MALAYSIAN DIETITIANS' ASSOCIATION



Medical Nutrition Therapy (MNT)

Guidelines for Dyslipidemia



MALAYSIAN ASSOCIATION DIETITIANS' 1994

MINISTRY OF HEALTH, MALAYSIA

Second Edition
2026

Medical Nutrition Therapy Guidelines for Hypertension (second edition) Working Group



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Acknowledgement:

We would like to extend our gratitude and appreciation to:

- National Heart Institute for the use of the meeting rooms and refreshments.
- Cardiac Vascular Sentral, Kuala Lumpur for the use of the meeting rooms.
- The peer reviewers for their time and professional expertise.

Disclaimer

All committee members declare no competing interests.

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LIST OF ABBREVIATIONS

AHA/ACC: American Heart Association/ American College of Cardiology

AND: Academy of Nutrition and Dietetics

ASCVD: Atherosclerotic cardiovascular disease

BMI: Body mass index

CHD: Coronary heart disease

CI: Confidence interval

CPG: Clinical Practice Guidelines

CVD: Cardiovascular disease

DHA: Docosahexaenoic acid

EPA: Eicosapentaenoic acid

ESC: European Society of Cardiology

GRADE: The grading of recommendations assessment, development, and evaluation

HDL: High-density lipoprotein

HR: Hazard ratio

LDL: Low-density lipoprotein

MUFA: Monounsaturated fatty acid

NHFA: National Heart Foundation of Australia

PEN: Practice based Evidence Nutrition

PUFA: Polyunsaturated fatty acid

RACGP: Royal Australian College of General Practitioners

RCT: Randomised control trials

RR: Risk ratio

SFA: Saturated fatty acid

Tbsp: Tablespoon

TG: Triglycerides

WMD: Weighted mean difference

Chapter 1: INTRODUCTION

Dyslipidemia is described as high levels of total cholesterol, low-density lipoprotein (LDL) cholesterol, and triglyceride (TG), and low levels of high-density lipoprotein (HDL) cholesterol. Dyslipidemia is a major risk factor for atherosclerosis which leads to several cardiovascular diseases (CVD) [1]. The different types of dyslipidemia are as described in appendix A.

The prevalence of dyslipidemia among the Southeast Asian (30.3%) were reported to be much lesser than that among European (53.7%) and the American (47.7%) [2]. In Malaysia, a cohort study (106,527 participants, aged 35 to 70 years) demonstrated that the prevalence of hypercholesterolemia (TC≥ 6.2 mmol/L) was 44.9% in total population, and 51.0%, 40.8%, 41.6% and 34.4% by ethnic groups of Malays, Chinese, Indians and others, respectively [3].

This is an updated and revised Medical Nutrition Therapy (MNT) guideline for adult patients with dyslipidemia. This guideline supersedes the previous MNT published in 2005 and incorporates the best available evidence up to 2024.

1.1 STATEMENT OF INTENT

This guideline is intended to guide dietitians involved in providing Medical Nutrition Therapy (MNT) to adult patients with dyslipidemia. The development of this guideline is based on the best available current evidence up to 2024, together with relevant recommendations, expert opinion, and clinical practice considerations. Adherence to this guideline alone may not necessarily guarantee the best outcome in every case. Individual patient presentations, precise nutrition diagnosis and clinical judgment remain paramount in clinical practice decisions and management.

1.2 OBJECTIVES

The aim of this guideline is to provide evidence-based recommendations to assist dietitians in providing Medical Nutrition Therapy to adult patients with dyslipidemia.

1.3 CLINICAL QUESTIONS

The clinical questions were developed and divided into subtitles and members of the working group were assigned topics within these subtitles.
The clinical questions of this guideline were:

a) What are the nutrition assessments to be performed?
b) What nutrition diagnosis to be considered?
c) What are the nutritional interventions for patients with dyslipidemia?
    i. Nutrient recommendations
    ii. Lifestyle modifications (weight management, exercise, smoking, alcohol & caffeine)
    iii. Nutrition education in practice
d) What are the nutritional monitoring and evaluation for patients with dyslipidemia

1.4 TARGET POPULATION

The recommendations in this guideline are applicable to adult patients with dyslipidemia and those at risk of developing cardiovascular disease (CVD).

1.5 TARGET GROUP

This guideline is designed mainly for dietitians who are involved in provision of nutrition therapy to adult patients with dyslipidemia and those at risk of developing CVD. Other healthcare professionals including doctors, nurses and pharmacists may use this guideline as reference.

1.6 LEVELS OF EVIDENCE AND GRADE OF RECOMMENDATION

The recommendations in this guideline were mainly adapted from international guidelines on nutrition therapy for patients with hypertension namely ACC/AHA 2019 [4], ESC 2021 [5], Canadian guidelines for management of dyslipidemia 2021 [6], RACGP 2018 [7], NHFA 2017 [8], PEN 2020 [9], JAS 2018 [10], Malaysian CPG for Dyslipidemia 2017 [11], Singapore CPG for Lipids 2017 [12], Taiwan lipid guidelines 2017 [13], and Korean guidelines for management of dyslipidemia 2016 [14]. When there are clinical questions not answered by these guidelines, additional literature search through electronic databases such as PubMed, Cochrane, Science Direct, and Google Scholar was performed to identify relevant articles. The literature search of this guideline was updated until 31st December 2021. Evidence in the guidelines and literature were discussed, tabulated in the evidence table and suitable statements and recommendations were formulated.

Consensus from the working group was used in the areas of inconclusive or insufficient evidence. In addition to the evidence-based practices, other factors such as cultural practices, individual patient conditions, preferences, availability of resources and cost were critically appraised in the process of decision-making.

The Evidence Grading System used is shown in Table 1 and 2. The level of evidence is based on the study design and the grade or recommendation of A, B or C is given based on the level of evidence. Grading will not be put forward if there is insufficient or inconclusive evidence.

Table 1: Levels of Evidence

Level	Type of Evidence
1a	Evidence from meta-analysis of randomized controlled trials
1b	Evidence from at least one randomized controlled trial
IIa	Evidence from at least on well-designed controlled study without randomization
IIb	Evidence from at least one other type of quasi-experimental/cohort study
III	Evidence from well-designed non-experimental descriptive studies such as comparative studies, correlation studies and case control studies
IV	Evidence from expert committee reports or opinions and/or clinical experiences of respected authorities

Source: U.S. Preventive Services Task Force, 2012; Canadian Task Force on Preventive Health Care, 2011

Table 2: Grades of Recommendation

Level	Type of Evidence	Action Words
Grade A Level 1a, 1b	At least one meta-analysis, systematic review, or randomized controlled trial, or evidence rated as good and directly applicable to the target population.	Recommended
Grade B Level IIa, IIb and III	Evidence from well-conducted clinical trials, directly applicable to the target population, and demonstrating overall consistency of results; or evidence extrapolated from meta-analysis, systematic review, or non-randomized controlled trial.	Encouraged
Grade C Level IV	Evidence from expert committee reports or opinions and/or clinical experiences of respected authorities; indicates absence of directly applicable clinical studies of good quality.	Should be considered

Source: Modified from Scottish Intercollegiate Guidelines Network (SIGN), 2001

Chapter 2: NUTRITION CARE PROCESS

The nutrition care process should be implemented for all individuals with hypertension whom referred to dietitian. Figure 1 shows the flow and each components of nutrition care process [15].

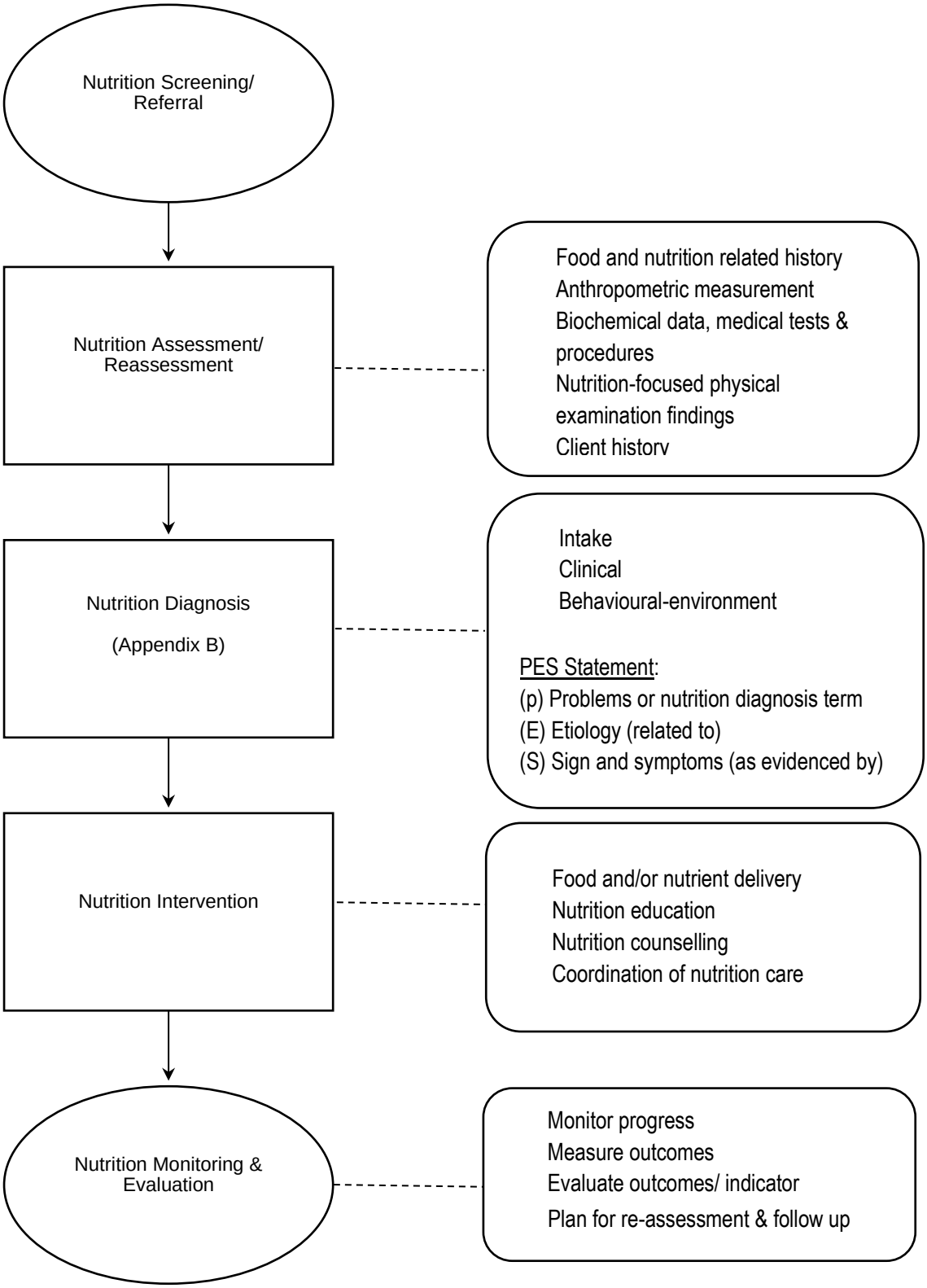


Figure 1: Nutrition Care Process.

Chapter 3: NUTRITIONAL ASSESSMENT

Nutritional assessment should be carried out for all individuals with dyslipidemia whom referred to dietitian. The following is the example of nutritional assessment for each component [9].

Component	Assessments
Food and nutrition related history	■ Food and nutrient intake: focusing on fat, fruits and vegetables intake, alcohol consumption, food checklist ■ Type of medications ■ Complementary/ alternative medicine/ supplement use ■ Knowledge/beliefs: Motivation, readiness to change ■ Food and supplies availability: grocery, dining out, online food delivery and food preparations/cooking methods ■ Behaviors: avoidance, restriction of certain foods, skipping meals ■ Social/family support ■ Physical activity: determine level of physical activity and exercise habits (duration, frequency, intensity) ■ Nutrition quality of life (patients' perception of the nutrition intervention and its impact on their lives)
Anthropometric measurement	■ Height ■ Weight ■ Body mass index (BMI) ■ Waist circumference ■ Weight history ■ Body fat percentage
Biochemical data, medical tests & procedures	■ Fasting Serum Lipid Profile (TC, LDL-C, HDL-C, TG, TC/HDL) ■ Fasting glucose level, HbA1c ■ Renal profile ■ Liver function test
Nutrition-focused physical examination findings	■ Physical examination (subcutaneous fat, muscle, corneal arcus, xanthelasma) ■ Evidence of fluid retention (i.e., edema or ascites) ■ Vital signs: blood pressure, pulse rate, heart rate (ECG)
Client history	■ Personal & social history (current stress level and history of recent crisis) ■ Medical & family history ■ Past surgical history/ treatments ■ Smoking (if any)
Comparative standards	■ Estimated needs and method for estimating (predictive formula) ■ Weight recommendations
Follow up	*Two or more follow-up visits should be planned within six to eight weeks. Recommended to arrange the follow up on the same day as the doctor for better compliance.*

Sources: PEN 2020; Carson et al. 2014 [9,16]

Chapter 4: NUTRITIONAL INTERVENTIONS

A nutrition intervention is a purposely planned action(s) designed with the intent of changing a nutrition-related behaviour, risk factor, environmental condition, or aspect of health status to resolve or improve the identified nutrition diagnosis(es) or nutrition problem(s) [17]. Nutrition interventions are selected and tailored to the client needs by planning and implementing appropriate interventions [17].

4.1 Nutrient recommendations

4.1.1 Mediterranean diet

Recommendation

- For patients with dyslipidaemia and at increased risk of developing CVD, adopting Mediterranean diet is recommended (Grade A).
- Adapt the Mediterranean diet to suit the patient's usual dietary intake.

Supporting evidence:

- A meta-analysis of randomised controlled trials (RCTs) reported that patient on Mediterranean diet had significant greater reduction in body weight (weighted mean difference (WMD) -2.24kg, 95% CI: -3.85 to -0.63) and total cholesterol (WMD -7.35mg/dL, 95% CI: -10.32 to -4.39) as compared to patients on a low-fat diet (≤30% of calories from fat), at 2 years of follow-up [18].
- The PREDIMED (Prevención con Dieta Mediterránea) Spain trial randomised persons at higher risk of CVD to Mediterranean diet supplemented with either extra-virgin olive oil (≥4 Tbsp/day) or mixed nuts (15g walnuts, 7.5g almonds & 7.5g hazelnuts per day) has demonstrated a 31% and 28% lower incidence of cardiovascular events, respectively versus control group (low-fat diet), at 5 years of follow-up [19].
- A systematic review and meta-analysis of prospective cohort studies (2,190,627 participants; follow-up period of 4 to 20 years) has shown that higher adherence to a Mediterranean diet was associated with a 8% reduction in all-cause mortality (pooled RR 0.92; 95% CI: 0.90 to 0.94) and a 10% reduction in CVD incidence or mortality (pooled RR 0.90 (95% CI: 0.87 to 0.93) [20].

4.1.2 Total fat

Recommendation

- To limit total fat intake to 30% or less of total calorie intake per day is recommended (Grade A).

Supporting evidence:

- A meta-analysis of RCTs demonstrated that adults on low-fat diet (≤30% of energy from fats) had significantly higher reduction in total cholesterol (WMD -10.1 versus −4.6 mg/dL) and LDL-cholesterol (WMD −6.0 versus −2.1 mg/dL) compared to those on low-carbohydrate diet (≤45% of energy from carbohydrates), at 6 months of follow-up [21].
- There is no direct evidence showing that limiting the percentage of total fats from appropriate energy intake reduces risk of atherosclerotic cardiovascular disease (ASCVD), however, lifestyle modifications such as weight loss and modified and/or reduced total fat intake may lower risk of ASCVD [10].

4.1.3 Saturated fatty-acids (SFA)

Recommendation

- To limit SFA intake to <10% of total calorie intake is recommended (Grade A).

Supporting evidence:

- A Cochrane systematic review of RCTs reported that lowering dietary SFA (<10%) reduced the risk of CVD events (heart disease and strokes) by 17% (risk ratio (RR) 0.83; 95% CI: 0.70 to 0.98), however had little effect on the risk of mortality [22].
- Replacing dietary SFA with PUFA (-25%), MUFA (-15%), and to a minor extent carbohydrate from whole grains (-9%) shown all associated with decreased risk of CHD [5].
- A systematic review showed that reducing SFA intake by replacing SFA with cis-PUFA (predominantly linoleic acid and α-linolenic acid) or cis-MUFA (predominantly oleic acid) were more beneficial than replacing SFA with carbohydrates [23].
- Substituting 5% of energy from SFA with equal amount of energy from PUFA and MUFA shown associated with estimated reductions in total mortality of 27% (Hazard ratio (HR): 0.73; 95%CI: 0.70-0.77) and 13% (HR: 0.87; 95%CI: 0.82-0.93), respectively [24].

4.1.4 Polyunsaturated fatty acid (PUFA)

Recommendation

- Replacing SFA with PUFA should be considered (Grade B).

Supporting evidence:

- A systematic review reported that each 1% of SFA replaced with an equivalent amount of cis-PUFA had significant decrease in total cholesterol of 0.064 mmol/L (95% CI: –0.070 to –0.058), in LDL cholesterol of 0.055 mmol/L (95% CI: –0.061 to –0.050) and in triglyceride of 0.010 mmol/L (95% CI: –0.014 to –0.007) [23].
- A RCT study showed that the LDL cholesterol lowering effect of replacing SFA with PUFA may be lesser in obese (5.3%) than in normal-weight individuals (9.7%) [25].
- In a meta-analysis of cohort studies (310,602 participants; follow-up period of 5.3 to 30 years) demonstrated that high intake (median intake 7.7%) of dietary linoleic acid (LA) (n-6 PUFA) as compared to low intake (median intake 1.1%) of energy from LA was associated with 15% and 21% of reduction in risk of CHD (pooled RR: 0.85; 95% CI: 0.78–0.92) and CHD mortality (RR: 0.79; 95% CI: 0.71–0.89), respectively [26].

4.1.5 Monounsaturated fatty acid (MUFA)

Recommendation

- Replacing SFA with MUFA should be considered (Grade B).

Supporting evidence:

- A systematic review reported that each 1% of SFA replaced with an equivalent amount of cis-MUFA, there was a significant decrease in total cholesterol of 0.046 mmol/L (95% CI: –0.051 to –0.040), in LDL cholesterol of 0.042 mmol/L (95% CI: –0.047 to –0.037), and in triglyceride of 0.004 mmol/L (95% CI: –0.007 to –0.001; P = 0.022) [23].
- The effect of replacing SFA with MUFA on CVD events was unclear and increasing MUFA intake may improve serum lipid levels, however this effect may disappear with excessive intake of MUFA [10].
- A meta-analysis (108 systematic reviews & 20 RCTs) demonstrated that there was high GRADE evidence for a moderate (0.20–0.40 mmol/L) to large (>0.40 mmol/L) reduction in LDL cholesterol, when SFA was substitute by ~17% of total energy from rapeseed/canola oil (high in n-6 PUFA and/or MUFA) [27].
- Meta-analysis (10 studies) reported that avocado (½ to 1½ whole) consumption as source of MUFA significantly reduced TC by -0.48mmol/L (95% CI: -0.64 to -0.34), LDL cholesterol by -0.43mmol/L (95% CI: -0.59 to -0.26), and TG by -0.31 mmol/L (95% CI: -0.50 to -0.11) [28].

4.1.6 Trans fatty acid

Recommendation

- To limit trans fatty acid intake to <1% of total calorie intake is recommended (Grade A).

Supporting evidence:

- Industrially produced trans fatty acids increase LDL cholesterol and lipoprotein (a) [Lp(a)] which is a lipoprotein that stimulates atherosclerosis and reduces HDL cholesterol as compared to other fatty acids, whereas its effect on TG is inconclusive. Decreasing the intake of trans fatty acid is effective in preventing ASCVD [10].
- A systematic review (17 studies) revealed that each 1% substitution of trans fatty acid with an equivalent amount of cis-MUFA, there was a significant reduction in total cholesterol of 0.027 mmol/L (95% CI: –0.036 to –0.018), in LDL cholesterol of 0.034 mmol/L (95% CI: –0.042 to –0.027), in TG of 0.013 mmol/L (95% CI: –0.022 to –0.003) and significant increase in HDL cholesterol of 0.010 mmol/L (95% CI: 0.006 to 0.015) [29].
- The systematic review too reported that each 1% substitution of trans fatty acid with an equivalent amount of cis-PUFA, there was a significant reduction in total cholesterol of 0.045 mmol/L (95% CI: –0.053 to –0.037), in LDL cholesterol of –0.048 mmol/L (95% CI: –0.055 to –0.041), in TG of 0.017 mmol/L (95% CI: –0.026 to –0.009) and significant increase in HDL cholesterol of 0.008 mg/dL (95% CI: 0.005 to 0.012) [29].

4.1.7 Dietary cholesterol

Recommendation

- Reducing dietary cholesterol intake to <200mg per day should be considered (Grade B).

Supporting evidence:

- Most of the high cholesterol foods also contain high levels of SFA. When guidelines to lower saturated fat intake are followed, reductions in dietary cholesterol intake follow [5].
- Dietary cholesterol is not necessary for our body needs. The relationship between dietary cholesterol and blood cholesterol level is still questionable [30].

4.1.8 Carbohydrates

Recommendation

- Reducing carbohydrate intake to 50 – 60% of total calories should be considered (Grade B) with emphasis on whole grains.

Supporting evidence:

- Reduction in intake of dietary carbohydrate, monosaccharides, and disaccharides can decrease TG. Decreasing intake of dietary trans fatty acids and carbohydrates shown associated with increase in HDL cholesterol [13].
- High dietary intake of carbohydrates (>60% of total calories) will increase TG, whereas a diet that substitutes SFA with unsaturated fatty acids does not increase TG level [31].
- A meta-analysis (38 studies; 6499 participants; follow-up period of 6–12 months) demonstrated that low-carbohydrate diet (≤40% of total calories) was associated with reduction in weight (mean difference -1.30kg; 95% CI: -2.02 to -0.57) and TG (-0.10 mmol/L; 95%CI:- 0.16 to -0.04) and increase in HDL cholesterol (0.05 mmol/L; 95% CI 0.03 to 0.08) as compared to low-fat diet (≤30% of total calories) [32].

4.1.9 Dietary fiber

Recommendation

- Dietary fiber intake of 25 – 30g per day is recommended (Grade A).

Supporting evidence:

- A systematic review of 9 prospective cohort studies (221 080 individuals) reported that the risk of CHD decreased by 4% (RR 0.96, 95% CI: 0.93–0.99) for each additional portion of vegetables and fruit intake per day [33].
- A systematic review (12 cohort studies; 278 459 individuals, median follow up 11 years) demonstrates that increased consumption of fruit and vegetables from <3 servings/day to >5 servings/day was related to a 17% reduction in CHD risk, whereas increased intake from <3 servings/day to 3–5 servings/day is associated with a smaller and borderline significant reduction in CHD risk [34].
- Soluble dietary fiber is effective in improving blood cholesterol and TG levels [14].
- Diets high in total fiber and soluble fiber, as part of a cardioprotective eating pattern, can reduce total cholesterol by 2% to 3% and LDL by up to 7% [35].
- Dietary patterns that are high in viscous, soluble fiber (7-27 g/day from oats, barley, psyllium, pectin or konjac) can reduce LDL levels by about 3-10 % [9].

- A systematic review (28 RCTs; 1924 individuals) showed consumption of psyllium powder (～10.2g/ day) significantly reduced LDL cholesterol (MD: -0.33 mmol/L; 95% CI: -0.38 to -0.27 mmol/L; p< 0.001), non-HDL cholesterol (MD: -0.39mmol/L; 95% CI: -0.50 to -0.27 mmol/L; p< 0.001), and apoB (MD: -0.05 g/L; 95% CI: -0.08 to -0.03 g/L; p< 0.001) [36].

4.1.10 Fish

Recommendation

- Fish (especially fatty fish) consumption of 2 – 3 servings per week is encouraged (Grade B)

Supporting evidence:

- In people without existing CHD, a meta-analysis (17 studies) found that compared with the lowest fish intake (<1 serving/month or 1–3 servings/month), either low (1 serving/week) or moderate (2–4 servings/week) consumption of fish was associated with a significant reduction in CHD mortality (0.84; 95%CI: 0.75–0.95 and 0.79; 95%CI: 0.67–0.92, respectively). An intake of 2–3 servings of fish (including oily fish) per week (250–500mg of combined DHA and EPA) was associated with reduced CHD risk [37].
- Marine-derived omega-3 fatty acids, preferably from fish of at least two servings (150g each) per week can reduce risk of CVD [35].
- Eating fish, particularly fish rich in n-3 PUFA, at least once a week, was associated with a 16% lower risk of CAD, and eating fish two to four times a week was associated with a 6% lower risk of stroke. The highest risk was observed in the range of no or very low intakes of fish [5].

4.1.11 Other foods

Recommendation

- There is limited evidence on intake of cocoa, dark chocolate and garlic, etc for reduction of blood lipids.

Supporting evidence:

- The influence of dark chocolate on lipid levels is mainly attributable to flavonoids, which could inhibit cholesterol absorption. Meta-analysis (10 RCT, 320 participants) showed that intake of dark chocolate for 2 – 12 weeks significantly reduced total cholesterol and LDL cholesterol (-0.16 and -0.15 mmol/L), respectively [13].

- A meta-analysis (108 systematic reviews & 20 RCTs) reported that the evidence was considered very low GRADE for a small (<0.20 mmol/L) reduction in LDL cholesterol by dark chocolate or cocoa products [27].

4.2 Functional foods

4.2.1 Soy protein

Recommendation

- Consumption of soy protein (≥25g) is encouraged (Grade B).

Supporting evidence:

- A systematic review of 30 RCTs (2,913 adults with normal or mildly elevated cholesterolaemia) reported that the inclusion of modest amounts of soya protein (~25g) into the diet of adults with normal-mild raised lipids resulted in small, but significant reductions in LDL cholesterol, TC and TG [33].
- Dietary patterns high in soy protein (≥30 g/day) can reduce LDL cholesterol levels by about 3-5% in adults with elevated lipid levels [9].
- A meta-analysis of RCTs (43 studies, average 9.2 weeks follow up) demonstrated that soy protein intake (~30g/day) resulted in weighted mean reductions of LDL cholesterol of -0.23 mmol/l or 5.5% lower and TG of -0.17 mmol/l or 10.7% lower and increase in HDL cholesterol of 0.044 mmol/l or 3.2% higher, as compared to the controls (non-soy protein) [38].
- A meta-analysis (108 systematic reviews & 20 RCTs) revealed that there was high GRADE evidence for a small (<0.20 mmol/L) to moderate (0.20–0.40 mmol/l) reduction in LDL cholesterol by soy protein (~ 26g per day) compared with controls [27].

4.2.2 Plant sterol/ stanol

Recommendation

- Consumption of 2–3g of plant sterol/stanol per day from enriched foods is recommended (Grade A).

Supporting evidence:

- Systematic reviews studying the efficacy of phytosterols (or plant sterol) have shown that phytosterol/stanol enriched foods can significantly lower LDL cholesterol in normocholesterolaemic, hypercholesterolaemic and diabetic individuals. A daily intake of phytosterols (~ 2.5g) from enriched

breakfast cereal, reduced fat yoghurt, reduced fat milk or bread reduces LDL cholesterol by approximately 5 to 15% [39].

- Functional foods containing phytosterols (plant sterols and stanols) are effective in lowering LDL-C levels by an average of 10% when consumed in amounts of 2 g/day [5].
- Daily intake of foods containing 2 to 3 g of plant sterol and stanol ester-enriched foods can reduce total cholesterol by 4 to 11% and LDL cholesterol by 7 to 15%. Plant sterol or stanol intake of more than 3g per day does not provide additional benefit [35].
- A meta-analysis (108 systematic reviews & 20 RCTs) revealed that there was high GRADE evidence for a moderate (0.20–0.40 mmol/l) reduction in LDL cholesterol by foods enriched with plant sterols and stanols (~ 2.2g per day) compared with diets without foods enriched with plant sterols/ stanols [27].

4.2.3 Beta glucan

Recommendation

- Consumption of β-glucan (≥3g) is encouraged (Grade B).

Supporting evidence:

- A meta-analysis of RCTs (28 studies, 2-12 weeks of follow up) demonstrated that adding ≥3 g oat β-glucan to the diet reduces LDL and total cholesterol by 0.25 mmol/L (95% CI: 0.20–0.30) and 0.30 mmol/L (95% CI: 0.24–0.35), respectively, without changing HDL cholesterol or TG [40].
- A meta-analysis of RCTs (14 studies, 615 healthy & hypercholesterolemic participants, ~ 4 weeks of follow up) showed that intake of barley β-glucan of 6.5 and 6.9 g/day significantly reduces LDL cholesterol (−0.25 mmol/l; 95% CI: −0.30 to −0.20) and non-HDL cholesterol (−0.31 mmol/l; 95% CI: −0.39 to −0.23), respectively, compared with controls [41].
- A meta-analysis (108 systematic reviews & 20 RCTs) revealed that there was high GRADE evidence for a moderate (0.20–0.40 mmol/l) reductions in LDL cholesterol by soluble fiber β-glucans from barley (~5.6g per day) or oats (~4.8g per day), compared with controls [27].

4.2.4 Nuts

Recommendation

- Consumption of 30g of nuts (mixed) per day is encouraged (Grade B).

Supporting evidence:

- Dietary patterns with tree nuts (28-100 g/day) can reduce LDL levels by about 2.5–7 % [9].

- Daily consumption of 30g of (mixed) nuts was associated with a ~30% lower risk of ASCVD [5].
- A meta-analysis (108 systematic reviews & 20 RCTs) demonstrated that there was high GRADE evidence for a small but highly significant reduction in the LDL cholesterol (mean: -0.12 mmol/L; 95% CI: -0.14 to -0.11) by tree nuts. High GRADE evidence was demonstrated for almonds (~60g per day) and moderate GRADE evidence for hazelnuts (~39g per day) and walnuts (~46g per day), compared with controls [27].

4.2.5 Flaxseeds

Recommendation

- Flaxseed (whole) consumption should be considered (Grade C).

Supporting evidence:

- Meta-analyses of clinical trials support the observation that flaxseed consumption (20 to 50 g/day or 2 to 5 Tbsp/day of whole or ground flaxseed) can modestly reduce total and LDL cholesterol levels, particularly in adults with elevated cholesterol levels, but has no effect on HDL cholesterol or triglyceride levels [9].
- A meta-analysis (108 systematic reviews & 20 RCTs) revealed that there was high GRADE evidence for a small (<0.20 mmol/L) to moderate (0.20–0.40 mmol/l) reduction in LDL cholesterol by flaxseeds (whole) (38g per day) compared with controls [27].

4.2.6 Chia seeds

Recommendation

- Chia seeds consumption should be considered (Grade C).

Supporting evidence:

- A meta-analysis of RCTs (10 studies, 7-24 weeks of follow up) demonstrated that chia seeds (15 – 50g per day) consumption reduces total cholesterol (MD: 0.07 mmol/L, 95% CI: -0.25–0.10), LDL cholesterol (MD: 0.05 mmol/L, 95% CI: -0.13–0.23), and TG (MD: 0.15 mmol/L, 95% CI: -0.37–0.05) and increases HDL cholesterol (MD: 0.07 mmol/L, 95% CI: -0.15–0.001) [42].
- Chia seeds seem to be beneficial in increasing serum fatty acid and omega-3 levels, alpha-linolenic acid (ALA) in all forms of chia seeds however, heat treatment can compromise its chemical composition, reducing its concentration of PUFAs [42].

4.3 Lifestyle modifications

4.3.1 Weight reduction

Recommendation

- Weight loss is recommended to improve blood lipid levels in adults who are overweight or obese (Grade A).
- Encourage to achieve and/ or maintain healthy BMI (<25kgm^2) and waist circumference (<90cm for men and <80cm for women) (Grade B).
- Customize the recommendation to patient's needs and set realistic goals.

Supporting evidence:

- A systemic review (7 studies, 2000 participants; 12 months follow up) reported that weight loss interventions was associated with significant reductions in body weight (-3.0 kg; 95% CI: -5.1 to -0.9) and total cholesterol (-0.36mmol/L; 95% CI: -0.75 to 0.04) and favourable (although not statistically significant) changes in LDL cholesterol, HDL cholesterol and TG, compared with controls [43].
- Although there is no direct evidence showing that limiting total energy intake and maintaining an appropriate body weight suppresses ASCVD, improvement in serum lipid levels by lifestyle modification including body weight reduction in response to diet therapy may prevent ASCVD [10].
- For high and borderline-high TG levels (2.26-5.65 mmol/L or 1.70-2.25 mmol/L, respectively), TG levels can be decreased by ~20% with 5-10% weight loss in individuals who are overweight or obese. Weight loss (5-10%) in individuals who are overweight/ obese can increase HDL cholesterol by 3% [9].
- Weight loss of 5–10% in patients who are obese, can improve their cholesterol and TG levels although a normal body weight was failed to achieve [14].
- Weight reduction improves insulin sensitivity and decreases TG levels. A drop in TG of 0.15mmol/L is observed for every 10kg of weight loss. Stabilised weight reduction has a beneficial influence on HDL cholesterol levels, and a 0.09mmol/L increase for every 10 kg body weight reduction was found [13].

4.3.2 Exercise

Recommendation

- An increase of physical activity with moderate intensity aerobic exercise for at least 30 minutes, 5 times a week or accumulative of 150 minutes per week is recommended (Grade A).

Supporting evidence:

- A meta-analysis (22 observational studies, 977925 participants) demonstrated a dose response-relationship for exercise duration: 2.5 hours/week (equivalent to 30 minutes daily of moderate intensity activity on 5 days a week) compared with no activity, was associated with a reduction in mortality risk of 19% (95% CI 15–24), while 7 hours/week of moderate activity compared with no activity reduced the mortality risk by 24% (95% CI 19–29). Any form of exercise appears to be effective, with a positive influence on CVD risk factors: lowering LDL cholesterol and TG, increasing HDL cholesterol [44].
- Physical activity of at least 150 minutes per week of accumulated moderate-intensity or 75 minutes per week of vigorous-intensity aerobic physical activity can be beneficial to reduce ASCVD risk [4].
- Moderate to vigorous exercise (30-60 min/day) can increase HDL cholesterol by 5-10% [9].
- There is an inverse relationship between moderate-to-vigorous physical activity and all-cause mortality, cardiovascular mortality and morbidity [5].

4.3.3 Smoking cessation

Recommendation

- Smoking cessation (in any form) and passive smoking avoidance are recommended (Grade A).

Supporting evidence:

- Smoking is a risk factor for CHD, stroke, abdominal aortic aneurysm and peripheral artery disease (PAD). Passive smoking is a risk factor for CHD and stroke [10].
- Smoking cessation is associated with reduced coronary artery disease mortality and an increase in HDL cholesterol levels by 7-12% [9].
- Smoking, hypertension, low HDL cholesterol, and a family history of CVD are associated with increased incidence of CVD [14].

4.3.4 Alcohol

Recommendation

- Individuals who drink should limit alcohol intake to 2 standard drinks per day for men and 1 standard drink per day for women is recommended (Grade A).

Supporting evidence:

- Reduction of alcohol consumption along with a diet low in refined carbohydrates (added sugar) and increasing physical activity can reduce TG levels by 10-20% [9].

- Excessive alcohol intake is associated with hypertriglyceridemia [14].
- TG level increased by 0.19 mg/dL per gram of alcohol consumed in a day and 5.69 mg/dL per 30g consumed in a day [13].
- Higher (>100g pure alcohol) alcohol consumption is linearly associated with a higher risk of all stroke subtypes, coronary disease, heart failure, and several less common CVD subtypes [5].

4.3.5 Caffeine

Recommendation

- There is limited evidence on caffeine intake (coffee or tea) and reduction of blood lipids.

Supporting evidence:

- Consumption of ≥9 drinks per day of non-filtered coffee (contains LDL cholesterol raising components called cafestol and kahweol) may be associated with 25% increased risk of ASCVD mortality. Non-filtered coffee includes boiled, Greek, and Turkish coffee and some espresso coffees. Moderate coffee consumption (3-4 cups per day) is probably not harmful, perhaps even moderately beneficial [5].
- A meta-analysis (108 systematic reviews & 20 RCTs) reported that unfiltered coffee (~6.1 cups/ day) was associated with a moderate (0.20–0.40 mmol/L) to large (>0.40 mmol/L) increase in LDL cholesterol as compared to filtered coffee (high GRADE evidence). Coffee (mocha, espresso, instant, or filtered coffee) intake (6.8 cups/ day) tended to increase LDL cholesterol in small (<0.20 mmol/L) to moderate (0.20–0.40 mmol/L) amount (moderate GRADE evidence). Filtered coffee (4.6 cups/ day) did not increase the LDL cholesterol level compared with water or no coffee intake (high GRADE evidence). No clear effects on LDL cholesterol were observed when comparing regular coffee with decaffeinated coffee (high GRADE evidence) [27].
- A meta-analysis (108 systematic reviews & 20 RCTs) reported that there is small (<0.20 mmol/L) reduction in LDL cholesterol level by green tea (~263mg of catechins/ 2–5 cups per day) (moderate GRADE evidence) and black tea (very low GRADE evidence) [27].
- A meta-analysis (12 RCT) reported that coffee consumption (>3 cups, 125ml per cup) may be associated with an increased risk for dyslipidemia and CVD. Coffee consumption (>3 cups/day) in 6 weeks of intervention studies showed significant increase in TC (WMD: 0.21 mmol/L; 95% CI: 0.04 - 0.39; p=0.017), TG (WMD: 0.12 mmol/L; 95% CI: 0.03 - 0.20, p=0.006) and LDL cholesterol (WMD: 0.14 mmol/L; 95% CI: 0.05 - 0.24, p=0.003), however there was no significant effect on HDL cholesterol (WMD: -0.01 mmol/L; 95% CI: -0.06 to 0.04; p=0.707) [45].

4.4 Supplementation

4.4.1 Fish oil

Recommendation

- Fish oil supplementation (2-4 g per day) for patient with hypertriglyceridemia is encouraged (Grade B).

Supporting evidence:

- A systematic review (1,199,246 participants; followed up for >1 year) reported increased consumption of n-3 fatty acids from fish or fish-oil supplements, but not of alpha linolenic acid (ALA), reduced all-cause mortality, cardiac and sudden death, and possibly stroke. The evidence for the benefits of fish oil is stronger in secondary than in primary-prevention settings [33].
- Omega-3 fatty acid supplements (2-4 g/day) can decrease TG levels by 20-45% in individuals with high and borderline-high TG levels [9].
- High-doses of supplemental EPA and DHA (> 200mg/dL) have shown to lower TG in patients with elevated TG. In patients with CHD (but no angina or implantable cardioverter defibrillators (ICD)) supplementation with 850mg per day EPA and/or DHA can reduce sudden death by 45%. For patients with CHD with angina or ICDs, EPA and DHA supplements may be contraindicated. Consumption of >3g of omega-3 fatty acids per day may cause gastrointestinal symptoms [35].
- A RCT in Taiwanese patients with hypertriglyceridemia (TG levels 2.2–11.2 mmol/L) received a concentrated preparation of omega-3 EPA plus DHA for 8 weeks demonstarted that the TG levels were reduced 32.1% (EPA plus DHA dosage of 4 g/d), and 29.7% (EPA plus DHA dosage of 2 g/d) in omega-3 fatty acid group compared with 5.4% in placebo group. However, the antithrombotic effects may increase the risk of bleeding, especially when given in addition to combination therapy with aspirin and other antiplatelet drugs [13].
- Fish oils from supplements do not offer any clear advantages for CVD event risk reduction. However, there are consistent triglyceride-lowering effects at high doses (2-4 g/d) of omega-3 PUFA [6].
- Several meta-analyses and a recent Cochrane review showed no benefits of fish oils on CVD outcomes and/or mortality, although a 7% lower risk of CHD events was observed [5].

4.4.2 Krill oil

Recommendation

- There is limited evidence on krill oil supplementation and reduction of blood lipids.

Supporting evidence:

- A meta-analysis (7 trials; 662 participants) showed that supplementation with krill oil had a significant reduction in plasma LDL cholesterol (WMD: −0.40 mmol/L; 95%CI: −0.73 to −0.06) and TG (WMD: −0.15 mmol/L; 95%CI: −0.24 to −0.07) and increase in HDL cholesterol (WMD: 0.17 mmol/L; 95%CI: 0.05 to 0.28), while reduction in total cholesterol did not reach statistical significance [46].
- A meta-analysis (64 RCT) demonstrated that 1g of n-3 fatty acids contained in krill oil and fish oil lowered TG by 0.11 mmol/L (95%CI: 0.00–0.21) and 0.10 mmol/L (95%CI: 0.02–0.15), respectively. However, the net differences in TG, LDL cholesterol, HDL cholesterol and total cholesterol were not significantly different between the krill oil and fish oil groups [47].

4.4.3 Red yeast rice

Recommendation

- There is limited evidence on red yeast rice supplementation and reduction of blood lipids.

Supporting evidence:

- In a meta-analysis (red yeast rice from 1200 mg/d to 4800 mg/d, containing 4.8-24 mg monacolin K) reported that red yeast rice could reduce LDL cholesterol from 0.49 – 1.57 mmol/L (pooled estimate of 39 mg/dL), compared to placebo. Red yeast rice or monacolin K (10 mg/ day) intake can be as effective as statins in lowering LDL cholesterol. However, the safety outcomes of red yeast rice have not been extensively studied [13].
- Red yeast rice products (1.2 – 3.6 g/day for up to 6 months) can be as effective as statins medication at lowering blood cholesterol in adults with hyperlipidaemia. However, red yeast rice products content may lead to serious adverse outcomes including liver, kidney and muscle problems [9].

4.4.4 B-vitamins

Recommendation

- There is limited evidence on B-vitamins supplementation and reduction of blood lipids.

Supporting evidence:

- Prescription doses of niacin (1 to 3 g/day of crystalline niacin or 0.5 to 2 g/day of extended-release niacin) can be effective at increasing HDL and decreasing TG. On the other hand, the effects on LDL cholesterol are not significant. Hence, niacin is most effective for the management of mixed

hyperlipidaemia. However, use of niacin as an adjunct to statin therapy may increase risk of death from any cause [9].

- A meta-analysis (30 RCT; 4749 participants; in mixed primary and secondary populations) reported that niacin (1 to 3 g/day of extended-release or immediate-release niacin) significantly reduced total cholesterol (10%), LDL cholesterol (12%) and TG (20%) and increased HDL cholesterol (16%). However, there was adverse effect of flushes with supplementation of niacin [48].
- For hypertriglyceridemia, niacin can be considered as the first-line medication, whereas it is effective for patients with low HDL cholesterol. However, niacin is contraindicated for patients with chronic liver disease, severe gout, diabetes, hyperuricemia, and peptic ulcer [14].
- B-vitamins supplementation (folic acid, vitamin B6 and B12) have a small beneficial effect on reducing the risk of stroke, but have no effect on other CVD events. In particular, folic acid supplements (1 mg/day) may be beneficial to reduce the risk of total CVD and stroke in individuals with and without a history of CVD [9].

4.4.5 Coenzyme Q10

Recommendation

- There is limited evidence on coenzyme Q10 supplementation and reduction of blood lipids.

Supporting evidence:

- There is insufficient evidence for the association of coenzyme Q10 and CHD. The clinical significance of normalising coenzyme Q10 levels in patients treated with statin medications is inconclusive [35].
- Clinical trials provide conflicting data regarding a benefit of coenzyme Q10 for preventing or treating CVD or CVD risk factors [9].
- Various coenzyme Q10 formulations, difference range of doses and duration of supplementation used in the clinical studies, makes it inconclusive for recommendation on benefit of coenzyme Q10 for therapeutic use with patients with CVD [49].

4.4.6 Coconut oil

Recommendation

- There is limited evidence on coconut oil supplementation and reduction of blood lipids.

Supporting evidence:

- Coconut oil may not be a suitable alternative to non-hydrogenated vegetables oils as it has been demonstrated to increase LDL levels, despite also modestly increasing HDL levels [6].
- A systematic review (7 studies) demonstrated that coconut oil significantly increases serum total cholesterol, LDL and HDL cholesterol levels as compared to PUFA and MUFA oils. Therefore, PUFA and MUFA oils should be encouraged over coconut oil as they lower LDL cholesterol and increase HDL cholesterol significantly [50].

4.4.7 Flaxseed oil

Recommendation

- There is limited evidence on flaxseed oil supplementation and reduction of blood lipids.

Supporting evidence:

- Flaxseed oil does not appear to affect blood lipid levels [6].
- A meta-analysis (108 systematic reviews & 20 RCTs) reported that there is no clear effect of flaxseed oil on LDL cholesterol (very low GRADE evidence) [27].

4.4.8 Olive oil

Recommendation

- There is limited evidence on olive oil supplementation and reduction of blood lipids.

Supporting evidence:

- A meta-analysis (27 RCTs; 1089 participants) reported that olive oil (virgin) was less potent (significantly lesser) than other vegetable oils (PUFA rich oils) in lowering total cholesterol, LDL cholesterol and TG. Olive oil consumption increased slightly more HDL cholesterol (0.03 mmol/L; 95% CI: 0.01–0.06) than other plant oils in more than 30 days intervention studies. There were no significant effects on Apo lipoprotein A1 and Apo lipoprotein B [51].
- The PREDIMED (Prevención con Dieta Mediterránea) Spain RCT study demonstrated that persons at higher risk of CVD provided with Mediterranean diet plus extra-virgin olive oil (≥4 Tbsp/day) had a 31% lower incidence of cardiovascular events, versus control group (low-fat diet), at 5 years of follow-up [19].

4.4.9 Spirulina

Recommendation

- There is limited evidence on spirulina supplementation and reduction of blood lipids.

Supporting evidence:

- A meta-analysis (12 clinical studies) with intervention durations of 2 to 48 weeks reported that spirulina supplementation (1g to 19g per day) significantly lowered TC (−0.95mmol/L; p=0.0001), LDL cholesterol (−0.86mmol/L; p=0.0002), TG (−0.44mmol/L; p=0.0001), very-low-density lipoprotein cholesterol (−0.21mmol/L; p=0.0001) [52].
- A study randomized, placebo-controlled, double-blind crossover study in non-hypercholesteraemic men and women showed daily consumption of spirulina (4.8g) or wakame (4.8g) for 17 days did not affect serum lipids as compared with the placebo group [53].

Chapter 5: MONITORING AND EVALUATION

To reassess signs and symptoms from PES statements to determine if goal/prescription and interventions have been implemented. The suggested length of contact time is 30 - 45 minutes for first visit and 15 - 30 minutes for subsequent visit (s) [54].

Component	Assessments
At each visit, reassess	▪ Weight ▪ Dietary intake pertinent to nutrition diagnosis ▪ Comprehension of nutrition prescription ▪ Adherence to nutrition prescription ▪ Physical activity level ▪ Changes in medications and dietary supplements ▪ Stages of change ▪ Progress toward achievement of specified goals and any related barriers
As feasible, monitor monthly or quarterly	▪ Waist circumference ▪ Body fat percentage ▪ Newly reported lab values

Sources: Carson et al. 2014 [16]

Appendix A: CLASSIFICATION OF DYSLIPIDEMIA

There are two types of dyslipidaemia namely primary (genetic) dyslipidaemia and secondary dyslipidaemia.

Table 3: Types of primary (genetic) dyslipidemia

	Risk of CHD	Risk of Pancreatitis	Plasma Cholesterol	Plasma Triglyceride	Physical signs (if present)
Common ("polygenic") Hypercholesterolemia	↑	↔	↑↑	N	Corneal Arcus, Xanthelasma
Familial Combined Hyperlipidaemia	↑↑	↔	↑ or ↔	↑ or ↔	Corneal Arcus, Xanthelasma
Familial Hypercholesterolemia	↑↑↑	↔	↑↑↑	↑	Tendon xanthomata, (finger extensor, Achilles' tendons), Corneal Arcus, Xanthelasma, Aortic stenosis
Remnant Hypercholesterolemia	↑↑↑	↔	↑↑↑	↑↑	Tuberous xanthomata, (elbows), striae xanthomata, (palm creases) tendon xanthomata
Chylomicronemia Syndrome	↔ or ↑	↑↑↑	↑	↑↑↑	Eruptive xanthomata, (buttocks, elbows) retinal lipemia, hepatosplenomegaly
Familial Hypertriglyceridemia	↑	↑↑	↑	↑↑	Eruptive xanthomata, (buttocks, elbows) retinal lipemia, hepatosplenomegaly
High HDL-C	↓↓	↔	↑	↔	-
Low HDL-C	↑↑	↔	↔	↔ or ↑	-

LDL = low-density lipoprotein; HDL = high-density lipoprotein; ↑ = mildly increased; ↑↑ = moderately increased; ↑↑↑ = severely increased; ↔ = no effect; ↓↓ = moderately decreased; N = normal.

Source: Malaysian CPG 2018 [11]

Secondary dyslipidemia is caused by other factors such as nephrotic syndrome, cholestatic liver disease, hypothyroidism, Cushing's syndrome, drugs, alcoholism and insulin resistance states such as type 2 Diabetes Mellitus (T2DM) and metabolic syndrome.

Table 4: Causes of secondary dyslipidaemia

Causes	Cholesterol	Triglycerides	HDL-cholesterol
Lifestyle factors			
Alcohol	↔	↑ ↑	↑
Saturated fat/ trans-fat	↑	↑	↓
Cardio metabolic risk	↔	↑ ↑	↓
Smoking	↔	↔	↓
Physical inactivity	↔	↑	↓
Metabolic / Endocrine			
Hypothyroidism	↑ ↑	↔	↔
Type 2 Diabetes (T2DM)	↑	↑ ↑	↓
Cushing's Syndrome	↑	↑ ↑	↓
Renal			
End stage renal disease	↔ or ↓	↑	↓
Nephrotic syndrome	↑ ↑	↑ ↑	↓
Hepatic			
Obstructive liver disease	↑ ↑	↔	↓
Primary biliary cirrhosis	↑ ↑	↔	↑
Drugs*			
Thiazide diuretics	↔	↑ ↑	↓
Beta blockers[29]	↔	↑	↓
Aanabolic steroids[30]	↔	↑	↓ ↓
Glucocorticoids[31]	↔	↔	↓ for <60 years ↑ for ≥60 years

LDL = low-density lipoprotein; HDL = high-density lipoprotein; ↑ = mildly increased; ↑↑ = moderately increased; ↔ = no effect; ↓ = mildly decreased; ↓↓ = moderately decreased

Source: Malaysian CPG 2018 [11]

Appendix B: NUTRITION DIAGNOSIS-PES STATEMENT

Table 5: Sample nutrition diagnostic statements for patients with dyslipidemia:

PROBLEM	ETIOLOGY	SIGN AND SYMPTOMS
	INTAKE	
Excessive energy intake	▪ Food and nutrition related knowledge deficit concerning energy intake ▪ Unsupported beliefs/attitudes about food, nutrition and nutrition-related topics ▪ Lack of or limited access to healthful food choices ▪ Lack of motivation for behaviour change ▪ Failure to adjust for lifestyle changes (e.g., decrease in physical activity) or decreased metabolism (e.g., aging) ▪ Failure to adjust for restricted mobility due to recovery from injury or surgical procedure	▪ Self-reported estimated energy intake in excess of energy needs ▪ Intake of high caloric density or large portions of food/ beverages ▪ BMI > 25kg/m^2 or weight gain
Excessive fat intake	▪ Food- and nutrition-related knowledge deficit concerning appropriate amount of dietary fat ▪ Unsupported beliefs/attitudes about food, nutrition, and nutrition-related topics ▪ Lack of or limited access to healthful food choices ▪ Lack of motivation for behavior change ▪ Changes in taste and appetite or preference ▪	▪ Self-reported estimated fat intake in excess of fat needs ▪ Frequent intake of high-fat foods ▪ Frequent food preparation with added fat ▪ High TC, LDL values ▪ Abnormal LFT readings
Intake of types of fats inconsistent with needs (SFA, trans fat)	▪ Food- and nutrition-related knowledge deficit concerning appropriate amount of dietary fat (SFA/ trans fat). ▪ Unsupported beliefs/attitudes about food, nutrition, and nutrition-related topics.	▪ Self-reported estimated fat (SFA/ trans fat) intake in excess of the needs ▪ Frequent intake of high-SFA/ trans fat foods ▪ Frequent consumption of fats that are undesirable for condition (eg, SFA, trans fat)

	▪ Lack of or limited access to healthful food choices ▪ Lack of motivation for behavior change ▪ Changes in taste and appetite or preference	▪ Estimated intake of MUFA, PUFA less than the recommendation ▪ High TC & LDL values, low HDL ▪ Abnormal LFT readings ▪ High CRP
Inadequate fiber intake	▪ Food and nutrition related knowledge deficit concerning desirable quantities of fiber ▪ Lack of or limited access to fiber-containing foods/fluids ▪ Difficulty chewing or swallowing high-fiber foods ▪ Economic constraints that limit availability of higher fiber foods ▪ Lack of motivation for behavior change/ denial to increase fiber intake	▪ Estimated intake of fiber is insufficient as compared to recommendations by American Heart Association (38 g/day for men and 25 g/day for women). ▪ Self-reported estimated fiber intake (servings) fewer than the recommended fiber servings.
Inadequate bioactive substance (e.g. plant sterol/stanol, beta glucan, soy protein or psyllium) intake	▪ Food and nutrition-related knowledge deficit concerning recommended bioactive substance intake ▪ Lack of or limited access to food that contains a bioactive substance ▪ Alteration in gastrointestinal tract structure and/or function	▪ Estimated intake of bioactive substance is insufficient as compared to the recommendation. ▪ Self-reported estimated bioactive substance (frequency) intake fewer than the recommendation. ▪ Abnormal lipid levels (TC, LDL, HDL, TG).
Excessive alcohol intake	▪ Unsupported beliefs/attitudes about food, nutrition and nutrition-related topics ▪ Food and nutrition related knowledge deficit concerning appropriate alcohol intake ▪ Alcohol addiction ▪ Lack of motivation in reducing alcohol intake	▪ History of estimated alcohol intake in excess of recommendation o Intake of >2 drinks/day (men) o Intake of >1 drink/day (women) o Binge drinking ▪ Hypertriglyceridemia ▪ Abnormal LFT readings
CLINICAL (NC)		
Overweight/Obesity/ Unintended weight gain	▪ Excessive energy intake ▪ Physical inactivity ▪ Food and nutrition related knowledge deficit concerning energy intake	▪ BMI > 25kg/m^2 or weight gain ▪ Self-reported estimated energy intake in excess of energy needs ▪ Intake of high caloric density or large portions of food/ beverages

	▪ Failure to adjust for lifestyle changes (e.g., decrease in physical activity) or decreased metabolism (e.g., aging) ▪ Failure to adjust for restricted mobility due to recovery from injury or surgical procedure ▪ Increase in psychological and life stress	▪ Waist circumference higher for normative standard for age and sex. ▪ Body fat percentage of >25% for men and >32% for women. ▪ Longer duration of sedentary activities (e.g. screen time).
Altered nutrition- related laboratory values (e.g. TC, LDL, HDL, TG)	▪ Food- and nutrition-related knowledge deficit concerning appropriate amount of dietary fat (SFA/ trans fat). ▪ Lack of or limited access to healthful food choices.	▪ Specific laboratory value (e.g. TC, LDL, HDL, TG) above/ below the reference range. ▪ Self-reported estimated fat (SFA/ trans fat) intake in excess of the needs. ▪ Frequent intake of high-SFA/ trans fat foods.
BEHAVIORAL-ENVIRONMENTAL (NB)		
Food and nutrition related knowledge deficit	▪ Unsupported beliefs/attitudes about food, nutrition and nutrition-related topics ▪ Lack of or limited prior nutrition-related education ▪ Limited interest in learning/ applying information ▪ Uncertainty on how to apply nutrition information ▪ Prior exposure to incorrect information	▪ Verbalizes inaccurate or incomplete information ▪ No prior knowledge of need for food- and nutrition-related recommendations ▪ No prior education provided on how to apply food and nutrition related information ▪ Verbalizes unwillingness or disinterest in learning information ▪ New medical diagnosis
Unsupported beliefs/attitudes about food or nutrition related topics	▪ Disbelief in science-based food and nutrition information. ▪ Lack of prior exposure to accurate nutrition-related information. ▪ Desire for a cure for a chronic disease (e.g. CVD) through the use of alternative therapy.	▪ Food faddism. ▪ Estimated intake that reflects an imbalance of nutrients/food groups. ▪ Intake of complementary and alternative medicine products and dietary supplements that may be unsupported for health.
Not ready for diet/lifestyle change	▪ Unsupported beliefs/attitudes about food, nutrition and nutrition-related topics ▪ Limited interest in learning/ applying information	▪ Negative body language (e.g. frowning, limited eye contact, defensive posture, limited focus, fidgeting)

	▪ Limited social support for implementing changes ▪ Limited acceptance of need to change ▪ Limited self-efficacy for making change or demoralization from previous challenges associated with change	▪ Denial of need for food- and nutrition-related changes ▪ Inability to understand required changes ▪ Limited self-efficacy to make change or to overcome barriers to change ▪ Resistance to change
Limited adherence to nutrition related recommendations	▪ Unsupported beliefs/attitudes about food, nutrition and nutrition-related topics ▪ Limited interest in learning/ applying information ▪ Limited social support for implementing changes ▪ Limited value for behavior change ▪ Limited confidence in ability to change ▪ Perception that limited resources (eg, time, financial) prevent changes ▪ Food and nutrition-related knowledge deficit concerning how to make nutrition-related changes ▪	▪ Expected anthropometric outcomes (e.g. weight changes, waist circumference) are not achieved ▪ Negative body language (e.g. frowning, limited eye contact, defensive posture, limited focus, fidgeting) ▪ Limited ability to recall changes agreed upon ▪ Limited or inconsistent adherence with agreed nutrition plan ▪ Verbalizes frustration or barriers to change in applying the food/nutrition information.
Undesirable food choices	▪ Lack of prior exposure to accurate nutrition-related information ▪ Cultural practices that affect the ability to learn/apply information ▪ Lack of or limited access to recommended foods ▪ Perception that lack of resources (eg, time, financial) prevent selection of food choices consistent with recommendations ▪ Lacks motivation and/or readiness to change ▪ Unwilling or disinterested in learning/applying information ▪ Psychological causes such as depression and disordered eating	▪ High lipid level (TC, LDL, and/or TG) ▪ Estimated intake inconsistent with Malaysian Dietary Guidelines or Healthy Plate concept or nutrition prescription. ▪ Inability to apply the guidelines, Healthy Plate concept or nutrition prescription ▪ Unwillingness or disinterest in selecting food consistent with the guidelines or recommendations

Physical inactivity	▪ Injury, lifestyle changes, medical conditions leading to physical disability or limitations that reduces physical activity of daily living ▪ Food and nutrition related knowledge deficit concerning health benefits of physical activity ▪ Limited social support for implementing changes ▪ Limited or limited access to safe exercise environment and/or equipment ▪ Limited value for behavior change ▪ Time constraints	▪ Obesity: BMI >30 kg/m^2 or waist circumference of >90cm for men and >80cm for women ▪ Infrequent, low-duration and/or low-intensity physical activity (e.g. less than 30 minutes of exercise five days a week) ▪ Longer duration of sedentary activities (e.g. screen time)

Source: AND (eNCPT) 2020 [17].

Appendix C: NUTRITION EDUCATION IN PRACTICE

<table>
<tr><td>Eating out</td><td>▪ Order foods that are grilled, roasted, stir-fried, steamed, cooked soup, singgang/pindang, asam pedas or asam rebus.
▪ Do not finish off the gravy/ soup of a dish.
▪ Cut down the processed foods, canned foods and fast foods.
▪ Remove skin of the poultry.
▪ Use a squeeze of lemon, vinegar or low fat yogurt instead of mayonnaise or cream dressing on salads.</td></tr>
<tr><td>Recipe modification</td><td>▪ When a recipe calls for high cream, butter or oil usage, suggest reducing the cream, butter or oil usage appropriately.
▪ Replace coconut milk with low fat milk/ yogurt in the cooking.
▪ Consider using non-stick pan, oven or air fryer to minimise oil usage in the cooking.
▪ Choose lean meat/ poultry instead of fatty meat and processed foods.</td></tr>
<tr><td>Simple changes for implementing the Mediterranean diet in patient's diet
(Adapted from RACGP 2018 [7])</td><td>▪ Using olive oil instead of other oils/ fats for cooking and dressing salads.
▪ Consuming vegetables with every meal (including leafy greens and tomatoes)
▪ Consuming 2–3 serves of fresh fruit per day
▪ Consuming legumes (cooked dried beans) 3 times per week (in salads, soups, casseroles)
▪ Having 2–3 serves of fish per week (at least one oily fish such as salmon or sardines)
▪ Eating at least 3 serves of nuts per week (include walnuts and almonds)
▪ Choosing white meat (poultry without skin) instead of fatty processed meats (sausages, burgers) and keep red meat portions small and lean.
▪ Choosing low fat (unsweetened) yogurt as snack on most days.
▪ Cooking regularly (at least twice a week) with tomato, garlic and onion, and aromatic/culinary herbs as a base for pasta sauces, casseroles and baked dishes.</td></tr>
<tr><td>Food labelling</td><td>▪ Educate the patient to read food label on fat content per serving.
▪ Below is the guide to common claims on food labels and their definitions:
<table>
<tr><th>Component</th><th>Claim</th><th>Not more than</th></tr>
<tr><td rowspan="2">Fat</td><td>Low</td><td>3 g per 100 g (solids)
1.5 g per 100 ml (liquids)</td></tr>
<tr><td>Free</td><td>0.5 g per 100 g (solids)
0.5 g per 100 ml (liquids)</td></tr>
<tr><td rowspan="2">Saturated fat</td><td>Low</td><td>1.5 g per 100 g (solids)
0.75 g per 100 ml (liquids)
and 10% of total energy of the food</td></tr>
<tr><td>Free</td><td>0.1 g per 100 g (solids)
0.1 g per 100 ml (liquids)</td></tr>
</table></td></tr>
</table>

	Cholesterol	Low	0.02 g per 100 g (solids) 0.01 g per 100 ml (liquids)
		Free	0.005 g per 100 g (solids) 0.005 g per 100 ml (solids)
	Trans fatty acids	Low	1.5 g per 100 g (solids) 0.75 g per 100 ml (liquids) and 10% of total energy of the food
		Free	0.1 g per 100 g (solids) 0.1 g per 100 ml (liquids)
	Source: MDG 2020 [55]		

Appendix D: FATTY ACID COMPOSITION OF SELECTED DIETARY FATS AND OILS

Type of fats and oils	SFA	MUFA	PUFA
Beef fat	50.6	42.1	2.8
Canola oil	7.4	56	35.6
Cocoa butter	60.4	35.6	2.9
Coconut oil	91.9	6.5	1.5
Corn oil	14.2	27.8	57.1
Groundnut oil	9.6	71.2	18.2
Lard	38.7	48.2	11
Olive oil	18.8	68.2	14.6
Palm kernel oil	84.2	13.7	2
Palm oil	44.9	43.4	10.8
Palm olein	42.4	44	11.8
Safflower oil	9.2	11.6	79.2
Shea butter	46	48	5.1
Soybean oil	14.8	24.1	59.9
Sunflower oil	9.1	28.1	62.4

Notes: values represent % per 100g edible fat

Source: Malaysian CPG 2018 [11]

Appendix E: FOOD HIGH IN MONOUNSATURATED FAT (MUFA) AND POLYUNSATURATED FAT (PUFA)

	MUFA, %	PUFA, %	SFA, %
PLANT BASED OILS			
Almond oil	70	17	8
Avocado oil	71	13	12
Canola oil	63	28	7
Coconut oil	6	2	86
Corn and canola oil	58	29	8
Flaxseed oil, cold press	18	68	9
Hazelnut oil	78	10	7
High-oleic safflower oil	72	13	7.5
Mustard oil	59	21	12
Olive oil	73	10.5	14
Peanut oil	46	32	17
Sesame oil	40	42	14
Soybean oil	23	58	16
Sunflower oil (linoleic acid <60%)	45	40	10
Sunflower oils (linoleic acid >70%)	14	75	6
Walnut oil	23	63	9
NUTS & SEEDS			
Almonds	31	11	4
Cashew nuts, dry roasted	27	7	9
Chia seeds	5	33	8
Flaxseed	8	29	4
Hazelnut	46	8	4
Macadamia	59	12	2
Peanut	41	22	6
Pistachio nuts	24	14	5
Safflower kernels, dried	5	28	4
Sesame, whole, roasted and toasted	18	21	7
Sunflower seed kernels, dried	19	23	4
Walnuts	15	35	3
PRODUCTS OF ANIMAL ORIGIN			
Beef, steak	4.5	0.4	4.3
Beef, steak	4.5	0.4	4.3
Butter, salted	21	3	51
Cheese, cheddar	9	1	21
Chicken	0.9	0.75	0.8
Egg	3.6	2	3
Fish oil, cold liver	47	23	23
Mackerel	5.4	3.3	3.2
Milk, 3.7% fat	1	0.1	2.2
Pork, ham	8.3	2	6.5
Salmon	2.1	2.5	0.9

MUFA: Monounsaturated fat; PUFA: Polyunsaturated fat; SFA: Saturated fat

Sources: Minzangi et al. 2015; Schwingshackl & Hoffmann 2012; Sheashea et al. 2021 [56-58]

Appendix F: SATURATED FAT AND CHOLESTEROL CONTENT IN COMMON FOOD

Food Group	Food Item	Portion	Saturated Fat (g)	Dietary Cholesterol (mg)
Dairy Product	Butter	1 tbsp	7.2	31
	Ghee	1 tbsp	13.14	58
	Cheese, cheddar	30g	6	30
	Cheese, mozarella, reduced fat	30g	3.1	15
	Cream cheese	1 tbsp (20g)	4.08	18.6
	Cottage cheese	1 tbsp (20g)	0.69	4.4
	Blue cheese	1 tbsp (20g)	3.73	15
	Ricotta cheese	1 tbsp (20g)	1.28	9.8
	Parmesan cheese	1 tbsp (20g)	3.75	18
	Feta	1 tbsp (20g)	2.66	17.77
	Edam	1 tbsp (20g)	3.73	17.77
	Gouda	1 tbsp (20g)	3.52	22.78
	Mascarpone	1 tbsp (20g)	5.33	23.33
	Cooking cream	1 cup	5	30
	Margarine	1 tbsp	2.1	0
	Milk, full cream	1 glass (250 g)	5.6	34
	Milk, nonfat / skimmed	1 glass (250 g)	0	5
	Milk, reduce fat	1 glass (250 g)	2.9	20
	Milk, sweetened condensed	1 tbsp	1.1	N/A
	Whipping cream	1 tbsp	3	20
	Evaporated milk, full cream	1 glass (250 g)	13.08	60
	Evaporated milk, reduced fat	1 glass (250 g)	3.38	25
	Filled milk	1 glass (250 g)	1.93	5
Dairy-free, plant-based milk alternative	Almond milk	1 glass (250 g)	0.28	N/A
	Almond milk, unsweetened	1 glass (250 g)	0.78	N/A
	Macadamia milk, unsweetened	1 glass (250 g)	1.0	N/A
	Hazelnut milk (packaged)	1 glass (250 g)	0	N/A
	Oat milk	1 glass (250 g)	0.9	N/A
	Rice milk	1 glass (250 g)	0.25	N/A
	Soy milk	1 glass (250 g)	0.68	N/A
	Mixed nut milk	1 glass (250 g)	0.5	N/A
	Coconut milk, thin	1 glass (250 g)	22.6	0
	Coconut milk, thick	1 glass (250 g)	54.4	0
Eggs	Egg, fresh, whole	1 medium	1.4	187
	Century egg, whole	115g	N/A	325
	Duck egg, whole	70g	2.86	358.8
	Quail egg, whole	12g	0.36	86.1
Meats and Poultry	Beef, ground, lean	90g	5.9	86
	Beef, tenderloin, meat only	90g	3.1	73
	Chicken, breast (with skin)	90g	1.8	71
	Chicken, breast (without skin)	90g	0.9	73
	Chicken, thigh (without skin)	90g	2.5	80
	Ham, regular	90g	2.9	49
	Hotdog, beef	45g	5.4	27

	Lamp, loin chop, lean only	90g	3.2	74
	Pork, loin chop, meat only	90g	3	69
	Oxtail, raw	121g	4.21	55.18
	Chicken liver, raw	44g	0.67	148.76
	Chicken gizzard, raw	145g	0.77	348
	Chicken feet, raw	35g	0.77	16.46
	Pig lung, raw	101g	0.97	323.2
Fish and Seafood	*Bawal hitam*	90g	N/A	51
	Bawal putih	90g	N/A	27
	Clams	90g	0	57
	Cod	90g	0	45
	Crab	90g	0.2	70
	Fish roe	30g	N/A	98
	Ikan bilis, without head	½ cup	N/A	37
	Ikan cencaru	90g	N/A	46
	Ikan kembong	90g	N/A	55
	Ikan merah	90g	N/A	14
	Ikan selar	90g	N/A	47
	Ikan terubuk	90g	N/A	74
	Lobster	90g	0	69
	Oysters	90g	0.9	85
	Salmon (varies by species whether farm-raised or wild)	90g	0-2.7	47-81
	Sardine, canned in tomato sauce	90g	N/A	107
	Scallops	90g	0	28
	Shrimp	90g	0	166
	Squid	90g	N/A	90
	Tenggiri	90g	N/A	3
	Tuna, canned in water, light	90g	0	26
	Tuna, fresh	90g	0	50

N/A: Not available

Sources: Energy and Nutrient Composition of Food, Singapore Health Promotion Board 2011; Malaysian Food Composition Database Programme, c/o Institute for Medical Research, Kuala Lumpur, 1997-2015; The Australian Food Composition Database– Release 3, Food Standards Australia New Zealand (FSANZ) 2025.

Appendix G: SAMPLE MENU OF MEDITERRANEAN DIET

The following are one-day sample menus of Mediterranean diet. However, adapting the Mediterranean diet to suit the patient's usual dietary intake should be considered.

One day sample menu of Mediterranean diet for 1500 kcal:

Meal	Food & drinks	Quantity
Breakfast	Overnight oats with + low fat milk + chia seeds + banana + walnuts	6 tablespoons 1 cup 2 teaspoons 1 small 1 tablespoon
Lunch	Brown rice Steamed fish with ginger & scallion, drizzled with olive oil Stir-fried kailan Watermelon Plain water	1 cup 1 palm size ½ cup 1 slice 1 cup
Dinner	Wholemeal chapati Lentil dhal Cucumber raita Guava Plain water	2 pcs ½ cup ½ cup ½ medium 1 cup
Snack (e.g.teatime)	Tauhu sumbat Plain tea (no sugar)	1 piece 1 cup

* Suggest using olive oil in cooking
** Ensure to choose Greek yogurt and not Greek style yogurt or flavoured yogurt.

Nutrition content per day:

Calories (kcal)	1510
Carbohydrate (g)	201
Protein (g)	66
Fat (g)	50
SFA (g)	6.9
MUFA (g)	14
PUFA (g)	11
Trans-fat (g)	0.1
Cholesterol (mg)	65
Fiber (g)	23
Sodium (mg)	801

One day sample menu of Mediterranean diet for 1800 kcal:

Meal	**Food & drinks**	**Quantity**
Breakfast	Mee hoon soto + bean sprout + mustard green + shredded chicken + toasted peanuts Coffee + low fat milk	1 cup ¼ cup ¼ cup ½ palm size 2 tablespoons ½ cup
Morning tea	Toasted almonds Plain low fat yogurt	2 tablespoons 1 tub
Lunch	Rice Assam pedas fish + ladies finger + brinjal + tomato *Ulam* Watermelon Plain water	1½ cup 1 palm size ¼ cup ¼ cup ¼ cup ½ cup 1 small slice 1 cup
Afternoon tea	Mung bean porridge + low fat milk + brown sugar	1 cup ½ cup 2 teaspoons
Dinner	Rice Sesame chicken Mixed vegetable soup + spinach + carrot Papaya Infused water	1½ cup 1 palm size ½ cup ½ cup 1 slice 1 cup

***** Suggest using olive oil in cooking

Nutrition content per day:

Calories (kcal)	1864
Carbohydrate (g)	223
Protein (g)	110
Fat (g)	60
SFA (g)	10.6
MUFA (g)	17
PUFA (g)	11
Trans-fat (g)	0.2
Cholesterol (mg)	140
Fiber (g)	28
Sodium (mg)	1150

Appendix H: EXAMPLES OF AEROBIC EXERCISE

Aerobic exercise	Examples
Moderate-intensity	▪ Brisk walking (at least 4km per hour) ▪ Water aerobics ▪ Dancing (ballroom or social) ▪ Gardening ▪ Tennis (doubles) ▪ Cycling slower than 16km per hour
Vigorous-intensity	▪ Hiking uphill or with a heavy backpack ▪ Running ▪ Swimming laps ▪ Aerobic dancing ▪ Heavy yardwork like continuous digging or hoeing ▪ Tennis (singles) ▪ Cycling 16km per hour or faster ▪ Jumping rope

Source: AHA 2021 [59]

Appendix I: ALCOHOL STANDARD DRINK

Beer 12 fl oz (~350ml)	 about 5% alcohol
Malt liquor 8-9 fl oz (~240 – 270 ml)	 about 7% alcohol
Table wine 5 fl oz (~150ml)	 about 12% alcohol
Distilled spirits 1.5 fl oz shot (~45ml) (e.g. gin, rum, tequila, vodka, whiskey)	 about 40% alcohol

1 standard unit = 14g of pure alcohol

Source: National Institutes of Health [60]

Appendix J: TYPES OF SMOKING

Types of smoking	Description
Tobacco	▪ Tobacco is a plant grown for its leaves, which are dried and fermented before being put in tobacco products. ▪ Tobacco contains nicotine, the ingredient that can lead to addiction. ▪ People can smoke, chew, or sniff tobacco. a) Smoked tobacco products include cigarettes, cigars, bidis, kreteks, loose tobacco in a pipe or hookah (water pipe) b) Chewed tobacco products include chewing tobacco, snuff, dip, and snus. c) Snuff can also be sniffed.
Electronic cigarettes	▪ Electronic cigarettes, also known as e-cigarettes or e-vaporizers. ▪ They are battery-operated devices that deliver nicotine with flavorings and other chemicals to the lungs in vapor instead of smoke.

Source: National Institute on Drug Abuse [61]

Appendix L: LIST OF LIPID LOWERING MEDICATION

Classes	Example of drugs	Mechanism of action	Indication
HMG-CoA Reductase Inhibitors	Atorvastatin Simvastatin Rosuvastatin	Statins are inhibitors of HMG CoA reductase, the rate limiting enzyme in hepatic cholesterol synthesis.	▪ Reducing LDL-C ▪ Reducing risk of CVD
Fibric-Acid Derivatives	Fenofibrate Gemfibrozil Bezafibrate Ciprofibrate	▪ Fibrates are Peroxisome Proliferator Activated Receptor (PPAR) – α agonist which have an important role in fatty acid oxidation. ▪ They reduce serum TG effectively and increase HDL-C modestly.	Its use is limited to the treatment of patients with very high TG levels who do not respond to non-pharmacological measures.
Proprotein convertase subtilisin/ kesin type 9 (PCSK 9)	Evolocumab Alirocumab	▪ Target the proprotein convertase subtilisin kexin type 9 (PCSK9) ▪ Inhibiting the binding of PCSK9 to the LDL-receptors. This interaction decreases the degradation of the LDL-receptors, resulting in higher LDL-receptors density at the cell surface. The higher expression of LDL-receptors at the cell surface leads to increased clearance with resulting decrease in LDL-C levels	▪ Individuals with high CVD risk who have persistently elevated LDL-C despite optimum lipid-modifying therapy ▪ Familial hypercholesterolemia
Small interfering RNA (siRNA) PCSK-9 Inhibitors	Inclisiran	▪ A long-acting, synthetic siRNA molecule that binds to a RNA-induced silencing complex (RISC), thereby inhibiting the translation of PCSK9 messenger RNA (mRNA) causing a marked reduction in LDL-C levels	▪ Reducing LDL-C
Bile-Acid Sequestrants (Anion exchange resins)	Cholestyramine	▪ Bile acid sequestrants bind to bile acids to promote their secretion into the intestines ▪ They are effective in lowering LDL-C	Discouraged in patients with TG ≥3.4 mmol/L.2
Nicotinic Acid	Nicotinic acid (Niacin)	▪ Decreases mobilization of free fatty acids from adipose tissues. ▪ It increases HDL-C and lowers TG levels	Alternative therapy to fibrates in individuals with elevated TG not responsive to non-pharmacological methods.
Cholesterol Absorption Inhibitors	Ezetimibe	▪ Selectively blocks intestinal absorption of both dietary and	▪ Combination with any dose of any statin to

		biliary cholesterols and other phytosterols. ▪ This leads to a reduction in hepatic cholesterol delivery – a mechanism which complements the action of statins.	further lower LDL-C if targets are not achieved ▪ May be considered as monotherapy in patients who cannot tolerate statins

Source: Malaysian CPG 2018 [11]

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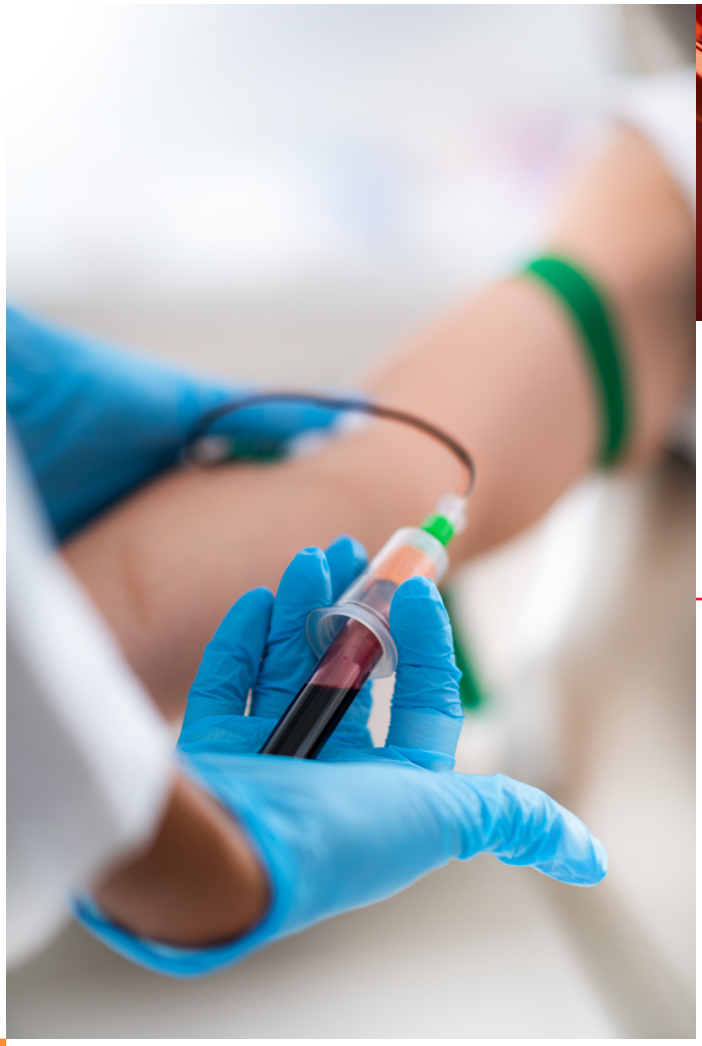
Post-Guideline Development Update

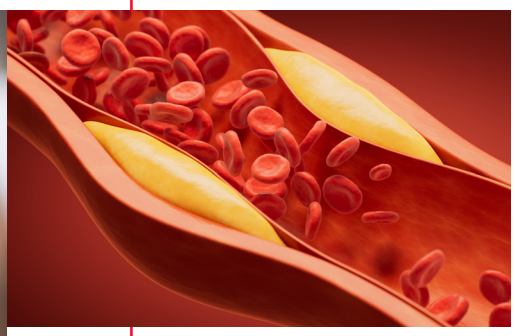
Recent evidence (2025–2026) highlights emerging areas in dyslipidemia management, including the growing emphasis on lipid risk assessment beyond low-density lipoprotein (LDL) cholesterol, incorporating markers such as apolipoprotein B (ApoB), non–HDL cholesterol, and triglycerides to better reflect overall atherogenic burden.

It also reinforces the importance of dietary patterns that extend beyond single nutrient-based approaches to encompass overall dietary quality and habitual eating patterns. In addition, increasing attention has been given to the role of food processing, particularly the impact of ultra-processed foods on cardiometabolic health.

Dietitians are encouraged to remain abreast of evolving evidence and updated clinical guidelines to ensure that medical nutrition therapy remains evidence-based and responsive to current advances in dyslipidemia and cardiometabolic care.

To be advocators of nutrition and dietetics for the health and well-being of the nation





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