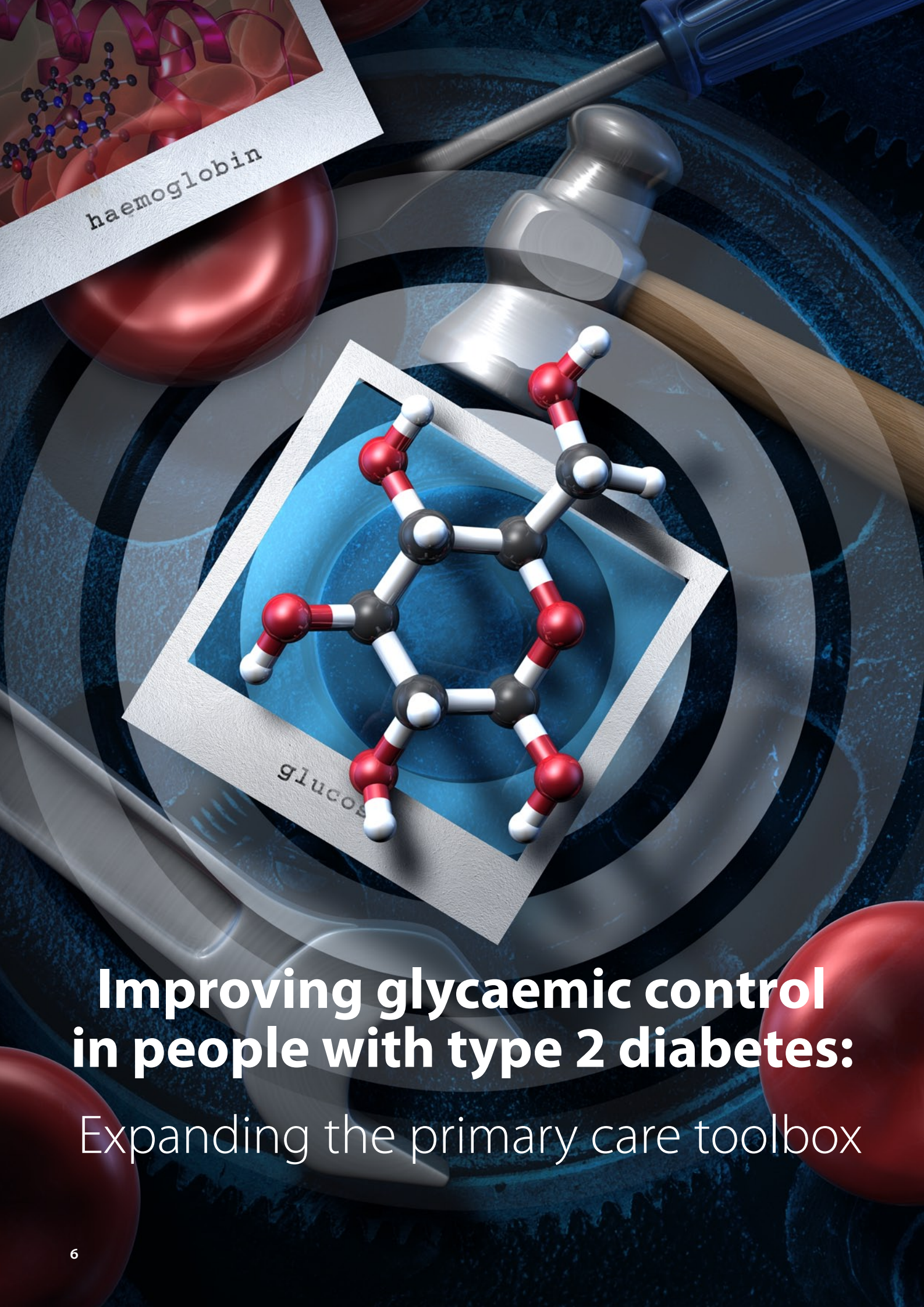


Improving glycaemic control in people with type 2 diabetes:

Expanding the primary care toolbox

Most people with type 2 diabetes require regular and intensive management to achieve individualised HbA_{1c} targets. Patients should be urged to pursue lifestyle interventions, to the best of their ability, to reduce their risk of diabetes-related complications. Type 2 diabetes is a condition of increased insulin resistance and progressive failure of pancreatic beta-cell function. Inevitable escalation of treatment and the role of insulin should be explained early as this helps patients adjust to the introduction of new medicines and reduces the perception of failure. When considering intensifying treatment, the benefits of improved glycaemic control need to be balanced against the specific risk profile of the medicines selected. Where more established treatments are not tolerated, are inappropriate or ineffective in achieving agreed HbA_{1c} targets, other medicines, e.g. acarbose and pioglitazone, may be considered. Some of these medicines, such as pioglitazone, have recently been associated with safety concerns.

Glycaemic control in type 2 diabetes

Widespread obesity, a sedentary lifestyle and an ageing population has resulted in type 2 diabetes being labelled as a global pandemic.[1] Every day in New Zealand 50 people are told by their doctor that they have diabetes.[2] Type 2 diabetes is most prevalent in Pacific males (10.6%) and females (9.9%), Asian males (8.4%), and Māori males (7.9%) and females (6.8%).[3] It has a prevalence of approximately 4 – 5% among European males and females, and Asian females.[3] This "pandemic" is being driven by the high prevalence of intermediate hyperglycaemia, which is estimated to currently affect one-third of Māori and Pacific peoples and one-quarter of New Zealand Europeans aged 45 – 64 years.[4]

Many people with type 2 diabetes will benefit from improved glycaemic control

It is widely accepted that, despite receiving treatment, many people with type 2 diabetes are spending the majority of their life after diagnosis with inadequately controlled blood glucose levels.[5, 6] A recent primary care study of over 26 000 patients diagnosed with type 2 diabetes in the Hamilton region found that approximately 40% of Māori, 30% of people of Asian descent and 20% of New Zealand Europeans had HbA_{1c} levels greater than 64 mmol/mol.[7]

Good glycaemic control in people with type 2 diabetes is known to delay the onset of microvascular complications including renal failure, retinopathy and neuropathy. Good glycaemic control will also have a beneficial effect on macrovascular complications, e.g. coronary artery disease, stroke and peripheral vascular disease, if it is achieved early and maintained.[8]

Type 2 diabetes is a progressive disease which requires lifestyle measures, monitoring and medicines to increase in intensity as pancreatic beta-cell failure progresses. This should be discussed with the patient at an early stage, so that the initiation of additional treatment, including insulin, is not viewed by the patient as being a personal failure. Young people with type 2 diabetes have the most to benefit from intensive management of glycaemic control as they are likely to be exposed to hyperglycaemia for longer due to an increased life expectancy.[9] However, the benefits of the reduced risk of complications need to be balanced against the harms of hypoglycaemia and weight gain associated with more intensive treatment for all patients.

Improved glycaemic control should always be underpinned by lifestyle measures and every person with type 2 diabetes should have an individualised care plan for lifestyle intervention.[9] Dietary assessment should be undertaken for people with type 2 diabetes. Care plans should be reviewed and when agreed goals are not achieved, discussions should be initiated to overcome barriers to change.

"Exercise is the best medicine". Walking has been shown to increase weight loss, improve glycaemic control and reduce cardiovascular mortality in people with type 2 diabetes. Regular exercise may be more effective than medicines for the treatment of type 2 diabetes in some patients. The number needed to treat (NNT), to prevent one death per year, is reported to be 61 for people with type 2 diabetes who walk at least two hours per week.[10] This compares to a reported NNT of 141 for overweight people with diabetes who are taking metformin.[10]

Setting HbA_{1c} targets

Clinicians in partnership with patients are recommended to set individualised HbA_{1c} targets which take into consideration the potential duration of the patient's exposure to hyperglycaemia.[9] HbA_{1c} levels should be regularly monitored to enable review if targets are not being achieved.

New Zealand guidelines recommend that HbA_{1c} targets be appropriate for, and agreed with, the individual patient. In general, a HbA_{1c} target of 50 – 55 mmol/mol is recommended.[8] In younger patients, who are likely to be exposed to hyperglycaemia for longer, a lower target may be agreed, which should be balanced against the increased risk of hypoglycaemia if sulfonylureas or insulin are prescribed. Patients who have a significant risk of hypoglycaemia or its consequences, e.g. older patients, may have less stringent targets (see: "How low to go?").[9]

Management intensification is the cornerstone of all type 2 diabetes care plans and glycaemic control should be constantly revisited during consultations. Some people with type 2 diabetes may not be aware of the hidden damage that hyperglycaemia can cause, particularly if they feel they are functioning at an acceptable level. Discussing the significance of any laboratory or tests results, e.g. microalbuminuria or retinal imaging, with the patient is one way to reinforce the benefits of tighter glycaemic control.

Best Practice tip: The new *Bestpractice* Decision Support diabetes common form standardises retinal images to retinal reports and is useful for illustrating to patients the hidden damage of retinopathy.

Choosing the right tools for the job

Metformin, sulfonylureas and insulin are the front-line medicines in the management of glycaemic control in people with type 2 diabetes. When considering other medicines, e.g. when metformin or sulfonylureas are less well tolerated, contraindicated or not effective, it is important to select the right medicine for the right patient.

How low to go?

"The price of intensive glycaemic control is an increased risk of severe hypoglycaemia." [11]

Trials and systematic reviews have produced conflicting results as to what effect intensive glycaemic control has on all-cause mortality. Intensive glycaemic control in the Action to Control Cardiovascular Risk in Diabetes (ACCORD) trial and the Veterans Affairs Diabetes Trial (VADT) was defined as a HbA_{1c} target of ≤ 42 mmol/mol.[9] The Action in Diabetes and Vascular disease (ADVANCE) trial had a target level of 48 mmol/mol, and the United Kingdom Prospective Diabetes Study (UKPDS) achieved a HbA_{1c} level of 53 mmol/mol in the intensively managed arm of its trial.[9]

There was no significant change in cardiovascular or all-cause mortality in the ADVANCE or VDAT trials, although a trend towards increased mortality was seen in the VDAT trial. However, in the intensively managed groups in the ACCORD trial, there were significant increases in both cardiovascular and all-cause mortality resulting in the trial being stopped early. The extent to which this result was influenced by hypoglycaemia, or the use of newer medicines, e.g. dipeptidyl peptidase-4 inhibitors and glucagon-like peptide-1 (GLP-1), is unknown. Rates of hypoglycaemia were three-fold higher in the ACCORD trial.[9]

The United Kingdom Prospective Diabetes Study (UKPDS) ten-year follow-up demonstrated that the relative benefit of having received intensive glycaemic management was maintained despite the mean HbA_{1c} levels of the two treatment groups converging shortly after randomisation had ceased.[9] This has been termed a "legacy effect" of treatment.

These apparently conflicting results suggest that glycaemic control should not be a "one size fits all" approach. Generally, patients in the ACCORD, ADVANCE and VADT studies were older, had a longer history of diabetes at study entry and had a history of cardiovascular disease or multiple cardiovascular risk factors. Younger patients, who were recently diagnosed and had lower cardiovascular risk in the UKPDS study appeared to benefit more from intensive management. Glycaemic control should therefore be appropriate for the individual.

For further information see: "HbA_{1c} targets in people with type 2 diabetes: do they matter", BPJ 30 (Aug, 2010).

Metformin first-line

Metformin is the first-line medicine for all people with type 2 diabetes.[8] Metformin decreases glucose formation in the liver and increases peripheral utilisation of glucose. It is particularly effective in people with type 2 diabetes who are overweight.[12] Evidence now suggests that initiation of metformin for intermediate hyperglycaemia, or at the time of type 2 diabetes diagnosis, may confer cardiovascular protection beyond that provided by its blood glucose-lowering ability.[13] It is important to start patients on a low dose of metformin to avoid initial gastrointestinal adverse effects and to gradually increase the dose according to response. A typical adult starting dose is 500 mg, once daily – although it is not uncommon to start a patient on half a tablet. Generally, the total daily dose should not exceed 2 g, but in selected patients this may be increased to 3 g per day if tolerated and renal function is not impaired.[14, 15]

Lactic acidosis is a possible rare adverse effect of metformin treatment.[14] It is triggered by tissue hypoxia, which can be a feature of acute renal failure, and acute cardiac or respiratory failure. This is most commonly seen in general practice in association with chronic kidney disease (stage 4 – 5). Temporary cessation of metformin should be considered in situations which may lead to lactic acidosis.[14] Explain to patients that if they develop an illness leading to dehydration they should temporarily cease taking metformin.[14]

Doses should be reduced in patients with eGFR 30 – 60 mL/min/1.73m^2 (maximum 1 g daily).[14] Treatment should not be begun in patients with significant renal impairment (eGFR < 30 mL/min/1.73m^2) without prior discussion with a Nephrologist.[14]

Add sulfonylurea

A sulfonylurea can be added to metformin for people with type 2 diabetes who have not reached their agreed HbA_{1c} target after three months.[8] Sulfonylureas are effective at increasing insulin secretion if the patient has functional pancreatic beta-cells, but this can also cause hypoglycaemia and weight-gain.[14] Due to the risk of hypoglycaemia sulfonylureas should be avoided in patients with severe hepatic or kidney impairment.[14] Sulfonylureas are also contra-indicated in patients with ketoacidosis and should be avoided in patients with acute porphyria.[14] There are currently three fully-subsidised sulfonylureas in New Zealand – glipizide, gliclazide and glibenclamide. Glipizide and gliclazide are shorter-acting and are preferred, with caution, in older patients.[14] Glibenclamide is long-acting and should be avoided in older patients.[14] Table 1 lists recommended doses.

Insulin

Insulin is eventually required for many people with type 2 diabetes and early initiation can be appropriate. Beta-cell function declines linearly and after ten years 50% of people

Table 1: Recommended doses of sulfonylureas[14]

	Adult starting dose	Dose titration	Notes
Glipizide	2.5 – 5 mg daily, with or shortly before breakfast or lunch	Adjust according to response by 2.5 – 5 mg daily, at weekly intervals; usual maintenance dose is 2.5 – 30 mg daily, maximum 40 mg daily; no more than 15 mg in a single dose.	Divided doses are recommended for patients who have high post-prandial blood glucose
Gliclazide	40 mg daily, with breakfast	Adjust according to response, up to 160 mg in a single dose; maximum 320 mg daily	Higher doses should be divided and taken with food
Glibenclamide	2.5 – 5 mg daily, with or immediately after breakfast	Adjust according to response by 2.5 mg daily, every one to two weeks; maximum 10 mg as a single dose; maximum 15 mg daily	Long-acting; not recommended for use in older people

with type 2 diabetes will require insulin.[16] Insulin has a greater blood glucose lowering ability than any other hypoglycaemic medicine, and early initiation may reduce beta-cell damage and is thought to slow disease progression.[17] Early initiation of insulin should be strongly considered for people with type 2 diabetes who have significant hyperglycaemia, e.g. HbA_{1c} > 65 mmol/mol, particularly if there are signs such as ketonuria and weight loss.[8] If there are immediate health concerns, insulin initiation, even if temporary, may be the only treatment option. However, it is important to remember that type 1 diabetes can occur at any age and if there are severe signs, or rapid progression, then testing for autoantibodies may be appropriate. Women with type 2 diabetes who become pregnant almost always require initiation of insulin treatment.[12]

For further information see: "Initiating insulin for people with type 2 diabetes", BPJ 42 (Feb, 2012).

Additional treatments require extra considerations

Alternative medicines may need to be considered for select patients where:

- Glycaemic control remains poor following standard treatment
- There is a significant risk of hypoglycaemia, or the patient's circumstances places them at risk if hypoglycaemia does occur, e.g. lives alone
- Standard treatments are either not tolerated or are contraindicated
- Doses of standard treatments cannot be increased

When discussing further treatment options with patients it is important to consider their age, the risk if hypoglycaemia occurs, the potential for weight gain associated with treatment and their preferences regarding the management of adverse effects. Table 2 provides an approximate comparison of the relative efficacy of oral anti-diabetic medicines available in New Zealand.

α-Glucosidase inhibitors

Acarbose is the most widely studied α-glucosidase inhibitor and is the only fully-subsidised medicine in this class available in New Zealand. Acarbose is a safe and mildly effective medicine for improving glycaemic control. It is taken orally and reduces the amount of glucose absorbed in the small intestine by blocking the α-glucosidase enzyme, which breaks down complex carbohydrates into glucose. Acarbose is the most effective oral anti-diabetic medicine available in New Zealand for reducing post-prandial hyperglycaemia, which is thought to be a significant contributor to cardiovascular disease and the microvascular complications of type 2 diabetes.[19] However, it has little effect on fasting glucose levels.

Acarbose can be used as a first-line treatment where metformin or sulfonylurea are contraindicated or not tolerated.[20] When taken as a monotherapy, acarbose does not increase the risk of hypoglycaemia.

Acarbose can also be added to any of the oral anti-diabetic medicines, and insulin, if monotherapy with these medicines fails to achieve HbA_{1c} targets and post-prandial glucose levels continue to be a concern.[19] When used in combination

Table 2: The relative efficacy of anti-diabetic medicines available in New Zealand, adapted from Klam et al (2006)[18]

Medicine	Dose interval	Expected HbA_{1c} reduction (mmol/mol)*
Metformin	One – three times daily	12 – 22
Sulfonylureas	One – three times daily	15 – 20
Acarbose	Three times daily	6 – 11
Pioglitazone	One – two times daily	20 – 21

* The expected reduction is an estimate that excluded the highest and lowest effects reported by studies

with a sulfonylurea or insulin, acarbose may enhance the hypoglycaemic effect of these medicines. If hypoglycaemia occurs in this situation, because of the enzyme-inhibiting action of acarbose, patients should consume glucose, not sucrose which is a complex carbohydrate, e.g. glucose tablets not jellybeans.

How to initiate and monitor acarbose use

Acarbose is available in 50 mg and 100 mg tablets, which should be chewed and swallowed with water immediately before eating, or with the first mouthful of food.[14] Adults begin with 50 mg, three times daily, which is increased to 100 mg, three times daily, after four to eight weeks.[14] The maximum recommended dose is 200 mg, three times daily.[14]

Acarbose adverse effects and contraindications

Flatulence is reported by approximately three-quarters of people taking acarbose. Soft stools and diarrhoea are also common.[19] Abdominal distension, pain and rarely, hepatitis have also been reported.[19]

Acarbose is contraindicated in people who: are pregnant, have hepatic or renal impairment (eGFR < 25 mL/minute/1.73m^2), have inflammatory bowel disease or a history of intestinal obstruction or hernia, have had previous abdominal surgery or have a gastrointestinal disorder with malabsorption.[14]

Glitazones (pioglitazone)

The glitazones are oral anti-diabetic medicines which are classified as insulin sensitisers (like metformin) because they increase the body's ability to transport glucose across cell membranes. When used as monotherapy, glitazones do not cause hypoglycaemia.[9] Glitazone use has been associated with heart failure (see "Glitazone use and cardiovascular risk"), bladder cancer and increased risk of bone fractures.

In New Zealand, pioglitazone is the only glitazone approved for use in the treatment of type 2 diabetes. It is available under Special Authority criteria to patients who are already taking maximum doses of metformin or a sulfonylurea, or where one or both medicines are contraindicated or not tolerated, or for patients taking insulin who have not achieved glycaemic control. Rosiglitazone was available in New Zealand, but has now been withdrawn due to concerns about adverse cardiovascular effects (see "Glitazone use and cardiovascular risk").

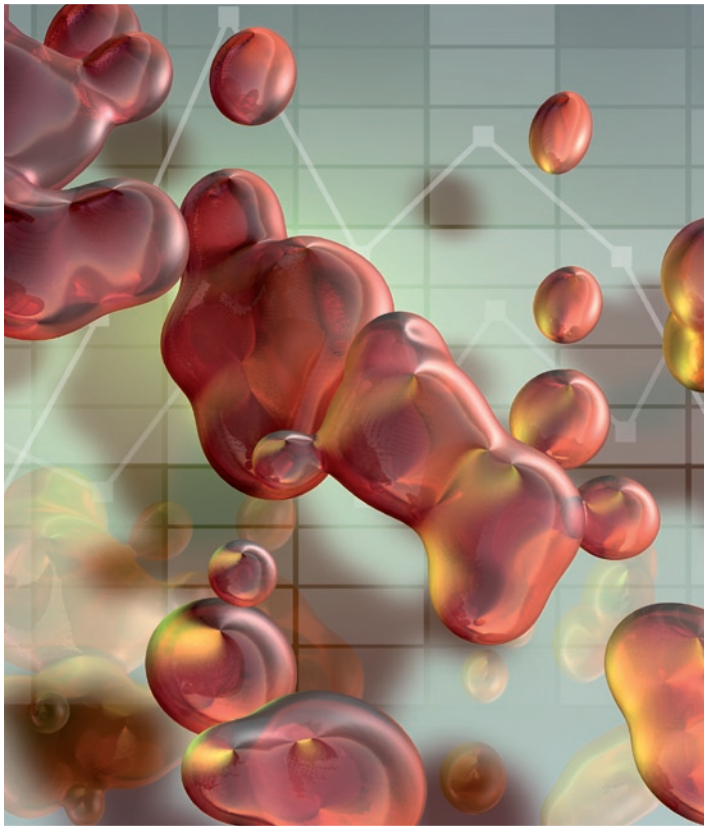
Pioglitazone may be cautiously considered in select patients

NICE guidance states that pioglitazone may be considered

Glitazone use and cardiovascular risk

In 2010, the United States Food and Drug Administration (FDA) placed restrictions on the prescribing of rosiglitazone following concerns that its use was associated with myocardial ischemia (N.B. the FDA is currently reconsidering this decision).[21] NICE guidelines note a possible increased risk of myocardial ischaemia associated with the use of rosiglitazone, which is further increased with concurrent use of insulin.[22] Rosiglitazone is no longer available in New Zealand.

The extent to which glitazones increase the risk of heart failure is complicated, as many studies reporting on the safety of anti-diabetic medicines are of limited duration and follow-up is often short, or the studies are not designed to assess cardiovascular effects. A meta-analysis of 140 randomised controlled trials found that there was moderately strong evidence to suggest either pioglitzone or rosiglitazone use increased the risk of congestive heart failure in comparison to use of sulfonylureas.[21] However, research on this topic is ongoing.



cautiously as:[22]

- A second-line treatment to metformin, instead of a sulfonylurea, if HbA_{1c} is ≥ 50 mmol/mol, or greater than agreed target, and the person is at significant risk of hypoglycaemia or its consequences, e.g. works at heights or lives alone, or sulfonylurea treatment is not tolerated or is contraindicated
- A second-line treatment to first-line sulfonylurea if HbA_{1c} is ≥ 50 mmol/mol, or greater than agreed target, and the patient does not tolerate metformin, or metformin is contraindicated
- A third-line option to add to metformin and sulfonylurea if HbA_{1c} is ≥ 59 mmol/mol or greater than agreed target, and insulin treatment is either inappropriate, or unacceptable
- A combination treatment with insulin if the patient has previously had a therapeutic response to pioglitazone treatment, or the patient is already taking high-dose insulin and their blood glucose is inadequately controlled

How to initiate pioglitazone

Pioglitazone is available in 15 mg, 30 mg and 45 mg tablets. The recommended starting dose for adults is 15 – 30 mg, once daily.[14] The concurrent use of other hypoglycaemic medicines may increase the risk of hypoglycaemia and influence the starting dose, e.g. 15 mg, once daily may be more appropriate for patients taking insulin.[14, 23] The dose may be increased after four weeks to 45 mg, once daily, if greater therapeutic effect is required.[23] If a sulfonylurea or insulin is being taken concurrently then doses of these medicines may need to be reduced.

Pioglitazone should only be continued beyond six months in patients who have achieved at least a 5 mmol/mol reduction in HbA_{1c}.[22]

The adverse effects of pioglitazone

Pioglitazone can cause weight gain, fluid retention, peripheral oedema and expansion of plasma volume, which can increase the risk of anaemia and heart failure.[22] Pioglitazone is contraindicated in people with a history of heart failure.[14]

In New Zealand, in 2009, there were five new registrations of bladder cancer per 100 000 people.[24] Bladder cancer risk is increased by 40% in people with type 2 diabetes.[25] One study found that this risk was increased almost two-fold in people with type 2 diabetes who take pioglitazone, with those who use the medicine for longer, or at higher doses, exposed to the greatest risk.[26] Pioglitazone use is contraindicated in patients with a history of bladder cancer, or in patients with un-investigated haematuria.[27] Assess the risk factors for bladder cancer, e.g. age, smoking history and history of chronic bladder infections, before considering pioglitazone treatment and use with extreme caution in older patients who have an increased risk of bladder cancer, as well as heart failure.[13] Bladder symptoms, in particular haematuria, should be investigated promptly in people taking pioglitazone.

Liver function should be assessed before pioglitazone treatment is begun and then monitored periodically.[14] If patients develop symptoms indicating liver toxicity, e.g. nausea, abdominal pain, dark urine or jaundice, the medicine should be stopped.[14]

The long-term use of glitazones is associated with an increased risk of bone fractures in women.[22] Pioglitazone should not be initiated in people who are at increased risk of bone fracture, e.g. people with oesteoporosis.[22]

Pioglitazone use is also associated with weight gain. The addition of pioglitazone to insulin significantly increases weight gain; 2.3 – 4.9 kg with insulin plus pioglitazone compared to 0.04 – 2.4 kg with insulin alone.[22] Pioglitazone in combination with insulin increases the risk of hypoglycaemia.[22]

Glucose-lowering medicines with novel actions

GLP-1 (glucagon-like peptide 1) agonists, are medicines which mimic endogenous incretins, which are peptides with short half-lives that are secreted from the gut following a meal. The first GLP-1 agonist was derived from extracts of the salivary secretions of the lizard *Heloderma suspectum* (Gila monster) which eats once a month and therefore needs to be able to rapidly increase insulin production as required.[22] Administration of GLP-1 enhances endogenous secretion of insulin following eating and inhibits glucagon secretion.[28] It is also reported to suppress appetite and food intake and is associated with weight loss in overweight or obese people with, or without, type 2 diabetes.[28]

GLP-1 treatment has recently been reported to increase the likelihood of acute pancreatitis approximately two-fold in people with type 2 diabetes compared to a control group matched for age, sex and diabetes related complications.[29] There are also recent concerns that the use of GLP-1 agonists may increase the longer term risk of pancreatic tumours.[30, 31] The American Diabetes Association is now requesting that pharmaceutical companies make patient-level data available for independent review to investigate the link between incretin treatment (including Dipeptidyl peptidase-4, see:

"Anti-diabetic medicines not commonly encountered in New Zealand") and pancreatic abnormalities.[32]

Exenatide (subcutaneous injection) is a GLP-1 agonist approved for use in New Zealand, but not subsidised, as an adjunctive treatment for type 2 diabetes. [14] NICE guidelines recommend that exenatide may be considered as a third-line treatment, in addition to metformin and sulfonylurea, when glycaemic control is inadequate, e.g. ≥ 59 mmol/mol, or as individually agreed, and:[22]

- The patient has a body mass index (BMI) ≥ 35 kg/m^2; or
- The patient has a BMI < 35 kg/m^2 and insulin treatment is inappropriate or the patient is at risk from obesity-related complications

Exenatide is injected twice daily within one hour of the two main meals, at least six hours apart.[14]

Trials have shown exenatide to be effective in reducing HbA_{1c} levels by approximately 10 mmol/mol and to be associated with a reduction in body weight of 1 – 1.5 kg when added to metformin and sulfonylurea treatment.[22] Adverse effects include nausea and occasionally vomiting or diarrhoea when beginning treatment. Exenatide should not be initiated or continued in any patient with a history of pancreatitis.[33]

Surgery is an option if medicines are ineffective

Surgical intervention is an effective option for selected patients who are obese (BMI > 35 kg/m^2), when lifestyle interventions and medicines are ineffective.[34] Gastric bypass surgery and biliopancreatic diversion surgery are reported to have an NNT for diabetes remission at two-year follow-up of 1.3 and 1 respectively.[34] It is unknown for how long people who have had surgery can maintain this level of glycaemic control, and there are preliminary reports that by ten-year follow-up remission rates fall substantially.[34] Further studies are also required to determine the effects of surgery on mortality and long-term morbidity and the extent to which gastric surgery can result in nutritional deficiencies.

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Anti-diabetic medicines not commonly encountered in New Zealand

Dipeptidyl peptidase-4 (DPP-4) inhibitors block the enzyme which degrades the incretins. Sitagliptin and saxagliptin are approved in New Zealand, but not subsidised, as oral adjunctive treatments for type 2 diabetes. They may be considered as a second-line treatment to metformin or sulfonylurea, or third-line to metformin plus sulfonylurea.[22] DPP-4 inhibitors are not associated with weight gain and may be considered in preference to pioglitazone where weight gain is a concern, or pioglitazone is contraindicated or does not produce a sufficient response.[22] Unlike exenatide these medicines require only once daily oral dosing. Long-term trials are required to confirm the safety of DPP-4 inhibitors as the DPP-4 enzyme is active against other peptides and, as with GLP-1 agonists, safety in regards to the possible association with pancreatic changes need to be clarified.[30, 31]

The meglitinides are a class of medicine that, like sulfonylureas, increase insulin secretion. They are used if sulfonylureas are not tolerated. There are two anti-diabetic medicines in this class, repaglinide and nateglinide, however, neither are available in New Zealand.

PHO Performance Programme – Diabetes follow-up after detection

Diabetes follow-up after detection is a PHO Performance Indicator that accounts for 9% of performance funding; 6% for the total population and 3% for the high need population.[35] High need populations include Māori and Pacific peoples and people living in NZDep 9 & 10 (most deprived) socioeconomic areas. The target population is all people aged 15 to 79 years who have been identified as having diabetes.[35]

The programme goal is for 90% of individuals identified as having diabetes to have had an annual diabetes review.[35] The target is assessed by counting the number of enrolled people in a PHO with a record of an annual diabetes review (the numerator). This number is then divided by the number of people in the PHO who would be expected to have been diagnosed with diabetes using prevalence estimate data (the denominator).[35]

PHOs that have not achieved the programme goal are expected to make annual increases in the number of people with diabetes who have had an annual review in order to receive funding. In 2011, the diabetes prevalence figures for New Zealand were updated, therefore currently PHO progress data should be treated with caution.[36] Reported performance against this indicator has fallen to 63.8% for the high need population and 62% for the total population. The transition to the Diabetes Care Improvement Package from the "Get checked" programme may explain why there has been a reduction in the reporting of annual diabetic reviews.[36]

Further information regarding the PHO Performance Programme is available from: **www.dhbsharedservices.health.nz/Site/SIG/pho/Default.aspx**

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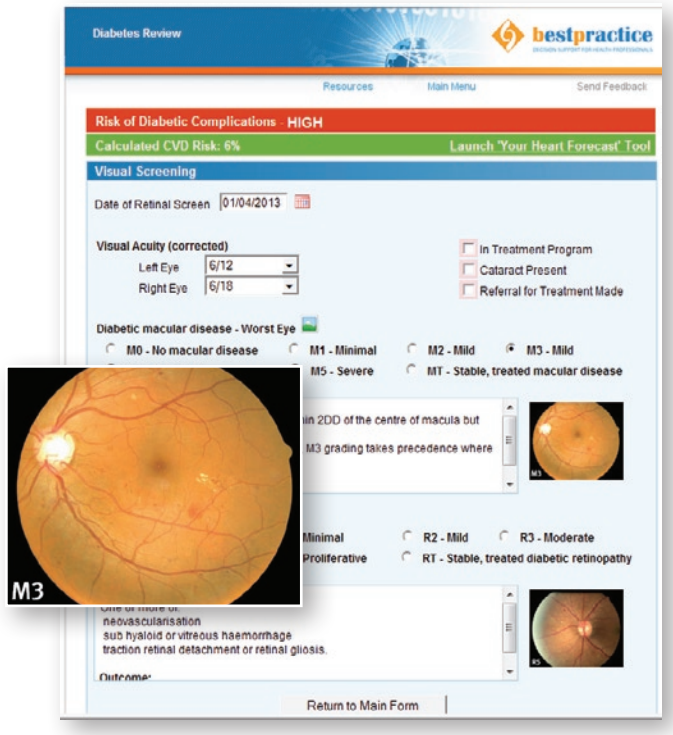
COMMON FORM

The **Common Form** combines features from the Diabetes and CVD modules to produce a streamlined standardised tool that assists in clinical review, disease monitoring and clinical management.

The **Common Form** module features the matching of retinal screening reports to standardised retinal images. The effects of microvascular complications can be visibly demonstrated to patients to facilitate understanding of their condition and as a method to reinforce good glycaemic control.



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