Diet and Men's Sexual Health

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ABSTRACT

Introduction: Male sexual dysfunctions are more prevalent with aging. With increasing evidence about the impact of various diets on chronic diseases, there is a growing interest in establishing an association between various diets and men's health and sexual dysfunction.

Aim: To review the current literature examining diet and dietary patterns and male sexual health.

Methods: A thorough literature search of peer-reviewed publications on the association between diet and dietary patterns and male sexual health (erectile dysfunction, hypogonadism, and infertility) was carried using the online PubMed database from 1977 through 2017 with the keywords *diet, dietary patterns, erectile dysfunction, male hypogonadism, low testosterone,* and *male infertility.*

Main Outcome Measures: Summary of significant findings on erectile function, androgen levels, and semen analysis parameters in relation to diets or dietary patterns.

Results: Thirteen studies on diet and erectile dysfunction and 15 studies on diet and testosterone levels were reviewed, including observational studies and randomized controlled trials. Thirteen studies analyzing the relation between diet and semen analysis parameters were reviewed but consisted of only cross-sectional and case-control studies.

Conclusion: Evidence exists demonstrating the association between various diets and men's sexual health. Erectile dysfunction appears to lessen in men adhering to the Mediterranean diet. Obese and overweight men who lose weight through low-fat, low-calorie diets seem to have improvements in their erectile function and testosterone levels. Furthermore, a Western diet is associated with lower semen quality. Future prospective and randomized controlled trials are necessary to establish the benefit of diet and dietary patterns on men's sexual health. **La J, Roberts NH, Yafi FA. Diet and Men's Sexual Health. Sex Med Rev 2018;6:54–68.**

Key Words: Diet; Dietary Patterns; Men's Health; Erectile Dysfunction; Hypogonadism; Infertility

INTRODUCTION

Male sexual dysfunction is defined as a physical or psychological condition preventing a man from achieving sexual satisfaction and encompasses erectile dysfunction (ED), premature ejaculation, loss of libido, and hypogonadism.[1] In this review on the current scientific literature, we assess and summarize the possible roles diets and dietary patterns play in sexual dysfunctions with specific attention to ED and male testosterone in addition to male infertility. Because of the paucity of literature investigating the role of diet in premature ejaculation and libido loss, we omit these two categories of sexual dysfunction from our review.

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Rates of Male Sexual Dysfunctions

As a disorder primarily affecting aging men, ED has a prevalence from 30% to 52% in men at least 40 years old and a crude incidence rate of approximately 26 cases per 1,000 man-years.[2–4] ED is a pervasive disorder affecting more than 20 million American men and is projected to affect 322 million men worldwide by 2025.[5,6] Most studies investigating ED use indices or questionnaires to quantify the presence and severity of ED, with the most popular tool being the five-item International Index of Erectile Function (IIEF-5).[7] Increasing literature has shown that men with healthier lifestyles can have better maintenance of erectile function.[8–12]

Male hypogonadism or testosterone deficiency, a condition believed to affect 5 million American men, is biochemically defined by low serum testosterone with clinical manifestations including psycho-neuro-cognitive deficits, decreased lean body mass, lower bone density, decreased libido, and ED, among others.[13,14] Hypogonadism is another disorder that affects aging men, with almost half of men at least 60 years old with the

Table 1. Health benefits and risks of popular diets

Diet	Benefits	Risks
Western	Relatively affordable and easy to obtain	Increases risk of total mortality, cardiovascular disease, obesity, metabolic syndrome, stroke, chronic kidney disease, and breast, colon, and prostate cancer
Mediterranean	Lower risk of overall mortality, cardiovascular and coronary heart diseases, overall cancer incidence, neurodegenerative diseases, diabetes, and metabolic syndrome	Unknown
Paleolithic	Lowers risk in all-cause and cause-specific mortality, cardiovascular disease, metabolic syndrome, and colorectal adenomas	Unknown
Vegetarian and vegan	Lowers ischemic heart disease and overall cancer mortality; absence of red meat, which is associated with increased risk of total, cardiovascular, and cancer mortality, diabetes, and breast, esophageal, gastric, colorectal, prostate, and bladder cancer	Potential protein and vitamin (calcium, B12, iron, and ω-3 fatty acids) deficiencies

disorder.[15–17] Multiple comorbidities have been associated with low testosterone such as obesity, type 2 diabetes mellitus (T2DM), other elements of the metabolic syndrome, and cardiovascular disease, which are chronic diseases in which lifestyle and dietary interventions have been shown to be beneficial.[18–22]

Although male infertility is not necessarily considered a sexual dysfunction, it causes significant psychosocial and marital stress in affected men.[23] Infertility affects approximately 10% to 15% of couples with an estimated 20% of infertility cases due to male factor alone.[24–27] Because infertility is dependent on the male and female partners, it is difficult to evaluate and assess the incidence and prevalence of male infertility. The gold standard for evaluating male infertility is through semen analysis (sperm count, motility, and morphology), and although these parameters might be normal, other unidentifiable factors could be contributing to infertility. Data from semen analyses have been conflicting but have nonetheless created increasing concern that male infertility might be increasing worldwide.[28–31] Because of changing diets in the past several decades globally, the increase in male infertility has often been linked to lifestyle and dietary factors, among many other modifiable factors.[32–34]

Rates of Obesity and Metabolic Syndrome and the Importance of Diet

Obesity (body mass index $\geq$ 30 kg/m^2) in adults has been increasing in epidemic proportions in the past several decades, with a recent study by the National Health and Nutrition Examination Survey (NHANES) in 2012 estimating a 35% prevalence in American adults.[35] The association between obesity and a multitude of health risks including increased mortality, cardiovascular disease, T2DM, and certain cancers is well established.[36–41]

Similarly, a 2013 NHANES study found that metabolic syndrome affects approximately one in five Americans.[42] Diagnostic criteria for metabolic syndrome consist of larger waist circumference, hypertension, hyperglycemia, and dyslipidemia.[43] Individuals diagnosed with metabolic syndrome have increased risks of cardiovascular disease, T2DM, and all-cause mortality.[44–46]

Lifestyle changes including healthy diets and increase in physical activity have been the mainstay in the prevention and management of obesity and metabolic syndrome.[47,48] Because there is mounting evidence supporting the association of obesity and metabolic syndrome with male sexual function such as hypogonadism, infertility and ED, it can be assumed dietary factors likely play a role in male sexual function.[22,49–51]

POPULAR DIETS

Western Diet

A Western diet is usually rich in red and processed meats, dairy, refined grains, processed and artificial sweets, and salt with minimal intake of fruits, vegetables, fish, and whole grains.[52] The Western diet, because of its predominantly processed nature, is increasingly cheap and relatively easy to obtain because of industrialization and globalization. There is increasingly more evidence validating the health detriments of the Western diet (Table 1). Adherence to a Western diet has been associated with increased risks of total mortality and multiple diseases including cardiovascular diseases, obesity, metabolic syndrome, stroke, chronic kidney disease, and breast, colon, and prostate cancer.[53–60]

Mediterranean Diet

The typical Mediterranean diet consists of fish, monounsaturated fats from olive oil, fruits, vegetables, nuts, legumes, and whole grains. The Mediterranean diet has been gaining increasing popularity during the past few decades given

increasing health benefits demonstrated in randomized controlled trials (RCTs). Most notably, the Prevención con Dieta Mediterránea (PREDIMED) study has shown that the Mediterranean diet is associated not only with lower T2DM incidence, metabolic syndrome prevalence, and lower risks of morbidity and mortality from coronary heart disease and stroke but also with a lower incidence of major cardiovascular events.[61–66] A recent umbrella review of meta-analyses of observational studies and RCTs found that adherence to this diet decreased the risk of overall mortality, cardiovascular diseases, coronary heart disease, myocardial infarction, overall cancer incidence, neurodegenerative diseases, and diabetes.[67] Although the Mediterranean diet has many advantages, it is unknown if it presents any health risks.

Paleolithic Diet

Although the Paleolithic diet, also known as the Paleo diet, is the ancestral hunter-gatherer human diet before the agricultural revolution of the modern era, it has recently been revived because of studies reporting health benefits to its adoption (Table 1). The diet consists of lean meats, plant-based foods, fruits, nuts, and vegetables and restricted consumption of dairy, grains, sugar, and salt. Risk decreases in all-cause and cause-specific mortality, cardiovascular disease, metabolic syndrome, and colorectal adenomas have been associated with the Paleolithic diet in the recent literature, which includes several RCTs.[68–73] Like the Mediterranean diet, health benefits are clear with the Paleo diet, but it is unknown whether there are any health risks with consuming this diet.

Vegetarian and Vegan Diets

The vegetarian diet is characterized by the absence of animal products, especially red meat, which results in lower saturated fat and cholesterol intake. The vegetarian diet also can be subcategorized into pesco-vegetarian (absence of all animal products except fish), lacto-ovo-vegetarian (absence of all animal products except eggs and dairy), ovo-vegetarian (absence of all animal products except eggs), and vegan (absence of all animal products). A multitude of health benefits come with consuming a vegetarian diet (Table 1). The literature, including meta-analyses and systematic reviews, describes lower mortality from ischemic heart disease and lower overall cancer incidence in vegetarians compared with non-vegetarians.[74–77] Studies examining the effect of red or processed meat on health outcomes have demonstrated an increased risk of total mortality, cardiovascular disease mortality, cancer mortality, T2DM, and breast, esophageal, gastric, colorectal, prostate, and bladder cancer.[78–83] Because vegetarians avoid red or processed meat consumption, the vegetarian diet presumably confers decreases in red meat–associated health risks. Vegetarians and vegans can develop protein and vitamin (calcium, B12, iron, ω-3 fatty acids) deficiencies, which can be prevented with careful monitoring of their diets and supplementation.

DIET AND MEN'S HEALTH

Diet and Erectile Dysfunction

Individuals with cardiovascular risks have higher prevalence of ED and the pathophysiology of ED seems to share pathways with atherosclerotic disease.[84–89] Diet has been shown to play an important role in decreasing congestive heart disease risk, so it is sensible to presume diet could play a role in ED.[90,91] The relation between diet and ED has been examined in several studies (Table 2).[92–104]

Several cross-sectional studies have associated greater adherence to the Mediterranean diet or components of a Mediterranean diet (fruit, vegetable, nuts, and monounsaturated fats) to a lower prevalence of ED.[93,96,100,101,103] In a prospective cohort study, men with metabolic syndrome and ED were placed on the Mediterranean diet and had increased IIEF-5 scores after 2 years. In addition, more men who already had ED regained erectile function compared with controls (37% vs 7%; $P = .015$).[94] Another RCT of overweight men randomized to advice on the Mediterranean diet for weight loss reported an increase in the percentage with normal erectile function after 2 years (56% vs 38%; $P = .015$).[95] In a long-term RCT, men with T2DM on a Mediterranean or low-fat diet had overall decreased IIEF-5 scores over a mean follow-up of 8.1 years, with men on the Mediterranean diet having less deterioration of their IIEF-5 scores compared with men on the low-fat diet.[102]

When looking at ED in overweight and obese men, several prospective studies and RCTs have demonstrated that weight loss by low-calorie or low-fat diets improved erectile function as assessed by IIEF-5 score.[97–99,104]

The current literature suggests the Mediterranean diet improves ED in the short term and lessens deterioration of erectile function in the long term. In addition, weight loss in overweight or obese men through lifestyle changes including low-fat, low-calorie diets seems to improve their erectile function.

Diet and Testosterone

To our knowledge, no published literature investigating the effect of diet on male hypogonadism exists; however, many studies have described the relation between diet and male androgen levels (Table 3).[97,104–118] The earliest prospective studies with small samples of predominantly healthy men in the 1980s showed serum total and free testosterone levels decreased or did not change when men followed a short-term low-fat diet.[105,106,108] Similar results were noted more recently in a study by Wang et al[116] in 2005.

Prospective studies have demonstrated that obese men on low-calorie diets had variable increases in serum testosterone, serum free testosterone, serum dihydrotestosterone, and/or SHBG.[97,112,115,118] A small number of RCTs studies have confirmed these findings.[104,111,114] Notably, in a study of overweight or obese men on low-fat diets for 12 weeks and weight maintenance for another 40 weeks, significant increases in

Table 2. Studies of diet or lifestyle changes and effect on erectile function

Study	Design	Sample size, n	Subjects	Intervention	Results
Esposito et al[92]	RCT	110	Obese (BMI $\geq$ 30) men with ED	Detailed advice on weight loss vs control (information)	After 2 y, intervention group had improved IIEF-5 score (13.9 vs 17; $P < .001$); control group had stable IIEF-5 score (13.5 vs 13.6; $P = .89$); intervention group had more men regain erectile function (31% vs 5%; $P = .001$); CRP ($P = .02$) and IL-6 ($P = .03$) decreased more in intervention group
Esposito et al[93]	Case-control	200	Men with or without ED		Men without ED adhered to the MedDiet more than men with ED (5.4 ± 0.5 vs 4.7 ± 0.5; $P < .01$)
Esposito et al[94]	Prospective cohort	65	Men with MetS and ED	MedDiet	Intervention group had higher IIEF-5 score (18.1 ± 4 vs 15.2 ± 3.5; $P < .01$) and increased mean change (3; 95% CI = 0.6–5.2) after 2 y; more men in intervention group regained erectile function (37% vs 7%; $P = .015$) and had greater decrease in CRP (−0.9 ± 0.4 vs −0.1 ± 0.1; $P < .01$)
Esposito et al[95]	RCT	209	Overweight men (BMI > 25)	Detailed advice on diet such as MedDiet and weight loss vs control (information)	Intervention group had more monounsaturated fat and fiber intake and less saturated fat intake ($P = .001$); intervention group had larger number of men with normal erectile function (56% vs 38%; $P = .015$) after 2 y
Giugliano et al[96]	Cross-sectional	555	Men with T2DM		Compared with lowest adherers to MedDiet, highest adherers had lower global ED (51.9% vs 62.0%; $P = 0.01$) and severe ED (16.5% vs 26.5; $P = 0.01$) as measured with IIEF-5
Khoo et al[97]	Prospective cohort	70	Obese men (BMI $\geq$ 30) with or without T2DM	Intervention: low-calorie diet (Kicstart)	After 8 wk, intervention group had greater increases in IIEF-5 score vs control group ($P < .01$); IIEF-5 improvements correlated with weight loss (R = −0.26; $P = .03$) and WC (R = 0.33; $P < .01$)
Wing et al[98]	RCT	306	Overweight and obese men with T2DM	Intensive lifestyle changes with diet and physical activity vs control (information)	After 1 y, intervention group showed significant improvement in IIEF-5 score (17.3 ± 7.6 and 18.6 ± 8.1; $P = .04$) and control group showed no improvements (18.3 ± 7.6 and 18.4 ± 8.0; $P = .06$); more control men without ED developed ED (19% vs 0%; $P = .001$) and no differences between groups in men with ED who showed improvement (18% and 19%; NS)
Khoo et al[99]	RCT with cross-over	31	Obese men with T2DM	LCD vs HP	LCD and HP groups had improved IIEF-5 score after 8 wk ($P < .05$); LCD men switched to HP; IIEF-5 score of 2 groups continued to improve after cross-over ($P < .05$); CRP and IL-6 significantly decreased in HP ($P < .01$) but not in LCD group

(continued)

Table 2. Continued

Study	Design	Sample size, n	Subjects	Intervention	Results
Wang et al[100]	Cross-sectional	1,466	Canadian men with T2DM		Inverse association between ED and fruit and vegetable consumption; 10% risk decrease with each additional daily fruit or vegetable serving (OR = 0.90, 95% CI = 0.82–0.98)
Ramírez et al[101]	Cross-sectional	440	Mediterranean men with CV risk factors		Consumption of nuts (>2 times/wk; OR = 0.41, 95% CI = 0.25–0.67) and vegetables (≥1 time/d; OR = 0.47, 95% CI = 0.28–0.77) was inversely related to ED; alcohol consumption (OR = 1.14, 95% CI = 1.04–1.26) was directly related to ED
Maiorino et al[102]	RCT	106	Men with T2DM	MedDiet vs low-fat diet	IIEF-5 score significantly lower than at baseline with MedDiet (mean difference = −1.22; $P < .001$) and low-fat diet (mean difference = −2.23; $P < .001$); men in low-fat group had greater decrease in IIEF-5 score than men in MedDiet group (between-group difference = −1.16, 95% CI = −2.16 to −0.15); CRP decreased from baseline in MedDiet group (change = 0.31; 95% CI = 0.08–0.62) but did not change in low-fat diet group (change = 0.03; 95% CI = −0.21 to 0.26); mean follow-up = 8.1 y
Chen et al[103]	Cross-sectional	2,584	Chinese men with BPH-associated LUTS		Consumption of vegetable ($P = .001$) and milk or dairy ($P = .001$) correlated with lower IIEF-5 score
Moran et al[104]	RCT	118	Overweight and obese men	High-protein low-fat diet vs high-carbohydrate low-fat diet	High-protein low-fat and high-carbohydrate low-fat groups had significant increase in IIEF total score at 12 wk ($P < .05$) with no difference between groups; no changes seen long term at 52 wk and no changes in IIEF-5 score within or between groups

BMI = body mass index; BPH = benign prostatic hyperplasia; CI = confidence interval; CRP = C-reactive protein; CV = cardiovascular; ED = erectile dysfunction; HP = low fat, high protein, low carbohydrate; IIEF-5 = five-item International Index of Erectile Function; IL-6 = interleukin-6; LCD = low-calorie diet; LUTS = lower urinary tract symptoms; MedDiet = Mediterranean diet; MetS = metabolic syndrome; OR = odds ratio; NS = not significant; RCT = randomized controlled trial; T2DM = type 2 diabetes mellitus; WC = waist circumference.

Table 3. Studies of diet and effect on male hypogonadism

Study	Design	Sample size, n	Subjects	Intervention	Results
Hämäläinen et al[105]	Prospective	30	Healthy men	Low-fat, high-fiber diet	Decrease in serum total T (22.7 ± 1.1 vs 19.3 ± 1.2 nmol/L; $P < .001$) and free T (0.23 ± 0.01 vs 0.20 ± 0.01 nmol/L; $P < .01$) when switching to low-fat, high-fiber diets for 6 wk; return to baseline T values when switching back to previous diets
Rosenthal et al[106]	Prospective	21	Men with CV disease or risks	High-complex carbohydrate, high-fiber, low-fat diet	After 21 d of high-complex carbohydrate, high-fiber, low-fat diet, serum total T levels did not change from baseline
Howie and Shultz[107]	Cross-sectional	30	SDA vegetarians and non-vegetarians		SDA vegetarians had 27% lower levels of serum T vs non-vegetarians ($P = .02$); no significant difference in serum DHT levels between groups
Reed et al[108]	Prospective	6	Healthy men	High-fat diet with switch to low-fat diet	Men fed high-fat diet for 2 wk had decreased SHBG (21.7 ± 6.8 vs 19.5 ± 7.6 nmol/L; $P < .02$); when switching to low-fat diet for additional 2 wk, SHBG increased (19.5 ± 7.6 vs 24.8 ± 7.9 nmol/L; $P < .01$) and serum free T decreased (573 ± 177 vs 453 ± 138 pmol/L; $P < .05$)
Bélanger et al[109]	Cross-sectional	29	Healthy men		No difference in serum total T, DHT, and DHEA between omnivores and vegetarians; vegetarian men had higher levels of SHBG ($P < .01$)
Key et al[110]	Cross-sectional	108	Healthy men		no difference in serum total T and calculated free T between omnivores and vegans; vegan men had higher levels of SHBG (39.5 vs 32.0 nmol/L; $P = .001$)
Dorgan et al[111]	RCT with cross-over	43	Healthy men	High-fat, low-fiber diet vs low-fat, high-fiber diet	Men on high-fat, low-fiber diet only had significantly higher SHBG-bound T vs men on low-fat, high-fiber diet (1.2, 95% CI = 0.1–2.4)
Tymchuk et al[112]	Prospective	27	Obese men	Low-fat, high-fiber diet with exercise	Low-fat, high-fiber diet with exercise increased SHBG (18 ± 2 to 25 ± 3 nmol/L; $P < .01$) after 3 wk
Longcope et al[113]	Cross-sectional	1,552	Normal men		SHBG directly correlated with fiber intake ($R = 0.05$) and inversely correlated with protein ($R = -0.05$) and animal fat ($R = -0.05$, $P = .05$) intake
Kaukua et al[114]	RCT	38	Obese (BMI $\geq$ 35) men	VLED vs control (regular diet)	Intervention group (VLED with weight loss and then weight maintenance) showed increased SHBG and T vs control group ($P < .05$)

(continued)

Table 3. Continued

Study	Design	Sample size, n	Subjects	Intervention	Results
Niskanen et al[115]	Prospective	58	Obese men with mets	VLCD	After 9 wk of VLCD, increases in SHBG (27.6 ± 11.9 vs 48.1 ± 23.5 nmol/L; $P < .001$), free T (185 ± 66 vs 208 ± 70 pmol/L; $P = .002$), and total T ($P < .001$).; after 12 mo of weight maintenance, SHBG ($P < .001$) and total T ($P < .001$) still higher than at baseline but lower than during VLCD; free T remained high ($P = .002$)
Wang et al[116]	Prospective	39	Healthy men	Low-fat, high-fiber diet	Low-fat, high-fiber diet for 8 wk decreased total T (16.9 ± 0.9 vs 14.9 ± 0.6 nmol/L; $P = .0001$), free T (0.20 ± 0.01 vs 0.18 ± 0.01 nmol/L; $P = .0045$), DHT (1.87 ± 0.12 vs 1.70 ± 0.1 nmol/L; $P = .0053$), and SHBG (46.4 ± 3.7 vs 42.6 ± 2.9 nmol/L; $P = .0105$) vs baseline
Dorgan et al[117]	RCT	354	Boys with high LDL-C	Low-fat diet vs control (regular diet)	No difference in serum total T, free T, DHT, androstenedione, and SHBG between intervention (low-fat diet) and usual-care groups; median follow-up = 7.1 y
Khoo et al[97]	Prospective cohort	70	Obese men (BMI ≥ 30) with or without T2DM	LCD	Men on LCD without T2DM had increased serum total T (3.0 ± 7.7 vs −2.6 ± 3.8 nmol/L; $P < .01$) and men on LCD with T2DM had no changes vs control group after 8 wk; plasma SHBG was significantly higher in the 2 intervention groups vs control group ($P < .01$); calculated free T decreased in control and LCD non-diabetic groups but increased in LCD diabetic group
Schulte et al[118]	Prospective	13	Obese men	VLCD	After 3 mo of VLCD, men had increased total serum T (6.97 vs 13.21 nmol/L; $P = .001$), free T (0.20 vs 0.27; $P = .13$), and SHBG (22.11 vs 42.12; $P = .001$)
Moran et al[104]	RCT	118	Overweight and obese men	High-protein, low-fat diet vs high-carbohydrate, low-fat diet	No differential effect of diet, high-protein, low-fat vs high-carbohydrate, low-fat, on serum total T, free T, and SHBG; after 12 wk of diet intervention, increases in serum total T (0.68 ± 0.30 nmol/L; $P = .037$) and SHBG (4.6 ± 0.5 nmol/L; $P < .001$) but not free T; after additional 40 wk of weight maintenance, increase in serum total T (2.0 ± 0.4 nmol/L; $P < .001$), free T (30.5 ± 7.1 pmol/L; $P = .002$) and SHBG (5.0 ± 0.7 nmol/L; $P < .001$)

BMI = body mass index; CV = cardiovascular; DHEA = dehydroepiandrosterone; DHT = dihydrotestosterone; LCD = low-calorie diet; LDL-C = low-density lipoprotein cholesterol; MetS = metabolic syndrome; RCT = randomized controlled trial; SDA = Seventh Day Adventist; T = testosterone; T2DM = type 2 diabetes mellitus; VLCD = very low-calorie diet; VLED = very-low-energy diet.

serum total and free testosterone and SHBG were noted. However, the investigators found no difference in effects between high-protein, low-fat diets and high-carbohydrate, low-fat diets.[104]

Only sparse studies have associated specific diet patterns with male testosterone levels. Two cross-sectional studies showed that men consuming vegetarian or vegan diets had higher serum levels of SHBG, but there were no associations between these diets and serum total and free testosterone levels.[109,110] In contrast, a cross-sectional study of a small sample of vegetarian Seventh Day Adventists found that vegetarians had significantly lower testosterone levels than non-vegetarians.[107]

Increases in testosterone levels seem to occur in overweight or obese men on low-fat or low-calorie diets, presumably from resulting weight loss and, thus, less aromatization of testosterone. However, the studies presented in this review seem to suggest healthy men placed on low-fat diets have resulting lower testosterone levels. The mechanism is not clear but the investigators have suggested this phenomenon might be due to lower cholesterol levels available for steroidogenesis. Many of the prospective studies had small samples, relatively short-term follow-up, or did not investigate a specific type of dietary pattern. Further large, prospective studies are needed to assess the impact of specific diets on male testosterone levels in the long term.

Diet and Male Infertility

Human sperm has been shown to generate reactive oxidative species in physiologic amounts; thus, oral antioxidants have been associated with improved sperm quality.[119–123] We review the current literature detailing the association of diets or dietary patterns on male semen quality, which includes cross-sectional and case-control studies (Table 4).[124–136]

Cross-sectional studies have described different associations between semen parameters and different food groups. In two separate cross-sectional studies composed of 800 men, higher saturated fat intake was inversely related to total sperm count and concentration, whereas another study suggested that trans fatty acid intake was inversely related to total sperm count.[126,129,133] In addition, several cross-sectional studies associated higher consumption of processed meat products with lower sperm count, percentage of progressively motile sperm, ejaculate volume, and percentage of normal sperm morphology, whereas cheese intake was inversely associated with lower sperm concentration.[130–132]

When looking at specific diet patterns in cross-sectional studies, the association varies slightly for semen parameters. A study by Gaskins et al[127] showed that adherence to a "prudent" diet pattern (fish, chicken, fruit, vegetables, and whole grains) was associated with a significantly larger percentage of progressively motile sperm, whereas no association between adherence to the Western diet (red and processed meat, butter, dairy, refined grains, and sweets) and semen parameters was found. In contrast, Liu et al[135] reported lower sperm concentration and percentage of normal sperm morphology with greater adherence to the Western diet, but failed to find any relation between a healthier diet and semen quality. In a cross-sectional study, life-long lacto-ovo vegetarians had significantly lower sperm concentrations and total motility compared with non-vegetarian men.[136]

The few existing case-control studies comparing men with normal and abnormal semen parameters parallel the previous cross-sectional studies. Mendiola et al[124] evaluated the intake of various foods in men with normospermia vs oligo-asthenoteratospermia and reported that men with oligo-asthenoteratospermia tended to consume less lettuce, tomatoes, and fruit and more dairy and processed meats. Similarly, Eslamian et al[128,134] looked at men with asthenospermia in two separate studies and reported a lower risk of asthenospermia with higher intake of fruit, vegetables, and adherence to the prudent diet pattern and a higher risk with higher intake of processed meat and adherence to the Western diet pattern.

These studies are limited by their observational nature, but all seem to imply that intake of healthier, prudent diets or food groups is associated with better semen quality, whereas the opposite is seen with consumption of the Western diet or its food groups. Better designed, prospective studies with large samples sizes are needed to evaluate any causative effects of diet and dietary patterns on male fertility.

LIMITATIONS

There are many facets to the human diet. This is evident in the diversity of the reviewed studies because they measured diet and dietary patterns in different ways, which make comparing the results fairly difficult. Although some studies investigated specific food groups, no studies looked at whether organic vs non-organic foods contribute to the dietary effects of male sexual health.

In addition, a limitation of many observational dietary studies is recall bias because the studies relied on questionnaires. Recruited male subjects in some studies had different comorbidities such as T2DM or metabolic syndrome, thus introducing additional variables that can alter outcomes.

A handful of the reviewed studies assessed the effect of dietary fat on male sexual health. Traditionally, fat, in general, was considered detrimental to human health especially with respect to cardiovascular health. However, increasing evidence supports subcategorizing certain fats (eg, monounsaturated) as health benefits compared with others (eg, trans fatty acids).[137] Therefore, studies using low-fat diets as interventions could be excluding "good" fats that could benefit sexual dysfunction.

FUTURE PERSPECTIVES

Increasingly more research is looking at the effect of holistic dietary patterns on the prevention and management of health

Table 4. Studies of diet and effect of male infertility

Study	Design	Sample size, n	Subjects	Results
Mendiola et al[124]	Case-control	61	Men with normal and abnormal sperm	Men with abnormal sperm (oligo/terato) had lower intake of lettuce and tomatoes (OR = 0.4, 95% CI = 0.2–0.8) and fruits (OR = 0.3, 95% CI = 0.1–0.6) and high intake of dairy (OR = 3.1, 95% CI = 1.1–8.5) and processed meat (OR = 2.6, 95% CI = 1.2–5.4)
Vujkovic et al[125]	Cross-sectional	161	Men in subfertile couples	Adherence to "health conscious" dietary pattern (fruits, vegetables, fish, whole grains, fish and seafood) associated with lower DFI score (P for trend = .05); adherence to "traditional Dutch" dietary pattern (meat, potatoes, whole grains) associated with higher sperm conc (P for trend = .01)
Attaman et al[126]	Cross-sectional	99	Men in fertility clinic	Higher total fat intake inversely related to total sperm count (P for trend = .01) and conc (P for trend = .01) driven by saturated fat intake; higher ω-3 intake positively associated with favorable morphology (P for trend = .02)
Gaskins et al[127]	Cross-sectional	188	Healthy men	Adherence to more Western pattern (red and processed meat, butter, dairy, refined grains, sweets) unrelated to sperm count, conc, motility, or morphology; adherence to "prudent pattern" (fish, chicken, fruit, vegetables, whole grains) associated with larger percentage of progressively motile sperm but unrelated to sperm count, conc, or morphology
Eslamian et al[128]	Case-control	241	Men with normal sperm and asthenozoospermia	Highest tertile of total fruit and vegetable (OR = 0.88, 95% CI = 0.73–0.96), poultry (OR = 0.53, 95% CI = 0.36–0.79), and seafood (OR = 0.91, 95% CI = 0.77–0.95) associated with lower risk of asthenozoospermia, specifically oranges, dark green vegetables, tomatoes; highest tertile of processed meat (OR = 2.03, 95% CI = 1.70–2.26) and sweets (OR = 2.05, 95% CI = 1.09 to 2.26) associated with higher risk of asthenozoospermia
Jensen et al[129]	Cross-sectional	701	Healthy men	Higher saturated fat intake inversely related to total sperm count (P for trend = .02) and conc (P for trend = .04); higher monounsaturated fat intake associated with lower normal morphology percentage (P for trend = .05) and higher ω-3 fatty acid intake associated with large semen volume (P for trend = .05)
Afeiche et al[130]	Cross-sectional	155	Men in subfertile couples	When comparing highest- with lowest-quartile consumers, low-fat milk intake was related to 30% higher sperm conc (95% CI = 1–51) and 8.7% higher progressive motility (95% CI = 3.0–14.4); cheese intake related to 53.2% lower sperm conc (95% CI = 9.7–75.7)
Afeiche et al[131]	Cross-sectional	189	Healthy men	Processed red meat intake inversely related to total sperm count (P for trend = .01), total progressive motility (P for trend = .01), and ejaculate volume (P for trend < .001); meat intake not associated with sperm conc or morphology
Afeiche et al[132]	Cross-sectional	155	Men in subfertile couples	Processed meat intake inversely related to sperm morphology (P for trend = .02), whereas fish intake related to higher sperm count (P for trend = .005) and normal sperm morphology percentage (P for trend = .01)

(continued)

Table 4. Continued

Study	Design	Sample size, n	Subjects	Results
Chavarro et al[133]	Cross-sectional	209	Healthy men	Trans fatty acid intake inversely related to total sperm count (P for trend = .03) and cholesterol intake inversely related to ejaculate volume (P for trend = .04)
Eslamian et al[134]	Case-control	342	Men with normal sperm and asthenozoospermia	Highest tertile of adherence to "prudent pattern" (vegetables, tomatoes, dish, poultry, seafood, fruit, whole grains) associated with lower asthenozoospermia risk (OR = 0.46, 95% CI = 0.25–0.97; P for trend = .003); highest tertile of adherence to Western pattern (red and processed meats, organ meats, sweets, soft drinks, refined grains, high-fat dairy) associated with higher asthenozoospermia risk (OR = 2.86, 95% CI = 1.83–2.97; P for trend = .038)
Liu et al[135]	Cross-sectional	7,282	Healthy men	highest quartile of adherence to Western diet had lower sperm conc (P for trend < .001) and lower normal sperm morphology percentage (P for trend < .001); highest quartile of intake of very sweet snacks and sugar-sweetened drinks also had lower sperm conc (P for trend < .001) and lower normal sperm morphology percentage (P for trend = .002); high-carbohydrate diet related to higher abnormal total (P for trend = .012) and progressive sperm motility (P for trend = .025); high sodium diet related to higher abnormal sperm morphology (P for trend = .035); healthy diet adherence not associated with any parameters
Orzylowska et al[136]	Cross-sectional	474	Lacto-ovo vegetarian, vegan, and non-vegetarian men	Lacto-ovo vegetarians had significantly lower sperm conc (50.7 ± 7.4 vs 69.6 ± 3.2 mil/ml; P < .05) and percentage of total motility (33.2 ± 3.8 vs 58.2 ± 1.0%; P < .05) than non-vegetarians

CI = confidence interval; conc = concentration; DFI = DNA fragmentation index; OR = odds ratio.

conditions. This also is the case with male sexual dysfunction and infertility. The studies summarized in this review present a compelling association between various diets and dietary patterns and men's sexual health, with the strongest evidence seen from the groups examining ED. However, more prospective studies and RCTs are needed to establish a substantial cause and effect of dietary patterns.

Inflammatory Markers

Much attention has been focused on inflammatory markers such as interleukin (IL)-6, IL-8, IL-18, and C-reactive protein, which have been linked to endothelial dysfunction and future cardiovascular events.[138,139] Endothelial dysfunction and ED share a similar pathophysiology such as the nitric oxide pathway and ED has been positively associated with endothelial dysfunction and the aforementioned proinflammatory markers, especially in obese men.[8,87,140–142] Hence, it is believed lifestyle modifications and interventions resolve ED likely through lowering of inflammatory markers and subsequent improvement of endothelial function.[11] In three of the studies evaluated in this review, the investigators demonstrated significant decreases in IL-6 and C-reactive protein in men who underwent dietary interventions, which was in conjunction with significant improvements in IIEF-5 scores.[92,94,99] Because most ED research measures erectile function primarily by subjective indices, inflammatory markers such as IL-6 and C-reactive protein could be objective tools to quantify improvements in future studies on ED in relation to diets.

Testosterone deficiency in older men is associated with cardiovascular disease, T2DM, dyslipidemia, and metabolic syndrome, which are conditions that have been associated with systemic inflammation.[143–147] There appears to be an inverse relation between androgens and inflammation, and some data have suggested that hypogonadism might actually result in systemic inflammation and ensuing cardiovascular and metabolic events.[148–150] Studies including RCTs, particularly of men with metabolic syndrome, have suggested increasing testosterone through testosterone replacement could decrease inflammatory markers.[151,152]

CONCLUSIONS

The current literature demonstrates compelling associations between diet and dietary patterns and men's sexual health. The

most convincing research concerns diet and ED in which the Mediterranean diet and Mediterranean food groups appear to improve or at least diminish its progression, whereas weight loss, through low-fat, low-calorie diets, alleviates ED in obese and overweight men. In addition, weight loss with low-fat, low-calorie diets in obese and overweight men might improve testosterone levels, and testosterone levels might decrease in healthy men on these diets. There also appears to be a negative relation between the Western diet and semen quality but studies are purely observational at this point. Establishing a definitive role for diet in the possible prevention and management of male sexual dysfunction will require further large-scale prospective studies.

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