

The efficacy of using the Therapeutic Lifestyle Changes diet for reducing comorbidities associated with overweight and obesity



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Learning objectives

After reading this article the reader will be able to:

1. Explain the rationale for the use of the Therapeutic Lifestyle Changes (TLC) diet as an effective therapeutic strategy for individuals at risk for type 2 diabetes and/or coronary heart disease (CHD).
2. Identify the difference(s) between the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP III) and TLC diets.
3. Explain the relationship between each of the “maximal diet therapy” components targeted in the TLC diet and CHD risk.
4. Recognize the steps of the Nutrition Care Process.
5. Understand how the Nutrition Care Process and Evidence-Based Nutrition Practice Guidelines relate to care for patients with diabetes, CHD and overweight/obesity.

The increasing prevalence of overweight and obesity is of major concern in the United States. According to National Health and

Nutrition Examination Survey (NHANES) 2003–2004 data, nearly two-thirds of U.S. adults are either overweight or obese (1). This represents an approximate 18% increase over the past three decades, with the prevalence of obesity contributing most to this increase, roughly doubling from 15% to 31% (2).

Obesity is recognized as an independent risk factor for numerous chronic disease conditions, including hypertension, dyslipidemia and type 2 diabetes (3–6). Type 2 diabetes is three to six times more prevalent in obese adults (body mass index [BMI] 30 and above) compared with normal-weight individuals (BMI between 18.5 and 24.9) (7). Moreover, an overweight adult (BMI between 25 and 29.9) triples his/her risk of developing type 2 diabetes within 10 years (8). In addition, individuals with diabetes are at an increased risk of developing CHD (9). CHD comprises more than 50% of all cardiovascular disease-related events in U.S. adults and is the leading cause of diabetes-related death (10).

Of concern is the tendency for type 2 diabetes and CHD risk factors to co-occur, thus complicating treatment options. An estimated 65% and 72% of overweight and obese Americans, respectively, have hypertension, dyslipidemia or both (3). A study examining over 1.9 million members of a large managed-care program found hypertension, dyslipidemia and/or type 2 diabetes to commonly co-occur in more than 50% of affected individuals (11). Consequently, it is likely that health-care practitioners will be required to treat overweight or obese individuals with comorbid conditions, including hypertension, dyslipidemia, and/or type 2 diabetes. Fortunately, healthy eating patterns and moderate weight loss often improve the risk factors associated with type 2 diabetes and CHD.

Tools such as the *American Dietetic*

Association’s (ADA) Evidence-Based Nutrition Practice Guidelines for Disorders of Lipid Metabolism, Adult Weight Management, and Diabetes 1 and 2 may be valuable resources for the nutrition care of patients and clients with these diseases (12–14). Additionally, using ADA’s Nutrition Care Process and Standardized Language can assist in prioritizing the nutrition problems and determining the appropriate interventions for care (15).

Medical nutrition therapy (MNT)

Therapeutic lifestyle changes (TLC) have been shown to be an effective therapy in reducing CHD risk (16). TLC is the nonpharmacologic component of the Third Report of the NCEP and ATP III, which emphasizes diet, physical activity, behavior change and weight loss (if indicated). The ATP III guidelines specifically target low-density lipoprotein cholesterol (LDL) due to its strong, positive correlation with CHD risk (9). Although drug therapy may also be implemented based on a person’s absolute risk for CHD, ATP III places a major emphasis on TLC as an essential modality for persons at risk for CHD (12). Registered dietitians (RDs) have a vital role in delivering MNT that involves each component of the TLC. Specifically, MNT by a RD over several patient/client encounters can lead to a reduction in total cholesterol (T-Chol), LDL, and triglycerides; improved weight status; and may lead to reduction or discontinuation in drug therapy (12). Further, increasing the number and duration of encounters with the RD may benefit the higher-risk patient (12).

Although the TLC diet specifically targets lowering LDL, it has also been shown to positively affect blood pressure (BP) and T-Chol (17). In addition, the TLC diet positively influences serum triglyceride (TG) levels with little or no effect on high-density lipoprotein cholesterol

(HDL) levels (18–19). This is especially important for those individuals with type 2 diabetes, the metabolic syndrome, and their associated dyslipidemias.

As previously mentioned, TLC is a multifaceted approach that includes specific dietary recommendations, referred from herein as the “TLC diet.” The TLC diet differs from previous NCEP diet guidelines (Step I and Step II diets) in that it de-emphasizes “total fat” while focusing on “types” of fat. Specifically, the TLC diet emphasizes reducing dietary cholesterol (<200 mg./day), saturated fatty acids (SFA) (<7% of total calories), and trans fatty acids (lower intake) while allowing total fat to comprise 25%–35% of total calories, and limiting total carbohydrates to no more than 60% of total calories (Table 1). Daily sodium intake follows the Dietary Guidelines for Americans 2005 recommendation of less than 2,300 mg./day (20).

In addition, optional nutrient considerations for “maximal” LDL reduction include augmenting the diet with plant stanols/sterols (2 g./day) and viscous (soluble) fibers (5–10 g./day) (Table 2). Incorporating TLC will be most effective when the RD works with the patient using the four steps of the Nutrition Care Process (assessment, diagnosis, intervention and monitoring and evaluation) in order to focus each intervention and promote the intended nutrition outcomes (21).

TLC dietary components

The TLC diet meets both the Dietary Guidelines for Americans 2005 and the 2006 American Heart Association (AHA) Diet and Lifestyle Recommendations (20,22). A preliminary nutrient analysis of the TLC menu plan at various calorie levels indicates that it provides the recommended nutrient levels of specific dietary components known to affect CHD risk (9). Overall, the TLC diet is low in SFA, dietary cholesterol and sodium, while providing adequate levels of monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), and potassium. This macro- and micronutrient distribution holds true at the reduced calorie levels,

Table 1. Nutrient composition of the TLC diet

Nutrient	Recommended intake
Saturated fat*	<7% of total calories
Polyunsaturated fat	Up to 10% of total calories
Monounsaturated fat	Up to 20% total calories
Total fat	25%–35% of total calories
Fiber	20–30 g./day
Protein	Approximately 15% of total calories
Cholesterol	<200 mg./day
Total calories‡	Balance energy intake and expenditure to maintain desirable body weight/prevent weight gain

* Trans fatty acids are another LDL cholesterol-raising fat that should be kept at a low intake.

† Carbohydrates should be derived predominantly from foods rich in complex carbohydrates including grains, especially whole grains, fruits, and vegetables.

‡ Daily energy expenditure should include at least moderate physical activity (contributing about 200 kcal./day).

Summary of the Therapeutic Lifestyle Change (TLC) diet based on NCEP ATP III guidelines. Adapted from (9).

making it a high-quality diet for weight reduction.

Lipids

Although the exact mechanisms by which diet affects serum lipids have yet to be elucidated, the serum TG and HDL effects observed with the TLC diet may in part be due to the diet’s macronutrient distribution. The TLC diet is slightly higher in total fat and MUFA and lower in carbohydrates than the previous NCEP II diet (23). Higher carbohydrate diets (>60% total calories) tend to increase serum TG and reduce HDL levels, whereas diets that replace a portion of the dietary carbohydrate with MUFA or PUFA, often decrease serum TG levels with little or no decrease in HDL levels (18,24–25). A recent study looking at the effect of various macronutrient distributions on CHD risk found that partial substitution of carbohydrate with either protein

(half from plant sources) or unsaturated fat (primarily monounsaturated) improved BP, T-Chol, LDL, HDL and TG levels and reduced the risk of CHD (18). This is an important consideration for those with insulin resistance or type 2 diabetes with concomitant dyslipidemia (elevated triglycerides and low HDL levels) (9).

Sodium

Of note, due to similar sodium, fruit and vegetable recommendations, the sodium and potassium content of the Dietary Approaches to Stop Hypertension (DASH) and TLC diets are comparable. It is well-established that the DASH diet is an effective MNT for the treatment of hypertension (26–28). The fact that the TLC diet also has a low sodium/high potassium nutrient profile likely accounts for its

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Table 2. Maximal dietary therapy for LDL-c reduction

Dietary modification	Recommendation	Approximate LDL-c reduction (%)
Saturated fat reduction	Reduce saturated fat to <7% total calories	8–10
Cholesterol reduction	Reduce dietary cholesterol to <200 mg./day	3–5
Dietary fiber	Viscous (soluble) fiber 5–10 g./day	3–5
Weight reduction	10-pound weight loss	5–8
Total LDL-c lowering		25–30

Recommendations for Maximal Dietary Therapy for LDL-c Reduction. Adapted from (9).

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blood pressure-lowering effects (17).

Weight loss

Another benefit of the TLC diet is that it has been shown to promote weight loss in free-living conditions (17,19). In addition to dietary modifications, moderate weight loss often improves fasting blood glucose, BP and serum lipid levels (6,29–31). Weight loss is an effective therapeutic strategy for reducing the risk of type 2 diabetes. Moderate weight loss in middle-aged adults with impaired glucose tolerance has been shown to reduce the risk of developing type 2 diabetes by more than 50% over a four year period (32). Weight loss has a similar effect on hypertension. Analyses of a Framingham Study cohort composed of overweight, normotensive middle-aged adults found that a moderate weight loss of 6.8 kg. or more reduced the long-term risk of hypertension by 28% (33). Several organizations including the ADA, the North American Association for the Study of Obesity, The Obesity Study (NAASO), the American Society for Clinical Nutrition (ASCN), and the AHA recommend weight loss for individuals at risk for type 2 diabetes and/or CHD by using TLC dietary components (22, 34–35).

Maximal dietary therapy

The primary goal of the TLC diet is to

achieve as much LDL lowering as possible, as this can be achieved through “maximal dietary therapy” as described in the ATP III Guidelines (16).

Recommendations for this therapy are listed in Table 2. The cumulative effect of these recommendations can reduce LDL by 25%, compared with a typical U.S. diet (16,36). This level of LDL reduction is similar to what can be achieved through drug therapy (37–38). The maximal dietary therapy modifications include SFA and cholesterol reduction, increased plant stanols/sterols and soluble fiber intake, and weight reduction. Each patient or client will have different nutrition adequacy, health status, and environmental/behavioral status, thus a systematic process for obtaining, verifying and interpreting data during assessment is important (21).

Saturated fatty acids and trans fatty acids

There is a robust scientific foundation supporting SFA intake as the primary LDL-raising dietary factor. It has been estimated that for every 1% increase in total kilocalories from SFA, there is a concomitant 2% increase in LDL (16). Conversely, LDL is lowered by 2% for every 1% decrease in total kilocalories from SFA. A recent weight loss study of obese adults found that a TLC-like diet deriving less than 7% of total kilocalories from SFA reduced LDL serum levels

an average of 8% (19).

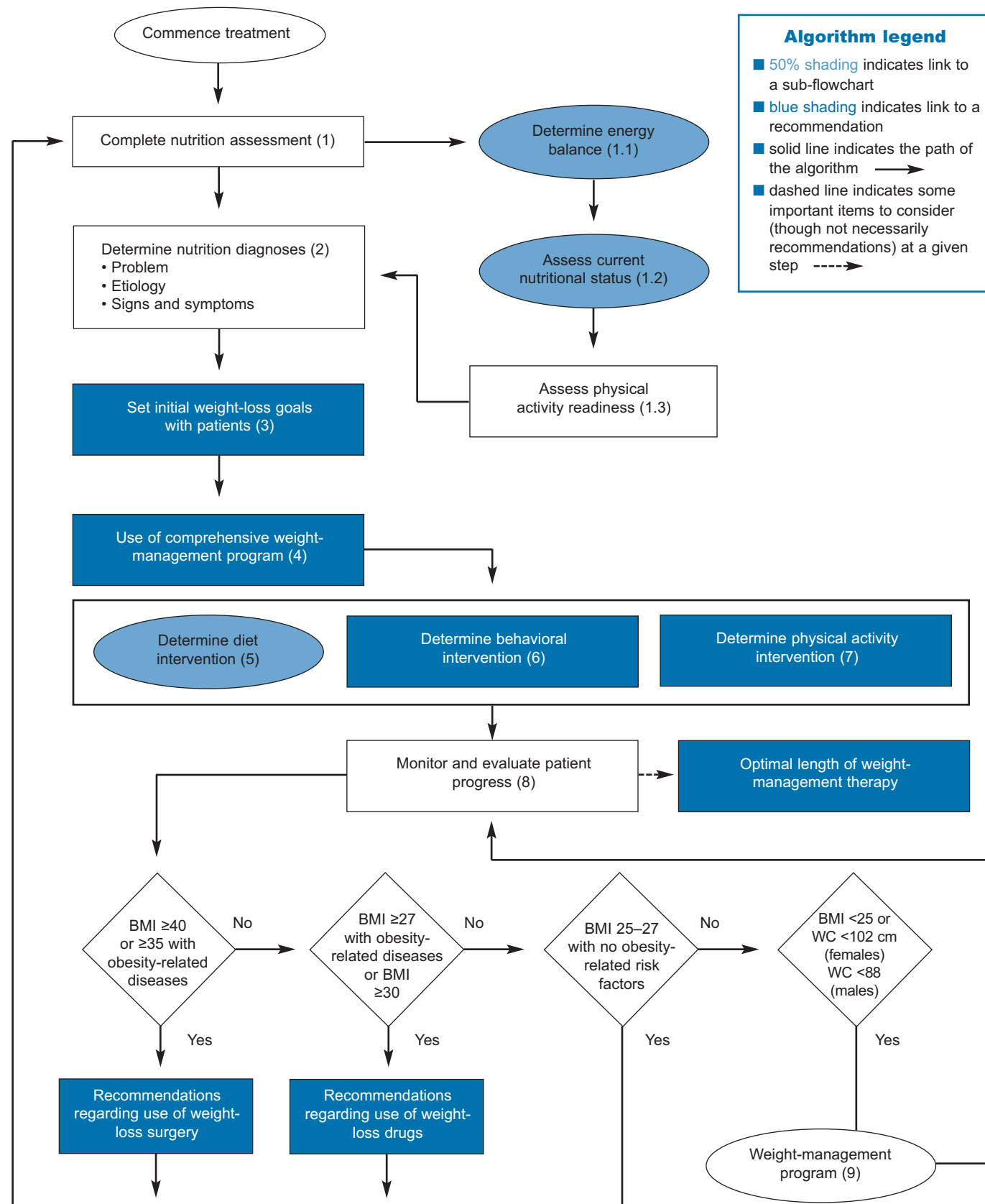
The TLC diet also recommends that intakes of trans fatty acids be kept low. Trans fatty acids raise LDL and lower HDL, which has a significant negative impact on the LDL-to-HDL ratio (39). Further, the ADA Disorders of Lipid Metabolism Evidence-based Nutrition Practice Guideline advises that trans fatty acid consumption be as low as possible and that a cardioprotective dietary pattern should contain less than 7% of calories from saturated fat and trans fatty acids (12). In addition to their effects on HDL and LDL, trans fatty acids are also believed to increase serum lipoprotein (a), TG, and endothelial dysfunction (39–40). Trans fatty-acid intake can be reduced by substituting liquid vegetable oils, soft margarine, and trans fatty acid-free margarines for butter, stick margarine, and shortening.

MUFA and PUFA

Replacing SFA (and trans fatty acids) with either MUFA or PUFA has been shown to lower CHD risk. Data from the 20 Years Follow-up of the Nurses’ Health Study found that a higher PUFA intake (approximately 7% of total kilocalories) in women is associated with a decreased risk of CHD (41). Although there are no known controlled clinical trials comparing MUFA and SFA on CHD risk, substituting MUFA with SFA has been shown

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Weight management treatment algorithm



Source: ADA Evidence Analysis Library, Adult Weight Management Evidence-based Nutrition Practice Guideline. Available at www.adaevidencelibrary.com/topic.cfm?cat=2846. Accessed Feb.16, 2007.

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to decrease serum LDL. Moreover, partially replacing carbohydrate with MUFA can decrease serum TG levels with little or no decrease in HDL levels (18, 24–25). Therefore, the TLC diet recommends MUFA and PUFA intakes of up to 20% and 10% of total kilocalories, respectively.

Dietary cholesterol

Although dietary cholesterol has been shown to cause a marked LDL increase in laboratory animals, this effect has not been shown to be as pronounced in humans. However, while the rise in serum LDL varies from person to person, meta-analyses of studies performed in controlled settings confirm that high dietary cholesterol intakes do indeed raise LDL levels (16).

Interestingly, although egg consumption accounts for more than one-third of cholesterol consumption in the United States, prospective data from the Nurses' Health Study and the Health Professionals Study found no significant impact on frequency of egg consumption on CHD risk, except among women with diabetes (42). Conversely, a more recent meta-analysis found that dietary cholesterol raises the T-Chol to HDL cholesterol ratio, negatively affecting the serum lipid profile (43). Therefore, due to the potential for dietary cholesterol to raise LDL, the TLC diet limits cholesterol to 200 mg./day.

Plant stanols/sterols

Plant sterols are derived from soybean and tall pine-tree oils. Sterol esters are typically produced by esterifying the plant sterols to unsaturated fatty acids, thus increasing lipid solubility. Hydrogenating the plant sterols produces plant stanols, which can also be esterified to produce stanol esters. Stanol/sterol esters are readily dissolved in oils or margarine and are currently available in a wide variety of foods, drinks, margarines and soft gel capsules. Once consumed, the esters undergo hydrolysis, releasing free stanols and sterols in a bioavailable form that blocks cholesterol absorption (44). Maximum dietary

effects occur at plant stanol/sterol intakes of approximately 2 g./day (16). In order to sustain LDL reductions, stanol/sterol-containing products must be consumed on a daily basis. In addition, adjustments to caloric intake need to be considered when margarine and food products containing stanols/sterols are consumed. ADA's Disorders of Lipid Metabolism Evidence-based Nutrition Practice Guideline recommends, "If consistent with patient preference and not contraindicated by risks or harms, then plant sterol and stanol ester-enriched foods consumed two or three times per day, for a total consumption of 2 or 3 g. per day, may be used in addition to a cardioprotective diet to further lower T-Chol by 4%–11% and LDL by 7%–15%. For maximal effectiveness, foods containing plant sterols and stanols (spreads, juices, yogurts) should be eaten with other foods. To prevent weight gain, patients should isocalorically substitute stanol- and sterol-enriched foods for other foods. Plant stanols and sterols are effective in people taking statin drugs (12)."

Soluble fiber

Soluble forms of dietary fiber can reduce LDL, whereas insoluble fibers have not been shown to significantly reduce LDL levels (16). A meta-analysis of trials related to soluble fibers found consuming 2–10 g./day produced a small, yet significant reduction in LDL (45). Therefore, ATP III guidelines recommend including soluble fiber-rich foods (oats, guar, pectin, and psyllium) totaling at least 5–10 g./day, which can reduce LDL by approximately 5%.

Weight reduction

The LDL-lowering effects of the TLC diet's "maximal dietary therapy" can be further enhanced by weight reduction in overweight individuals. A meta-analysis of 70 studies found weight loss to be associated with a significant decrease in LDL (46). This study estimated that a 10-pound weight loss is associated with a 4 mg./dL. decrease in LDL. In addition, the Multiple Risk Factor Intervention Trial (MRFIT) study

showed that weight reduction enhances the LDL-lowering effects of a low SFA and cholesterol diet (47). A more recent weight-loss study of obese postmenopausal women found that a modest 2% (4-pound) weight loss over a 10-week period was associated with a 9 mg./dL. decrease in LDL (48).

In addition to reducing LDL, weight loss is important for other CHD-related risk factors such as elevated TG and low HDL levels. Based on ATP III guidelines, the first three months of TLC therapy focuses on LDL reduction utilizing maximal dietary strategies (16). Thus, reduced intakes of SFA and cholesterol and other dietary options (plant sterols/stanols and increased viscous fiber) are introduced first. If the LDL goal is not achieved in the first three months, attention then shifts to other CHD risk factors, specifically elevated TG, low HDL levels, and the metabolic syndrome (NCEP-defined as including three of the following five conditions: increased waist circumference, hypertriglyceridemia, low HDL cholesterol, hypertension, and a fasting glucose of 110 mg./dL. or higher) (16).

Because overweight and obesity are highly correlated with the metabolic syndrome, weight reduction is a secondary target of TLC (16). As noted previously, the ADA, NAASO, and ASCN recommend weight loss for those individuals at risk for, or with type 2 diabetes (34). Based on NCEP criteria for the metabolic syndrome, an estimated 86% of adults with type 2 diabetes have the metabolic syndrome (49). Thus, weight reduction using a reduced kilocalorie TLC-based diet plan will likely be a consideration for those with CHD risk factors and/or at risk for type 2 diabetes.

Applying TLC using the Nutrition Care Process

Using the Nutrition Care Process to incorporate TLC involves a systematic method where RDs must think critically, evaluate data, and determine interventions that address the nutrition-related problems of the patient (21). An example

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of this flow of treatment is displayed in the algorithm on page 9, which outlines the Nutrition Care Process related to adult weight management (13). Similar to the TLC approach for CHD, weight loss or maintenance therapy that includes the combination of diet, physical activity and behavior therapy is more effective than using any one intervention alone (13).

Assessment data is used to focus the intervention and may come from a variety of sources, including referral information, medical records, the patient interview, and administrative data (21). Specific tools or questionnaires may also be used to obtain this data, such as physical activity readiness or nutrition quality of life. During the assessment, information is gathered about signs, symptoms, and etiology and is used to identify the pertinent nutrition diagnosis(es).

The nutrition diagnosis, or problem, is separate from a medical diagnosis and is the issue(s) that RDs are responsible for treating independently (15). The nutrition diagnosis can be determined by identifying the signs, symptoms, and etiology (i.e., cause or contributing risk factors) of the problem (15).

Determining the nutrition diagnosis involves critical thinking in order to find patterns and relationships among assessment data, making multidisciplinary connections, prioritizing the relative importance, and stating the problem clearly so that outcomes are measurable (21). While indicators such as saturated fat or cholesterol intake may be straightforward to measure, quantifying outcomes such as increased nutrition-related knowledge may not be as clear. Thus, the diagnosis documentation should be specific enough so that changes from one encounter to the next are apparent.

Signs and symptoms that characterize a nutrition diagnosis may include biochemical/anthropometric/physical exam measurements or findings, food/nutrition history, and client history information (15). Examples of etiologies that cause the symptoms are food- and nutrition-related knowledge deficit, lack of social

support, lack of value for behavior change, loss of appetite awareness, and lack of food planning (15). Common nutrition diagnoses for a patient with obesity, CHD or type 2 diabetes may include “excessive fat intake” or “excessive carbohydrate intake,” and interventions might focus on setting goals to decrease this behavior. A diagnosis of “food- and nutrition-related knowledge deficit” would require a different approach, which might include nutrition education as the intervention (15).

As the critical link from the assessment to the intervention, the nutrition diagnosis must be clearly stated and will be used to frame the intervention. Determining the intervention will involve matching the intervention with the patient’s needs, diagnosis and values, as well as setting goals, prioritizing, defining the nutrition prescription, and specifying time and frequency of care (21). Specifically, the intervention should focus on the etiology of the nutrition diagnosis (15).

A nutrition intervention focused around incorporating the TLC diet varies from patient to patient. However, in an outpatient setting, the intervention will most often include comprehensive nutrition education, nutrition counseling, and/or coordination of care. A nutrition counseling intervention involves working with the patient to “set priorities, establish goals, and create individualized action plans” that promote self-care (15). It includes determining and documenting the theory or approach (e.g., Behavior Modification, Transtheoretical Model, etc.), the strategy (e.g., goal-setting, motivational interviewing, self-monitoring, problem-solving, etc.) and the phase (e.g., involving, exploring-education, resolving, and closing) for the counseling intervention (15).

For example, if an assessment reveals that a patient has a diagnosis of physical inactivity related to a hectic daily routine and lack of a plan for physical activity, a counseling intervention for this diagnosis (aimed at the etiology) might include behavior modification (theory) to incorporate goal-setting to increase physical activity and self-monitoring

using activity logs (strategies) to help resolve (phase) the problem.

Some attention should be made to patients who may not be ready for change. A patient who is not ready for change will not benefit from certain interventions, such as nutrition education. Instead, an intervention using the Transtheoretical Model and focused on moving the patient/client from a pre-contemplation to a contemplation stage may be beneficial.

The final and often ongoing step of the Nutrition Care Process involves monitoring and evaluation, which can only be done effectively if the first three steps (assessment, diagnosis, intervention) have been done thoroughly.

The purpose of monitoring and evaluating is to determine if the patient is attaining the goals or desired outcomes (21). Nutrition care outcomes can be monitored, measured and evaluated when clear and measurable diagnoses have been documented. Outcomes such as biochemical or anthropometric levels may lend themselves to measurement; however, outcomes that involve change in knowledge or ability may require a more in-depth system or scale for viewing them in measurable terms. It is this step of the process that allows RDs to explain how a nutrition intervention—planned around the specific nutrition diagnosis—leads to improved nutrition outcomes.

The use of the Nutrition Care Process provides an effective *process* for care to improve health, while the TLC diet and ADA’s Evidence-based Nutrition Practice Guidelines provide the content of care for patients with CHD, diabetes, and/or overweight/obesity.

Summary

Due to the high prevalence of overweight/obesity and associated chronic disease risk in the United States, it is likely that health-care practitioners will be required to treat overweight or obese individuals with comorbid conditions. The TLC diet is an effective MNT treating both dyslipidemia and hypertension. Because it is also promotes weight loss while improving the CHD risk associated with the metabolic syndrome, the TLC

diet can also be an important therapeutic strategy in the prevention of type 2 diabetes. With the use of the Nutrition Care Process and tools such as ADA's Evidence-based Nutrition Practice Guidelines to focus the nutrition intervention, the patient has a better chance of reaching the expected outcomes of the TLC diet.

The findings and conclusions in this article are those of the authors and do not necessarily represent the views of CDC.

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