and share lessons learned (Centers for Disease Control and Prevention 2013).

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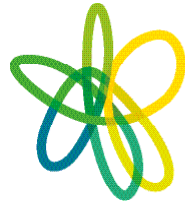
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