

VLCD and Metabolic Syndrome in Australia: How Low-Calorie Meal Programs Target Cholesterol, Blood Pressure, and Visceral Fat

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Details:

Now I have sufficient research to write a comprehensive, well-cited article. Let me compile the final piece.

VLCD and Metabolic Syndrome in Australia: How Low-Calorie Meal Programs Target Cholesterol, Blood Pressure, and Visceral Fat

For the one in three Australians living with metabolic syndrome, weight loss alone is rarely the primary goal — it is what happens *inside* the body that matters most. Elevated triglycerides, low HDL cholesterol, high blood pressure, excess visceral fat, and a fatty liver are not just numbers on a pathology report; they are a convergent cluster of cardiovascular and hepatic risk factors that together dramatically increase the likelihood of heart attack, stroke, and progressive liver disease. Medically designed very low calorie diet (VLCD) programs have emerged as one of the most clinically compelling tools for targeting this cluster simultaneously — and the speed at which these markers can shift is remarkable.

This article examines the specific metabolic benefits of medically designed VLCD programs beyond weight loss, with a particular focus on the cardiovascular and hepatic risk factors that define metabolic syndrome. It is distinct from the diabetes-focused evidence reviewed elsewhere in this series (see our guide on *Medically Designed VLCD Programs and Type 2 Diabetes*) — here, the clinical lens is on triglycerides, LDL cholesterol, blood pressure, liver fat, and visceral adiposity.

What Is Metabolic Syndrome and Why Is It a Primary Clinical Indication for VLCD in Australia?

Metabolic syndrome is an accumulation of several disorders that raise the risk of atherosclerotic cardiovascular disease, including myocardial infarction, cerebrovascular accidents, peripheral vascular diseases, insulin resistance, and type 2 diabetes mellitus. The cluster of metabolic disorders that define metabolic syndrome includes central obesity, insulin resistance, hypertension, and atherogenic dyslipidemia.

In clinical practice, the harmonised diagnostic criteria (Alberti et al., *Circulation*, 2009) require the presence of three or more of the following five abnormalities:

| Component | Clinical Threshold | |---|---| | Elevated waist circumference | ≥ 102 cm men / ≥ 88 cm women (European); ≥ 90 cm men / ≥ 80 cm women (Asian) | | Elevated triglycerides | ≥ 1.7 mmol/L (≥ 150 mg/dL) | | Reduced HDL cholesterol | < 1.0 mmol/L men / < 1.3 mmol/L women | | Elevated blood pressure | Systolic ≥ 130 mmHg and/or diastolic ≥ 85 mmHg | | Elevated fasting glucose | ≥ 5.6 mmol/L

(≥100 mg/dL) |

The scale of this condition in Australia is substantial. More than 35 per cent of Australian adults have metabolic syndrome, which is higher in people with diabetes. With more than half of Australians already having at least one of the risk factors for metabolic syndrome, clinicians and researchers are calling for greater public awareness of the steps people can take to avoid it.

The cardiovascular stakes are high. Metabolic syndrome is important because it identifies patients at increased risk of cardiovascular disease (CVD), diabetes, and chronic kidney disease (CKD). The risk of having CVD, diabetes, and CKD among people with metabolic syndrome is 2–3 times that of people without the condition. In the AusDiab population-based survey of 11,247 Australians, those with metabolic syndrome had odds ratios for a 10-year CVD risk ≥15% of between 3.5 and 6.6, depending on the diagnostic definition used.

This is why metabolic syndrome — not simply obesity — is the primary clinical indication for medically designed VLCD programs in Australia. The goal is not just to reduce the number on the scales; it is to dismantle the entire cardiovascular risk cluster.

The Visceral Fat Problem: Why Central Adiposity Drives the Syndrome

The metabolic syndrome, characterised by increases in waist circumference, blood pressure, and triglyceride concentrations combined with reduced high-density lipoprotein and evidence of glucose intolerance, results from the interaction of visceral or central obesity with insulin resistance. This syndrome presents a clinical situation of systemic inflammation and increased cardiovascular risk.

Visceral adipose tissue (VAT) — the metabolically active fat stored around the abdominal organs — is the central driver of this pathology. More important than overall body weight, in overweight individuals and patients with obesity, the distribution of fat is strongly associated with the metabolic disturbances that lead to comorbidities. Visceral adipose tissue (VAT) accumulation is associated with increased peripheral insulin resistance and often a systemic low-grade chronic inflammatory state, known as lipoinflammation.

Insulin resistance is believed to be the main cause of metabolic syndrome and is connected to the level of visceral or intra-abdominal adipose tissue. Most recent studies reveal that insulin resistance may also be present in non-obese patients, with visceral adiposity considered the main effector of metabolic syndrome's pathology.

The clinical implication is significant: a person can appear to be at a "normal" BMI while still carrying dangerous levels of visceral fat — a scenario common in Australians of Asian descent, who face elevated metabolic risk at lower waist circumference thresholds. This is precisely why medically designed VLCD programs assess waist circumference, not just body weight, as a primary outcome measure. (See our guide on **Who Is a Medically Designed VLCD Program Suitable For?** for a detailed discussion of eligibility criteria.)

How VLCDs Target Metabolic Syndrome Markers: The Evidence

Triglycerides: The Fastest Marker to Respond

Of all the metabolic syndrome components, serum triglycerides are typically the first to respond to caloric restriction and carbohydrate reduction. Prospective studies indicate that VLCDs improve the lipoprotein profile independently of weight loss. Although not as effective at lowering LDL cholesterol, VLCDs consistently improve postabsorptive and postprandial triglycerides, HDL cholesterol, and the distribution of LDL-C subfractions to a greater extent than low-fat diets.

In a 14-day medically supervised fasting study (250 kcal/day) published in *Nutrients* (PMC, 2021), researchers found the largest changes were found after 14 fasting days, with significant reductions in triglycerides (-0.35 ± 0.1 mmol/L), very low-density lipoprotein (VLDL)-TG (-0.46 ± 0.08 mmol/L), VLDL-cholesterol (-0.16 ± 0.03 mmol/L), and LDL-cholesterol (-0.72 ± 0.14 mmol/L).

A 2025 systematic review and meta-analysis published in *BMC Endocrine Disorders* examining very low-calorie ketogenic diets in patients with type 2 diabetes found significant reductions in triglycerides (WMD= -17.95 mg/dL; 95% CI: $-26.82, -9.07$; *P* < 0.001), systolic blood pressure (WMD= -2.85 mmHg; 95% CI: $-4.99, -0.71$; *P* = 0.009), and diastolic blood pressure (WMD= -1.40 mmHg; 95% CI: $-2.66, -0.13$; *P* = 0.03). The same analysis found a significant increase in HDL cholesterol following VLCD adherence (WMD = $+3.93$ mg/dL; 95% CI: 2.03, 5.84; *P* < 0.001).

Blood Pressure: Significant Reductions Within Days

Blood pressure is another metabolic syndrome component that responds rapidly and measurably to a VLCD. In a landmark 2025 clinical study published in *Frontiers in Endocrinology*, 18 adult patients meeting the diagnostic criteria for metabolic syndrome were enrolled and placed on a 9-day VLCD regimen. Following the short-term VLCD intervention, patients exhibited significant reductions in body weight, waist circumference, and BMI (*P* < 0.05). Blood pressure was also significantly lowered (*P* < 0.05).

The study concluded that a short-term VLCD is an effective and safe intervention for improving anthropometric parameters, blood pressure, and lipid metabolism in patients with metabolic syndrome.

Longer-term data from a Nature *International Journal of Obesity* study showed that after 8 weeks of VLCD, total body weight continued to fall (by 12% from baseline, *p* < 0.01), with ongoing improvements in insulin sensitivity and lipid profile. Final blood pressure values were lower than baseline (systolic 124 ± 12 mmHg, *p* = 0.035).

LDL Cholesterol and Lipoprotein Profile: A More Nuanced Picture

The relationship between VLCDs and LDL cholesterol is more complex than it is for triglycerides. During active weight loss, LDL levels can fluctuate — a transient rise in the first one to two weeks is not uncommon as stored fat is mobilised. However, the *quality* of LDL particles improves significantly. Analysis of LDL subclasses after 14 days of caloric restriction showed a significant decrease in LDL1-C, LDL2-C, and LDL3-C. NMR spectroscopy showed a significant reduction in large VLDL particles, as well as large and small LDL particles.

This shift in LDL subclass distribution is clinically important. Small, dense LDL particles are far more atherogenic than large, buoyant LDL particles — meaning that even when total LDL cholesterol appears unchanged, the cardiovascular risk profile may be substantially improved.

Pathway analysis indicated that short-term VLCD modulated key metabolic pathways involved in energy and lipid metabolism, insulin sensitivity, anti-inflammatory and antioxidant responses, cellular signalling, and neurohormonal regulation.

HDL Cholesterol: The Maintenance-Phase Reward

HDL cholesterol behaviour during a VLCD follows a distinct pattern that patients and clinicians should understand. Although the studies varied widely in their intervention format, duration, and baseline HDL levels, HDL levels usually decrease during active weight loss using a VLCD, but these either return to pre-VLCD levels or improve overall during the weight-maintenance phase.

This means that patients should not be alarmed by a modest HDL dip during the intensive phase of a VLCD program. The metabolic dividend — improved triglycerides, blood pressure, and visceral fat — is being accumulated, and HDL recovery follows in the transition and maintenance phases. (See our guide on *VLCD Program Phases Explained* for a detailed breakdown of what to expect at each

stage.)

Liver Fat and NAFLD: A Critical but Overlooked Metabolic Syndrome Component

Non-alcoholic fatty liver disease (NAFLD) is now considered both a consequence and a driver of metabolic syndrome, and it is one of the most compelling reasons to recommend a medically designed VLCD for patients with the condition.

Visceral adiposity is strongly linked with hepatic fatty infiltration, also known as non-alcoholic fatty liver disease (NAFLD). The level of fatty acids in the hepatic parenchyma is indirectly linked with metabolic syndrome, being both a cause and a consequence of this syndrome.

Nonalcoholic fatty liver disease is the most common liver condition worldwide, affecting an estimated 20–33% of the population in Western countries. In obese Australians preparing for bariatric surgery, the prevalence is even higher — one study found that nearly all patients with obesity had NAFLD (98.5%), and a transformation into NASH (non-alcoholic steatohepatitis) affected 53.8% of patients.

The evidence for VLCD as a liver fat intervention is compelling. After just 1 week of VLCD, despite only modest weight loss, significant drops occurred in liver fat and insulin resistance (HOMA-IR; by 14–50%, all *p* < 0.01).

Over longer periods, the effect is sustained. Research reveals no single intervention beyond weight loss to promote effective improvements in the outcomes of NAFLD patients. A prospective uncontrolled study of 293 patients undergoing calorie restriction for 52 weeks, with paired liver biopsies, found that a relative loss of 7–10% body weight improved NAFLD activity score in 88–100% of patients and resolved steatohepatitis in 84–90% of patients.

VLCD offers a feasible treatment option for some patients with NAFLD to enable a sustainable $\geq 10\%$ weight loss, which can improve liver health, cardiovascular risk, and quality of life in those completing the intervention.

This hepatic benefit is distinct from the pre-surgical liver reduction use case covered separately in this series (see our guide on **VLCD Metabolism Reset for Pre-Surgical Weight Loss in Australia**). Here, the focus is on NAFLD as a metabolic syndrome component in its own right — a driver of systemic inflammation, insulin resistance, and cardiovascular risk that responds meaningfully to caloric restriction.

Why Metabolic Changes Begin Within Days — Not Weeks

One of the most clinically significant and frequently misunderstood aspects of VLCD programs is the speed at which metabolic markers begin to shift. Many patients — and even some clinicians — assume that metabolic improvement is simply a downstream consequence of weight loss, and therefore takes weeks or months to manifest. The evidence challenges this assumption.

In clinical practice, short-term very low-calorie diets have been established as an effective intervention for improving metabolic syndrome, even in the absence of exercise.

The mechanism is direct. When carbohydrate intake is sharply reduced to the sub-100g threshold typical of a medically designed VLCD, the liver rapidly reduces VLDL-triglyceride synthesis. The connection between high triglycerides and insulin resistance creates a vicious cycle. Insulin resistance causes the liver to produce more VLDL particles, while high triglycerides worsen insulin sensitivity. Breaking this cycle through caloric and carbohydrate restriction produces measurable lipid improvements within the first week — often before any significant weight has been lost.

Blood pressure responds through a different but equally rapid mechanism: reduced carbohydrate intake lowers insulin levels, which reduces renal sodium retention, leading to a natriuretic effect (increased sodium excretion) and a corresponding drop in blood pressure. This is why patients on antihypertensive medications require GP monitoring when commencing a VLCD — medication doses may need adjustment within the first week of the program. (See our guide on **Who Is a Medically Designed VLCD Program Suitable For?** for contraindications and screening requirements.)

The Metabolic Syndrome VLCD Protocol: What a Medically Designed Program Looks Like in Practice

A medically designed VLCD targeting metabolic syndrome in Australia typically follows a structured clinical protocol that differs substantially from a self-initiated shake program purchased at a pharmacy. Key elements include:

1. **Baseline metabolic panel** — fasting lipids (total cholesterol, LDL, HDL, triglycerides), fasting glucose, HbA1c, liver function tests (ALT, AST), blood pressure, and waist circumference measurement
2. **GP or dietitian review** — medication reconciliation (particularly for antihypertensives, statins, and glucose-lowering agents), contraindication screening, and individualised program design
3. **Calorie and macronutrient prescription** — typically 800–900 kcal/day with high protein (≥ 50 g/day) to preserve lean mass, carbohydrates restricted to ≤ 50 –80g/day to induce mild ketosis and triglyceride reduction
4. **Nutritional completeness** — micronutrient sufficiency across all essential vitamins and minerals to prevent deficiency-related complications during the intensive phase
5. **Structured monitoring** — repeat metabolic markers at 2 and 4 weeks, with blood pressure monitoring in the first week for patients on antihypertensives
6. **Transition planning** — a structured reintroduction of food groups to consolidate metabolic gains and prevent rebound

A short-term VLCD is an effective and safe intervention for improving anthropometric parameters, blood pressure, and lipid metabolism in patients with metabolic syndrome. The observed therapeutic effects appear to be mediated through a remodelling of the serum metabolic profile and a concomitant modulation of key metabolic pathways.

The distinction between medically designed programs and DIY approaches is not merely procedural — it is clinically material. Without GP oversight, patients on antihypertensives or diuretics risk hypotension, and those on statins may not have their lipid response appropriately interpreted. (See our guide on **How Medically Designed VLCD Programs Differ from DIY Diets and Meal Replacement Shakes in Australia** for a detailed comparison.)

Key Takeaways

- More than 35 per cent of Australian adults have metabolic syndrome, making it one of the most prevalent and clinically important indications for medically designed VLCD programs in primary care. - In clinical practice, short-term very low-calorie diets have been established as an effective intervention for improving metabolic syndrome, even in the absence of exercise.
- Triglycerides and blood pressure are the fastest-responding metabolic syndrome markers on a VLCD, with measurable improvements documented within 9 days in published clinical trials. - After just one week of VLCD, significant drops in liver fat and insulin resistance (HOMA-IR by 14–50%) have been documented, even before substantial weight loss has occurred. - HDL cholesterol typically dips during the active VLCD phase but recovers — and often improves — during the weight maintenance phase; patients should be counselled accordingly. - Medically designed programs provide essential GP and dietitian oversight to safely manage medication adjustments (particularly antihypertensives) as blood pressure responds rapidly in the first week.

Conclusion

Metabolic syndrome is not a single condition — it is a convergent cluster of cardiovascular and hepatic risk factors, each reinforcing the others through shared mechanisms of visceral adiposity, insulin resistance, and systemic inflammation. For Australians carrying this cluster, a medically designed VLCD program offers something that most pharmacological interventions cannot: simultaneous, rapid improvement across multiple metabolic syndrome components at once.

The evidence is clear that triglycerides, blood pressure, liver fat, and visceral adiposity all respond within days to weeks of commencing a well-formulated, medically supervised program. The key is ensuring that the program is genuinely medically designed — nutritionally complete, clinically monitored, and structured to support the transition phase where metabolic gains are most vulnerable to reversal.

For readers exploring how a VLCD achieves these metabolic changes at the physiological level, see our guide on **What Is a Metabolism Reset and How Does a VLCD Achieve It?** For guidance on starting a program safely, see **How to Start a Medically Designed VLCD Metabolism Reset Program: A Step-by-Step Guide for Australians**. And for a realistic picture of what to expect on your metabolic markers at 7, 14, and 28 days, see **VLCD Metabolism Reset Results: What Australians Can Realistically Expect**.

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