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Obesity and malnutrition in children and adults: A clinical review

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ABSTRACT

Background: In the U.S., children and adults are consuming more low-nutrient foods with added sugar and excess fats as compared to healthy, high-quality calories and micronutrients. This diet is increasing the prevalence of malnutrition and nutritional deficiencies, despite high calorie intake. This is a review of the common micronutrient deficiencies, the risk factors for malnutrition, dietary plans, and the health consequences in children and adults with obesity in the U.S.

Methods: This clinical review of literature was performed on the MEDLINE (PubMed) search engine. A total of 1391 articles were identified and after review, a total of 130 were found to be most pertinent.

Discussion: The most common micronutrient deficiencies found in patients with obesity were vitamin A, thiamine (B1), folate (B9), cobalamin (B12), vitamin D, iron, calcium, and magnesium, especially prior and after bariatric surgery. Diets that produced the most weight reduction also further puts these individuals at risk for worsening malnutrition. Malnutrition and micronutrient deficiencies can worsen health outcomes if not properly managed.

Conclusion: Adequate screening and awareness of malnutrition can improve the health outcomes in patients with obesity. Physiologic changes in response to increased adiposity and inadequate intake increase this population's risk of adverse health effects. Malnutrition affects the individual and contributes to worse public health outcomes. The recommendations for screening for malnutrition are not exclusive to individuals undergoing bariatric procedures and can improve the health outcomes of any patient with obesity. However, clearly, improved nutritional status can assist with metabolism and prevent adverse nutritional outcomes post-bariatric surgery. Clinicians should advise on proper nutrition and be aware of diets that worsen deficiencies.

1. Introduction

In the U.S., children and adults consume 27–30% of their total daily calories from low-nutrient, sugary foods [1]. Despite consuming high-calories, these calories have lower quality nutrients that creates risk for malnutrition [2]. Over 50% of patients with obesity have nutritional deficiencies [3]. Malnutrition can arise from insufficient intake or absorption of essential nutrients, in which varying degrees of overnutrition or undernutrition may lead to changes in body composition and diminished functioning [4]. Micronutrient deficiencies can also significantly affect the intellectual and emotional states of the body [4]. Data from the National Health and Nutrition Examination Survey 2001–2008 showed people from all weight categories had inadequate intake of micronutrients, including vitamins A, C, D, E, and the minerals calcium and magnesium [5]. The aim of this clinical review includes describing nutritional problems associated with obesity, identifying common dietary practices and their health consequences, and describing risk factors associated with developing malnutrition amongst children and adults with obesity in the U.S.

2. Methods

A review of literature was performed on the MEDLINE (PubMed) search engine. Keywords used for the search were *"obesity, malnutrition, risk, micronutrient, deficiency, dieting"* which produced 1058 search results. Publications were limited to 2002–2023, narrowing to 726 search results. The articles were prescreened by reviewing titles, abstracts, and study relevance. After prescreening, the articles were read in-depth to verify that they met inclusion criteria and were within the scope of this study. A total of 61 articles remained. An additional review of literature was performed pertaining to diets and weight reduction in adults and pediatrics. Keywords used for the search were *"low-fat, ketogenic, low*

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carbohydrate diet, intermittent fasting, very low energy diets, portion control diet, and low-glycemic" which produced a total of 333 studies. The articles were prescreened, reviewed, and evaluated in-depth to see if they met inclusion criteria and were within the scope of this study. A total of 69 articles remained after the final screening.

3. Discussion

3.1. Mechanisms of malnutrition in obesity

Physiologic changes associated with obesity include increased blood volume, cardiac output, adiposity, lean mass, and organ size [1–3,5–8]. These influence the volume of distribution of micronutrients (vitamins and trace elements driving bodily functions) [1–3,5–9]. Deficiency is defined as inadequate stores with ranges below those stated as normal or with clinical manifestations; malnutrition is one of its many manifestations [9].

Mechanisms of malnutrition in obesity include inadequate intake, defective storage and bioavailability of nutrients, or small intestine bacterial overgrowth (SIBO) where the microbiota metabolize vitamins reducing availability for absorption [1–3,5–8]. Low-grade inflammation, often seen in patients with obesity, also contributes to malnutrition by altering nutrient metabolism and transporter synthesis, resulting in oxidative stress and increased antioxidant utilization [1–3,5,6,8]. One example of this is when adipose tissue increases hepcidin synthesis during inflammatory conditions, resulting in decreased intestinal iron absorption and anemia [1–3,5,6,8]. Obesity related malnutrition can also be caused by excessive consumption of simple sugars, milk, and fat [5–7]. For example, excessive intake of simple sugars depletes thiamine stores, an essential cofactor in carbohydrate metabolism and cell energy production [5–7]. Lastly, several medications [Table 1] have been implicated as contributors to malnutrition [5–7,10]. Awareness of this interaction is essential to consider when prescribing medications to patients with obesity.

3.2. Significance of malnutrition

Malnutrition in obesity intensifies comorbid diseases [2–5,8,11,12]. The body allocates micronutrients to vital organs while compromising other body parts [2–5,8,11,12]. For instance, vitamin K is prioritized for blood coagulation at the expense of bone health [2–5,8,11,12]. Vitamin D and magnesium (Mg^{2+}) deficiencies alter glucose metabolism and pancreatic beta cell function, exacerbating diabetes [2–5,8,11,12]. Anabolic pathways within skeletal muscle can also be compromised, increasing risk for sarcopenic obesity, a state of high adiposity with low total body muscle mass [2–5,8,12]. Other consequences include diminished cognitive ability, metabolic complications, and compromised women's health [2–5,8,11,12]. Around 78.4% of U.S. children with obesity also face these challenges, and like adults, improper nutrition can lead to dyslipidemia, high blood pressure, and insulin resistance [13]. This also influences public health concerns because patients with obesity account for one-third of admissions to intensive care units [2–5,8,12]. Health outcomes for these critically ill patients with obesity are worsened by comorbid malnutrition [2–5,8,12].

Table 1
Commonly prescribed medications with associated malabsorption of micronutrients.

Medication	Mal-Absorbed Micronutrient(s)
Loop diuretics	Thiamine
Metformin	Thiamine, B12
Orlistat	Fat soluble vitamins
Angiotensin-converting enzyme inhibitors	B12
H2 Blockers, proton pump inhibitor	B12, Thiamine, Iron, Calcium, Magnesium
Colchicine	B12
Anti-convulsant	Folate
Oral contraceptives	Folate
Cancer treatment	Folate

3.3. Micronutrient deficiencies associated with obesity

Adipose tissue is the storage site of lipophilic vitamins, such as vitamins A, D, E, and K [1–3,5–8]. All other micronutrients are water soluble, not stored substantially, and can quickly deplete in a few weeks [10]. These effects are exacerbated by inadequate intake, poor diet, alcoholism, medications, SIBO, malabsorptive diseases, pregnancy, and lactation [3,10]. Insufficient intake can precede any of the following deficiencies as well.

3.3.1. Vitamin D

Vitamin D is the most common micronutrient deficiency in patients with obesity [6,10]. Vitamin D regulates gene expression, supports bone health through increased calcium absorption, and improves healing following surgeries [6,10]. Additional functions include anti-inflammatory properties and increased pancreatic insulin release [13–16]. Pro-vitamin D absorbed through UV light undergoes hydroxylation to become active, 1,25(OH2)D [6,10]. Adipose tissue helps regulate vitamin D metabolism and prevents toxicity from excess amounts [17]. Patients with obesity exhibit lower levels of enzymes, CYP27B and CYPJ2, involved in vitamin D activation [17]. Besides inadequate intake, limited sun exposure and sunscreen utilization are risk factors for deficiency [1,6,17]. Weight reduction is associated with vitamin D release into serum, but these effects are not substantial enough to compensate for deficiency [1,6,17]. Deficiency is associated with an increased risk of infections, autoimmunity, cancers, chronic diseases, and poor bone mineralization [6,10].

Low vitamin D may be linked to insulin resistance [13]. Hypertrophic adipocytes amplify pro-inflammatory factors while reducing insulin-sensitive adipokines like adiponectin and IL-10 [13]. Low serum vitamin D and adipokine imbalance preclude insulin resistance and subsequent diabetes [13]. Increased parathyroid hormone, or secondary hyperparathyroidism, in response to vitamin D deficiency, is also associated with decreased insulin sensitivity [13–16]. Furthermore, vitamin D depletion impairs pancreatic beta cell function and decreases insulin release into the serum [13–16]. These effects are linked to a 2-fold increased risk of diabetes, a 1.5-fold increased risk of hypertension or hypertriglyceridemia, and a 2-fold increased risk of metabolic syndrome [13–16].

Children, with or without obesity, are also at risk of deficiency [13,18]. Studies have found 1 in 5 children aged 1–11 years are vitamin D deficient [13,18]. Those aged 6–11 were at greater risk of deficiency (73%) compared to younger children (63%) [13,18]. Similar to adults, vitamin D may protect pancreatic beta cells from cytokine-induced apoptosis in children with type 2 diabetes [13].

3.3.2. Iron

Iron is a component of hemoglobin and myoglobin which is critical for erythropoiesis, oxygen transport, and adequate cellular function [10,19]. It is stored as heme or non-heme iron [10,19]. Heme iron has greater bioavailability and is found in red meat, poultry, pork, and seafood [10,19]. Non-heme iron is obtained from beans, dark leafy greens, and iron-fortified cereals; its absorption is maximized with concomitant vitamin C intake [10,19]. Once absorbed in the duodenum and proximal jejunum, approximately 60% is stored in the form of hemoglobin and the remainder in iron-binding proteins like ferritin and transferrin [10,19]. Males have approximately three times the storage capacity compared to females. Variations in storage capacity and loss through menstruation place women at higher risk of iron deficiency [10,19]. Other risk factors for deficiency include excess tannin (tea) consumption and gastric acid insufficiency caused by disease or medications

such as H2 blockers or proton pump inhibitors [10,19]. Diminished gastric acid limits the conversion from ferric to ferrous iron, which is more readily absorbed [10,19].

Hepcidin also contributes to mechanisms linked to iron deficiency [6,19]. Hepcidin is a small peptide hormone in the liver and adipose tissue that binds ferroportin, a protein necessary for iron-exporting cells [6,19]. Ferroportin bound by hepcidin is degraded, blocking intestinal absorption of iron [6,19]. Iron is subsequently sequestered within the spleen, liver, and macrophages, causing decreased release into plasma [6,19]. Cytokines, including *anti*-TNF and IL-6, released from adipose tissue in response to inflammation can stimulate hepcidin secretion from the liver [6,19]. Increased hepcidin mRNA levels in adipose tissues and subclinical inflammation associated with obesity increase patients' risk of iron deficiency [6,19]. These findings were further supported by increased serum iron levels following significant weight reduction in individuals with obesity [19].

3.3.3. Vitamin B9

Vitamin B9, also known as folic acid or folate, is utilized for DNA synthesis and repair, cell division and growth, formation of red blood cells (RBCs), amino acid metabolism, and fetal neural tube development [10]. Sources are leafy green vegetables, orange juice, lentils, beans, and fortified grains [10]. Adiposity does not affect folate absorption due to its hydrophilic properties; however, its distribution from circulation into tissues is affected [6]. Increased folate levels within red blood cells (RBCs), especially among women with obesity, supports the theory of less folate reaching tissues from circulation [6]. Other risk factors include malabsorptive syndromes leading to poor bioavailability [20]. An indicator of inadequate folate stores is increased serum homocysteine, which is strongly linked to cardiovascular disease [6,10,20]. Deficiency is associated with cardiovascular disease, cancers, megaloblastic anemia, and fetal neural tube defects [6].

3.3.4. Vitamin B12

Vitamin B12 (Cobalamin) supports DNA synthesis, neurologic functioning, RBC maturation, and cellular metabolism [10]. Dietary sources include fish, red meat, eggs, and dairy [10]. The vitamin B12 is released from protein by gastric acid [5,6]. Parietal cells in the stomach secrete intrinsic factor, which binds free B12 for transportation and absorption in the terminal ileum [5,6]. B12 is a water-soluble vitamin stored in the liver but has an increased risk of depletion with small intestinal bacterial overgrowth and surgical alterations of the stomach and small bowel [5,6]. Deficiency can also be found in patients with Crohn or Celiac disease [5,6]. Medications linked to B12 deficiency include metformin, angiotensin-converting enzyme inhibitors, H2 blockers, proton pump inhibitors, and colchicine [5,6]. Clinically, B12 deficiency causes megaloblastic anemia, peripheral neuropathy, and neuropsychiatric manifestations [10].

3.3.5. Vitamin B1

Vitamin B1 (thiamine) is involved in digestion, carbohydrate metabolism, and electrolyte regulation within nerves and muscle cells [10]. Sources include whole grains, rice, lentils, beans, eggs, beef, pork, and fish [10]. The half-life of vitamin B1 is 9–18 days, and the body does not store levels greater than 30 mg [10]. Risk factors for deficiency include alcoholism, excessive vomiting, small intestinal bacterial overgrowth, and excessive caffeine and tannin (tea) intake [10]. Proton pump inhibitors and chronic usage of H2 blockers are also risk factors [10]. Diets with excess white flour, sugar, and processed carbs are also implicated [1,2,10,21–23]. Vitamin B1 deficiency poses the most significant risk of irreversible damage, especially to neurons, astrocytes, and mitochondria [1–3,10,21–23]. One associated condition is beriberi, divided into two separate syndromes [1–3,10,21–23]. Dry beriberi is associated with adverse neurologic outcomes, whereas wet beriberi has cardiovascular manifestations [1–3,10,21–23]. Additionally, the patient's risk of Wernicke-Korsakoff syndrome increases, characterized by cognitive deficits, oculomotor dysfunction, and cerebellar dysfunction [3,23].

3.3.6. Calcium

Intracellular calcium supports bone mineralization, muscle contraction, nerve conduction, hormone secretion, and cellular metabolism. Extracellular calcium functions as a hormone within parathyroid glands, bone, renal tubules, and thyroid C cells [24]. The greatest risk factor for deficiency is inadequate intake [24]. Most calcium is obtained from dairy products; lesser amounts are found in fish, nuts, and green vegetables [24]. Other risk factors include vitamin D deficiency, chronic kidney disease, end-stage liver disease, and malabsorptive diseases [25]. Patients with higher calcium levels have lower total body fat, suggesting calcium positively affects energy balance [5]. Low-calcium diets may lead to hunger, poor compliance, and reduced weight reduction [5]. Hypocalcemia has harmful consequences in every organ system [24,25]. The most severe effects include heart failure, arrhythmias, seizures, bronchospasm, bone disorders, and neuropsychiatric manifestations [25].

3.3.7. Magnesium

Magnesium ($Mg2+$) is an abundant intracellular mineral within bone and soft tissue [26]. Magnesium is an essential cofactor for enzymatic processes involved in energy metabolism, neurologic, and muscle functions [26]. Hypomagnesemia is prevalent in Western countries due to diets that often contain less than 50% of the daily recommended intake [26]. Sources of $Mg2+$ are whole grains, dark green vegetables, legumes, nuts, and seeds [26]. Additional risk factors include malabsorption syndromes, colon cancer, type 1 diabetes mellitus, impaired renal function, and alcoholism [26]. Magnesium deficiency may exacerbate inflammation associated with obesity through increased cytokine release, circulating C-reactive protein, and oxidative stress [26]. Additionally, magnesium deficiency may be associated with altered gut microbiota [26–29]. These risk factors may contribute to obesity, impaired glucose regulation, and the development of type 2 diabetes [26–29]. Low intracellular concentrations of $Mg2+$ impair the oxidative metabolism of glucose due to its role in activating thiamine diphosphate [26]. Glucose metabolism is thus shunted towards alternate pathways, including those involved in fatty acid biosynthesis, exacerbating the storage of triglycerides within adipocytes and further promoting obesity [26]. Deficiency also increases the risk of cardiovascular disease, dyslipidemia, and metabolic disorders [26]. Magnesium and vitamin D deficiencies are often linked due to magnesium's role in vitamin D synthesis and activation [26]. These deficiencies further exacerbate insulin resistance and the risk of cardiometabolic disorders [26].

3.3.8. Vitamin A

Vitamin A is vital for eye health, night vision, cell growth, and wound healing [10]. Sources included orange-colored fruits and vegetables, dark leafy greens, liver, fish, and dairy [10]. Vitamin A has a low likelihood of deficiency due to its lipophilic properties. Still, the associated risk is exacerbated after malabsorptive procedures such as Roux-en-Y gastric bypass (RYGB) [10]. Clinical consequences include immunosuppression, ocular symptoms, and anemia [30].

3.4. Diets for weight reduction worsen micronutrient deficiencies in individuals with obesity

Individuals with obesity at baseline may have risk factors for malnutrition and micronutrient deficiencies. Then when these patients start strict weight reduction diets, their nutritional deficiencies may further worsen. There are many weight reduction diets, with variable results. Diets associated with large amounts of weight reduction include the low-carbohydrate diet, the intermittent fasting diet, the ketogenic diet, and the very low-calorie diet [41,67]. In comparison, diets associated with smaller amounts of weight reduction include the low-glycemic index diet, the low-fat diet, the portion control/balanced

diet, and the elimination diet [41,67]. The comprehension of the nutritional status of children and adults with obesity is crucial for healthcare providers to identify potential deficiencies before prescribing weight reduction diets [3]. Despite low-carb, intermittent fasting, ketogenic, and very low-calorie diets being superior in assisting with medical weight reduction, they also have the highest risk of exacerbating nutrient deficiencies [31–57]. A summary of the diets and their impact on weight reduction and micronutrient deficiencies are outlined in Table 2.

3.4.1. Low carbohydrate diet

Low carbohydrate diets typically limit carbohydrate intake to 30–120 g per day [47,48]. The lower carbohydrate diets (<60 g per day) often have more significant weight reduction [47,48]. This can be an effective short-term treatment of adolescent obesity [47,48]. In addition, several studies show short-term weight reduction in adults as well [62–65,119–122]. Very low carbohydrate high fat diets (composed of 25% carbohydrates and greater than 35% fat) were very effective in promoting weight reduction in adults, but diet adherence was a major barrier [66,123]. Very low carbohydrate diets that limit carbohydrate intake to 20–30 g per day have yet to be studied in children and are not currently recommended for this population [47,48].

Low-carbohydrate diets are deficient in fiber, folate, potassium, calcium, magnesium, iron, vitamin A, iodine, linoleic acid, and a-linolenic acid [47,49]. As a result, current low-carbohydrate dietary interventions often include micronutrient supplementation to improve nutritional adequacy [47,49]. Studies have shown that a higher carbohydrate allowance of up to 120 g per day can optimize nutritional adequacy and dietary variety [47]. Poor adherence to accompanying micronutrient supplementation introduces a risk of deficiency in these dietary practices [50]. Excessive intake of saturated fat exceeding recommendations on a low-carbohydrate plan increases the risk of cardiovascular disease [47]. Dietary guidelines suggest limiting intake of saturated fat to less than 10% of total energy and replacing saturated fat with polyunsaturated to reduce cardiovascular disease risk [47].

3.4.2. Ketogenic diet

The ketogenic diet is a high-fat, moderate protein, and carbohydrate-restricted diet that has gained popularity for its potential to promote weight reduction [32,51]. In the first two weeks of the diet, individuals typically experience rapid weight reduction due to the depletion of water and glycogen stores [32,51]. Long-term studies must evaluate the efficacy of the ketogenic diet. However, some suggest proper meal timing can help mitigate rapid weight reduction by consuming higher-calorie breakfasts after overnight fasting [52].

Ketogenic diets, without adequate counseling and monitoring, can lead to severe malnutrition [32,51]. The ketogenic diet limits the intake of fortified carbohydrates, leading to limited thiamine intake [32,51]. A case study reported an adolescent with Wernicke's encephalopathy after losing 100 lbs. on a ketogenic diet [51]. The patient required intravenous thiamine infusions due to severe malnutrition from the diet [51]. Additional reported side effects include fatigue, headache, nausea, dizziness, vomiting, constipation, and low exercise tolerance [32,51]. Increased saturated fat intake is associated with increased LDL levels and, thus, cardiovascular risk [53]. Therefore, patients need to be monitored closely on this diet.

Table 2
Summary of weight reduction diets and risk of micronutrient deficiency.

Small Weight Loss & Adequate Micronutrient Intake	Large Weight Loss & High Risk of Micronutrient Deficiency
Portion Control or Balanced Diet	Low Carbohydrate Diet
Low Fat Diet	Intermittent Fasting or Time Restricted Feeding
Low Glycemic Index Diet	Ketogenic Diet
Elimination Diet	Very Low Energy Diet

3.4.3. Very low-calorie diets

Very low-calorie diets (VLCDs) may achieve rapid weight reduction through a restriction of ≤800 kcal per day, typically in the form of meal replacements or food-based diets [54,55,69,70,110–118]. To ensure the preservation of lean body mass, VLCDs are low in carbohydrates, containing less than 50 g/day, and high in quality protein, with a minimum of 0.8–1.5 g/kg body weight per day [54,55]. By inducing a mild state of ketosis, VLCDs help facilitate appetite suppression leading to weight reduction [54,55,71]. In addition to the typical short course of VLCDs, there is also promise for lasting long-term weight reduction effects [68,72]. One study that placed patients with fatty liver disease on an 8 to 12 week-long VLCD found that 68% of patients maintained ≥5% weight reduction 9 months after the study [68].

With very low-calorie diets, a high calorie-free fluid intake of at least 2 L per day, daily multivitamins, and mineral supplementation is recommended to prevent the risk of malnutrition [54,55]. Typically, a VLCD is prescribed as a short-term approach for 8–12 weeks under the guidance of a trained healthcare professional [54,55]. The long-term effects of VLCDs are not well understood in children, and they may be associated with risks such as electrolyte imbalances, gallstone formation, and cardiac complications [54,55]. Adult populations may benefit from very-low-carbohydrate ketogenic diets (VLCKD), as demonstrated by a greater reduction in weight, triglycerides, and diastolic blood pressure over one year when compared to a low-fat diet (less than 30% energy from fat) [56].

3.4.4. Intermittent fasting

Intermittent fasting is a dietary approach that involves the consumption of foods within a restricted time window to generate fasting periods within the body [32,47,49]. There are additional types of intermittent fasting including alternate day fasting, periodic fasting, and fasting mimicking diet [80,81]. Adult participants demonstrate adequate weight reduction with intermittent fasting [78,79,93–96,98–109]. Despite weight reduction while on a time-restricted eating diet, one study found there was no significant difference in weight reduction when compared to a structured diet of three meals throughout the day [97]. Additionally, long-term maintenance of intermittent fasting needs to be supported [57,73,79,95,103].

Modified alternate-day fasting, a type of intermittent fasting where calories are restricted every other day, is low in fiber, potassium, vitamin A, iron, calcium, iodine, magnesium, iron, linoleic acid, and a-linolenic acid [32,47,49]. Side effects of intermittent fasting include hunger, irritability, and inability to concentrate [32,47,49]. Additionally, adults displayed increased hunger levels at one year of adhering to intermittent fasting compared to adults adhering to low-calorie diets [57].

3.4.5. Low glycemic diet

An example of a low glycemic diet is the Mediterranean diet, which consists of fruits, vegetables, whole grain cereals, legumes, nuts, seeds, olive oil, fish, lean meats, and dairy products [32]. Diet adherence and proper portion control are associated with lower odds of overweight or obesity in adolescents [32]. In contrast, several studies have demonstrated minimal changes in weight and BMI with the low glycemic diet [37–44,83,84,91]. One meta-analysis found that low glycemic diets when compared to high glycemic diets did not have a significant impact on weight reduction in patients with a BMI ≥25, but it did show more weight reduction in patients with a BMI ≥30 [82]. Another adult study found that a combined low glycemic and fat restricted diet did produce significant weight reduction results after 3 months [92].

Research has shown that a low glycemic diet is associated with higher intakes of micronutrients than other diets. However, it may have unfavorable higher intakes of saturated fat and sodium [31]. The diversity of this diet has also shown evidence for reduced rates of osteoporosis in women and frailty and sarcopenia in older adults [33,34]. Compared to a low-fat diet, a low glycemic diet did show promising

reductions in long-term cardiovascular risk in adults [35,36].

3.4.6. Low-fat diet

Low-fat diets involve limiting fat intake to less than 30% of total energy intake, and this diet may not significantly impact children's BMI [41,45]. In fact, follow-up studies conducted over 2, 4, and 6 years of children on a low-fat diet experienced higher weight gain than those with an average to high fat intake [41,45]. Additionally, several studies have demonstrated that low-fat diets have minimal to no impact on weight reduction and can even increase the risk of weight regain [41, 45]. However, some studies have shown low glycemic and low-fat diets may decrease the BMI z-score [41,44]. Also, one clinical trial of 609 adults with overweight/obesity did demonstrate weight reduction after 12 months on a healthy low-fat diet, but these results were not statistically different when compared to a healthy low-carb diet [85]. Despite these varying results, comparing different diets for children and adults showed no significant difference in BMI and body composition from baseline and between the other diet groups [41,44,46]. There is little evidence that suggests significant micronutrient deficiencies from a low-fat diet, and it seems to vary on the dietary content of each individual person [124]. In fact, some studies report an increased intake of several micronutrients such as vitamin C and B vitamins [125].

3.4.7. Portion control diet

Portion control diets have been shown to be most useful for prevention of obesity in children [74]. Studies on portion control diets in children have demonstrated long-term improvement in BMI, but they did not show significant weight reduction when compared with standard nutritional counseling [75,76]. There are varied results in adult studies with some demonstrating decreased weight gain to others demonstrating adequate weight reduction with portion control diets [86–90]. One study with children compared the efficacy of an elimination diet that limited high energy-dense foods compared to a diet of increasing healthy food intake [77]. The study observed a greater reduction in BMI in the increased healthy food group compared to the elimination diet group [77]. One of the explanations for this difference is that food restriction may result in children eating in the absence of hunger [77]. While portion control diets may vary in their approaches, three studies examining the impact of portion control on the meal quality of children and adolescents reported that participants consumed foods with an overall higher nutrient content compared to their previous meal habits [126–129]. Conversely, evidence examining adults found higher consumption of fruits and vegetables at the expense of total meal energy, dairy, and grains [127,130].

3.5. Bariatric procedure considerations

Deficiencies are commonly identified during the evaluation before bariatric surgery [1,19,58–61]. Up to 85.5% of patients had at least one vitamin or mineral deficiency before bariatric surgery, while 50% displayed a deficiency of greater than 2 micronutrients [3,21–23]. Studies on nutritional status before bariatric surgery found 70–90% of participants were vitamin D deficient [1–3,5,10,21,23]). Additional micronutrient deficiencies encountered were iron (30–60%), vitamin C (50%), vitamin B9 (10–60%), calcium (10–48%), vitamin B6 (24%), vitamin B1 (20–34%), vitamin B12 (10–20%), vitamin A (2–17%), zinc (7–28%), copper (<5%), selenium (<5%), and chromium [1–3,5,10,21,23]. The mean body mass index (BMI) for patients with obesity who had micronutrient deficiency was 43 kg/m^2 [3,21–23]. Iron deficiency was more common in women [1–3,5,10,21,23].

However, clinicians are not actively screening for micronutrient deficiencies. Less than 25% of patients have their nutritional status assessed before bariatric surgery [2,6,10,22]. Patients should meet with an experienced registered dietician (RD) for a comprehensive nutritional assessment before bariatric surgery to identify and correct micronutrient deficiencies [3,22]. Compliance with taking multivitamins in patients before bariatric surgery is low. Only 10% of women and 18% of men followed standard vitamin supplementation recommendations 2–3 weeks before surgery [3,22]. Formulating a personalized plan for the patient may increase adherence to pre-surgery protocols and decrease complications following the procedure.

Understanding the consequences of preoperative malnutrition is vital for providing adequate counseling and preventing deleterious surgical outcomes [1,2,10,21–23]. Nutrient deficiency is worsened following bariatric procedures and will not quickly normalize with routine preventative supplementation [3,21–23]. Many of these patients require significant micronutrient replacement to correct these abnormalities [3,21–23]. Researchers have provided recommendations [Table 3] for routine micronutrient and vitamin screening before surgery [2,6,10,22]. Appropriate screening and supplementing will help prevent health consequences after bariatric surgery.

4. Conclusion

We reviewed micronutrient deficiencies found in patients with obesity. The paradox between obesity and malnutrition goes beyond high-caloric, low-nutrient dietary practices. Physiologic changes in response to increased adiposity and inadequate intake increase this population's risk of adverse health effects. Malnutrition affects the individual and contributes to worse public health outcomes. Our recommendations for adequate screening and awareness of malnutrition are not exclusive to individuals undergoing bariatric procedures and can improve the health outcomes of any patient with obesity. Clinicians should advise on proper nutrition and be aware of diets that worsen deficiencies. Further studies should continue to explore the relationship between malnutrition, obesity, and the clinical consequences of micronutrient deficiency.

Ethical review

Topics of ethical considerations have been understood and agreed with by all the authors in this review. This submission represents original work with properly cited sources. No human test subjects or volunteers were used for this study. Editors were not involved in decisions of papers they authored, or were written by Editor family members or colleagues, or that relate to products or services in which Editors had or have an interest. Sara Karjoo MD (Editor) was not involved in the peer-review and acceptance/rejection of this submission. Responsibility for the editorial process for this article was delegated to a non-author Editor or Associate Editor.

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Authorship contributions

The concept of the submission and supervision was by SK. Investigation through the literature was performed by SK, MB, JM, and RC. MB and JM wrote the first draft. RC wrote the second draft. MB, JM, RC, and

Table 3
Pre-bariatric procedure screening recommendations.

Routine Screening	Optional Screening	
Iron studies	Vitamin A	Red blood cell folate
Vitamin B12 (Cobalamin)	Vitamin E	Homocysteine
Vitamin B9 (Folic Acid)	Vitamin K	Methylmalonic Acid
Vitamin D 25-OH	Zinc	Vitamin B6 (Pyridoxine)
Vitamin B1 (Thiamine)	Copper	Parathyroid hormone (PTH)
	Selenium	Vitamin B2 (Riboflavin)

SK all reviewed, edited, and approved the final submission and publication.

Disclosure statement

None.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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