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NUTRITION AND ENDOTHELIAL FUNCTION: A REVIEW OF KETOGENIC AND PLANT-BASED DIETARY INTERVENTIONS

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ABSTRACT

Introduction: Endothelial function is crucial for maintaining vascular homeostasis by regulating vasomotor tone, inflammation, and immune responses. Impairment of endothelial function is an early indicator of cardiovascular disease (CVD). Given the increasing prevalence of CVD, identifying modifiable risk factors, including dietary patterns, is becoming increasingly important.
Aim of the study: This review compares the effects of ketogenic and plant-based diets on endothelial function, highlighting molecular mechanisms and clinical implications.
Methodology: Literature was searched in PubMed, Scopus, and Web of Science (June–September 2025) using the keywords endothelium, ketogenic diet, vegetarian diet, atherosclerosis, and oxidative stress. Studies published in English between 2010 and 2025 were analyzed.
Results: Ketogenic diets, although effective for weight loss, can lead to dehydration, micronutrient deficiencies, and increased endothelial inflammation due to low antioxidant intake. In contrast, plant-based diets are rich in fiber and antioxidants and low in trans fats, supporting endothelial integrity and reducing inflammation, though they may lack adequate EPA and DHA.

KEYWORDS

Endothelium, Ketogenic Diet, Vegetarian Diet, Atherosclerosis, Oxidative Stress

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Introduction

The vascular endothelium is a single layer of cells that forms the direct point of contact between blood vessels and the blood flowing through them. For years, endothelial cells (ECs) were considered a mechanical barrier separating blood components from the outer layers of blood vessels. Current knowledge suggests that under physiological conditions, ECs perform numerous functions, including normal nutrient and metabolite exchange, angiogenesis, and antiplatelet properties. They also regulate vascular tone through the production of mediators such as nitric oxide and other vasodilators, such as prostacyclin and endothelin, and by moderating local angiotensin II activity. Due to their antiadhesive properties, they underlie free blood flow. Recently, many researchers have also become interested in the important role of the endothelium in innate and adaptive immunity [1]. Endothelial damage is increasingly recognized as a key factor in the development of numerous cardiovascular diseases [2][3]. A deeper understanding of the role of the vascular endothelium in the cascade of pathomechanisms underlying these diseases may offer new opportunities for their prevention and treatment [3]. “The hallmarks” of endothelial dysfunction, as described by Hunt and Jurd, include: impaired vascular integrity with exposed subendothelium, increased expression of adhesion molecules promoting transendothelial leukocyte trafficking, prothrombotic propensity, inflammation, and expression of major histocompatibility complex (MHC) II on endothelial cells, enabling them to function as antigen-presenting cells (APCs)” [3][4]. Endothelial dysfunction is also associated with the following pathological conditions: diabetic vascular disease, stroke (mouse studies), hypertension, inflammation, atherosclerosis, and oxidative stress (rat studies), inflammatory bowel disease, heart failure, renal failure, and Alzheimer's disease (human studies) [5]. The condition of the cardiovascular system depends, among other things, on lifestyle. In the context of the impact of nutrition on body functions, it seems important to analyze the impact of restrictive dietary models, which have become popular in recent years. Energy availability, as well as increasing or eliminating a specific nutrient, can affect the functioning of the entire organism, modify its response, and modulate homeostasis [2][6]. In this review there are present the importance of a properly functioning vascular endothelium and the consequences of its damage, as well as potential benefits and side effects of the ketogenic and vegetarian diet (KD) in the context of endothelial cells.

Methodology

All available studies examining the relationship between dietary patterns and endothelial function were reviewed. Literature searches were conducted in PubMed, Scopus, and Web of Science databases between June and September 2025 using the keywords: endothelium, ketogenic diet, vegetarian diet, atherosclerosis, oxidative stress. Included were original research papers, narrative and systematic reviews, and meta-analyses published between 2010 and 2025 in English. Studies focusing solely on animal models, case reports, conference abstracts, or those unrelated to dietary effects on endothelial health were excluded. After screening and removal of duplicates, 38 studies were included. The data were analyzed to summarize current knowledge and identify gaps in understanding the effects of ketogenic and plant-based diets on endothelial physiology.

Results

Endothelial dysfunction

For understanding the processes leading to endothelial dysfunction, it is necessary to look at the immune-inflammatory axis in the endothelium. In the context of atherogenic stimulation, endothelial cells, which in physiological conditions are a quiescent and anti-inflammatory state, undergo activation, which is the initial step in endothelial dysfunction [3][7]. According to Hunt and Jurd, the core features of endothelial dysfunction include loss of vascular integrity leading to exposure of the subendothelial matrix, increased expression of adhesion molecules that facilitate leukocyte adhesion and transendothelial migration, a shift towards a prothrombotic phenotype, a local inflammatory response and, importantly, the expression of major histocompatibility complex (MHC) class II molecules on ECs, which allows them to function as atypical antigen-presenting cells (APCs) [3][6][8].

Endothelium in the atherosclerotic plaque formation

Endothelial dysfunction is one of the earliest events that set the stage for atherosclerosis [3]. Plaques do not appear randomly; they tend to form where blood flow is irregular - at arterial branches and curves, where shear stress on the vessel wall is low [6]. In these areas, endothelial cells become activated and start expressing adhesion molecules such as VCAM-1, ICAM-1, and E-selectin. They also release cytokines like TNF-α and IL-6, which attract circulating leukocytes that move beneath the endothelial layer. Inside the intima, monocytes turn into macrophages and begin taking up oxidized LDL particles containing apolipoprotein B (apoB). These cells gradually fill with lipids, forming foam cells - the classic sign of early atherosclerotic change [9]. As the process continues, vascular smooth muscle cells migrate from the media into the intima, contributing to plaque buildup. They form a fibrous cap that gives the lesion some stability, but over time, inflammatory cells release matrix metalloproteinases (MMPs) that weaken this cap. Eventually, the plaque may rupture, triggering acute coronary events or stroke [10]. Over time, vascular smooth muscle cells (VSMCs) migrate from the tunica media to the intima, where they contribute to plaque growth and stabilize the lesion by forming a fibrous cap. As a result, this cap is susceptible to degradation by matrix metalloproteinases (MMPs) released by inflammatory cells, which can ultimately lead to plaque instability and rupture - a critical event responsible for acute coronary syndromes and stroke [10].

Endothelial cells in inflammatory cascade

Endothelial cells are important regulators of inflammation. When activated by different stimuli, they release several cytokines, for example interleukin-1β (IL-1β) through NLRP3 inflammasome activation, as well as IL-6, IL-8, MCP-1, and TNF-α [11][12]. These molecules attract immune cells and increase the expression of adhesion proteins like P- and E-selectins, VCAM-1, and ICAM-1 [13]. This leads to leukocyte attachment to the endothelium, their rolling along the vessel wall, and later migration through the endothelial layer [14][15]. A similar process occurs with monocytes, which are key in the development of atherosclerosis. Their adhesion depends on interactions between integrins and VCAM-1 and is strengthened by chemokine signaling [16]. After crossing the endothelium, monocytes move into the intima, where they turn into macrophages, often of the proinflammatory M1 type. These macrophages take up oxidized LDL particles and become foam cells, which are involved in the formation of early atherosclerotic lesions [17]. In this way, interactions between endothelial cells and leukocytes link vascular inflammation with immune activation and endothelial dysfunction. On the luminal surface, the endothelial glycocalyx -a thin, gel-like layer -also plays an important role by regulating permeability, leukocyte adhesion, and mechanical signaling [18].

Importance of endothelial glycocalyx and diet-related risk factors for endothelium damage

The endothelial glycocalyx (EG), composed mainly of proteoglycans like syndecans and glypican-1 bound to glycosaminoglycans (GAGs) (e.g., heparan sulfate, chondroitin sulfate, hyaluronic acid), interacts with plasma components, acting as a molecular sieve and reservoir for signaling molecules [18]. It maintains vascular integrity and has anti-inflammatory, antithrombotic, and antioxidant functions. Pathological stimuli - such as hyperglycemia, high sodium intake, disturbed flow and cytokines like TNF-α - promote EG degradation via enzymatic and oxidative mechanisms [19]. This increases endothelial permeability, facilitating lipoprotein and leukocyte infiltration [18]. Breakdown products like hyaluronic acid fragments and syndecan ectodomains act as Damage Associated Molecular Patterns (DAMPs), activating Toll-like receptors (TLRs) and enhancing inflammation. Elevated levels of syndecan-1, endocan, and hyaluronan have been linked to endothelial dysfunction in conditions such as atherosclerosis, sepsis, heart failure, and diabetic vasculopathy. These biomarkers reflect the extent of endothelial damage and may help predict cardiovascular risk. In summary, the endothelial glycocalyx plays a key role in vascular homeostasis and immune regulation. Its degradation initiates endothelial dysfunction and promotes a shift toward a proinflammatory, activated state. By increasing permeability and leukocyte infiltration, it drives a self-perpetuating cycle of vascular inflammation. Therefore, preserving EG integrity represents a promising strategy to counteract endothelial dysfunction and cardiovascular disease progression [3][18][19].

Markers of endothelial dysfunction

Elevated levels of markers of endothelial dysfunction are associated with atherosclerotic cardiovascular disease (ASCVD), heart failure, and cardiovascular mortality. Imaging techniques such as intravital microscopy and sidestream darkfield (SDF) imaging allow for indirect assessment of glycocalyx thickness. The most direct method for assessing endothelial function is coronary angiography, which assesses the vascular response to acetylcholine, but it is limited by its invasiveness. A noninvasive and widely used alternative is flow-mediated dilation (FMD), which uses ultrasound to measure brachial artery dilation during reactive hyperemia, reflecting nitric oxide (NO) release. Early detection of endothelial dysfunction is important. However, the clinical use of biomarkers and imaging is limited by a lack of validation in large populations, a limited number of longitudinal and randomized studies, and poor standardization. Large biobanks and prospective cohort studies can help establish endothelial biomarkers as predictors of cardiovascular events. Adherence to international guidelines on FMD is essential to ensure reliable measurement and clinical implementation [3].

Importance of diet for endothelial health

In recent years, people's eating habits have changed significantly. Research indicates that the typical modern diet is increasingly based on highly processed foods, rich in refined sugar, trans fats, and artificial additives, while lacking in fresh fruits, vegetables, and other nutritious foods. This dietary pattern is strongly associated with unfavorable changes in the gut microbiota, disrupting the balance between commensal and pathogenic species. This dysbiosis is believed to lead to impaired mucosal immunity, low-grade systemic inflammation, and increased intestinal permeability, all of which are associated with the pathogenesis of chronic vascular inflammation and endothelial dysfunction. Moreover, high consumption of processed foods is associated with the activation of inflammatory pathways, suppression of regulatory immune responses, and changes in lipid metabolism, which further exacerbate vascular damage and promote the development of atherosclerosis [19][20]. In parallel, recent years have seen a growing awareness of and interest in dietary alternatives that differ from the typical Western diet. A growing number of people are adopting diets that deliberately limit or eliminate specific food groups. Among these, the ketogenic diet and the vegan diet have become the most popular [20].

Ketogenic diet

The ketogenic diet is based on a high fat intake, moderate protein intake, and very low carbohydrate intake to induce a state of nutritional ketosis, in which ketone bodies are the primary energy substrate. Although this diet has gained recognition for its potential metabolic benefits, concerns have been raised about its long-term effects on the cardiovascular system, particularly with regard to lipid profile and endothelial function. In the early stages of the diet, dehydration occurs due to increased nitrogen and water excretion [21]). Euglycemia is maintained in the body by increased gluconeogenesis. This process is regulated by increased secretion of glucocorticoids, glucagon, and growth hormone, which promote proteolysis, particularly in skeletal muscle

tissue. Long-term use of a ketogenic diet has been found to result in significant reductions in lean body mass and muscle protein stores [21][22]. Another concern with the ketogenic diet is its potential to exacerbate oxidative stress. Mitochondrial dysfunction, which may result from long-term exposure to a high-fat diet, increases the production of reactive oxygen species (ROS), especially superoxide anions. High dietary fat intake, in particular, may intensify free radical reactions and lipid peroxidation processes. ROS initiate lipid peroxidation, damaging cell membranes and proteins, contributing to long-term vascular damage [21][22][23]. The coexistence of oxidative stress and chronic inflammation is associated with the pathogenesis of many diseases, including hypertension. Oxidized lipids, especially oxidized LDL (oxLDL), play a key role in endothelial dysfunction and atherosclerosis and are also associated with age-related diseases such as chronic kidney disease and cardiovascular disease [24]. These observations emphasize the importance of maintaining lipid homeostasis, particularly in aging populations, to attenuate the progression of these diseases and reduce the associated mortality risk. Excessive ketone accumulation can lower serum pH and bicarbonate levels, inducing ketoacidosis [25]. Case reports have described ketoacidosis during high-ketogenic diets, particularly in individuals with metabolic disorders [26]. Acidosis increases bone resorption, potentially linking KD to vascular calcification and cardiovascular disease risk. Ketone bodies modulate appetite hormones by reducing ghrelin and neuropeptide Y levels, leading to reduced food intake [22]. However, the restrictive nature of this diet can lead to significant micronutrient deficiencies, with the most common being deficiencies of B vitamins, vitamin D, and electrolytes [21][25]. In addition to nutritional disorders, a number of other adverse effects associated with the ketogenic diet have been reported. Gastrointestinal disturbances - including nausea, vomiting, constipation, diarrhea, and gastroesophageal reflux - are relatively common, especially in the initial phase of adaptation. Metabolic complications, such as hyperlipidemia, electrolyte imbalance, and cardiac arrhythmias, have also been documented. Souad Nasser and colleagues report that elevated blood ketone body levels can predispose endothelial cells to a proinflammatory state. Elevated cytokine and chemokine levels promote the recruitment and adhesion of monocytes to the endothelium, initiating or exacerbating vascular inflammation. This cascade of events contributes to endothelial damage. The effect of ketone bodies on the vascular endothelium appears to be concentration-dependent. In pathological conditions such as diabetic ketoacidosis, blood ketone concentrations can rise to as high as 25 mmol/L. Physiological ketosis induced by calorie restriction, fasting, or a ketogenic diet typically leads to ketone levels of around 7 mmol/L in generally healthy individuals [23]. Despite the potential benefits of ketogenic diets, including short-term weight loss, these dietary patterns are not without risk. They can cause adverse effects, including episodes of ketoacidosis, which pose a significant health risk, particularly in individuals with impaired metabolic control [22][27]. Patients with type 1 diabetes are particularly susceptible to the harmful effects of chronic vascular inflammation and recurrent episodes of ketoacidosis. Such events are associated with vascular complications, including cerebral edema. At the same time, ketone bodies, especially beta-hydroxybutyrate (BHB), may protect blood vessels from age-related damage. As aging increases the risk of cardiovascular disease, finding ways to maintain a healthy circulatory system becomes increasingly important. Calorie restriction, which increases endogenous ketone production, has been shown to delay endothelial aging. Recent studies have identified a novel mechanism by which BHB may exert its antiaging effects: through direct interaction with heterogeneous nuclear ribonucleoprotein A1 (hnRNP A1). This binding stabilizes the mRNA of octamer-binding transcription factor 4A (Oct4A), a key transcription factor involved in cell pluripotency and longevity, thereby increasing its expression. Oct4A appears to play a crucial role in maintaining vascular cell quiescence and counteracting age-related decline in their function. These findings suggest that targeting the Oct4A pathway with BHB or structurally related analogs may represent a promising therapeutic strategy for preventing or delaying endothelial cell aging and prevention [21].

Vegetarian diet

The opposite of the ketogenic diet is the plant-based vegetarian diet. It focuses on fruits, vegetables, legumes, nuts, and grains, while limiting or eliminating animal products. There are several varieties of plant-based diets, including vegan, lacto-vegetarian, ovo-vegetarian, and pescatarian, which vary in the degree of exclusion of animal products. Each of these eating styles has its own health benefits, as well as ethical and environmental concerns. Choosing the right diet often depends on individual values, health needs, and taste [28][29]. Attention should also be paid to a whole food plant-based diet (WFPBD), which is based on good quality fruits, vegetables, whole grains, legumes and natural soy products, while eliminating animal products, highly processed foods, fat and refined sugar [29]. Despite the difficulties in separating the importance of diet from other environmental factors such as air pollution, physical activity and the use of stimulants, WFPBD

has been found to have a beneficial effect on the prevention of cardiovascular disease, diabetes and cancer [29][30]. Randomized trials have proven that Healthy Plant-Based Diets should be recommended as an option to support cardiovascular health [31]. Vegan diets are usually rich in fiber, beta-carotene, vitamins C and K and vitamin B9, which allows them to be called high-quality diets [32]. They are also often rich in vegetable oils and therefore in ω-6 fatty acids [33]. However, there is a risk of insufficient intake of vitamin B12, vitamin D, calcium, selenium, zinc, and iodine, as well as lower availability of ω-3 fatty acids (eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)), which can lead to energy, nutrient, and micronutrient deficiencies. Therefore, people following a vegan diet should be advised to supplement these nutrients [34]. For example, in a vegan diet, long-chain ω-3 fatty acids, which are critical for the functioning of the retina, brain, and cell membranes, can only be consumed in the form of α-linolenic acid (ALA), so it is recommended that vegans take an algae-based DHA supplement in addition to their regular intake of ALA sources [35]. In vegan diets, saturated fats mainly come from tropical oils such as coconut and palm oil, as well as other plant products. high in fat [36]. Diets low in trans and saturated fats are associated with reduced inflammation in the body and improved endothelial function [37]. Trans fats negatively affect lipid metabolism, inflammation, and endothelial function. Therefore, dietary patterns that limit or exclude these fats, such as vegetarian diets, may offer cardiovascular benefits [37]. Moreover, Tuso, Stoll, and Li emphasize that diets rich in fruits, vegetables, legumes, whole grains, and nuts provide high levels of antioxidant polyphenols, which protect endothelial cells from oxidative stress and LDL oxidation. These protective effects reduce inflammation and help maintain the bioavailability of nitric oxide (NO), a key factor in maintaining vascular tone and preventing endothelial dysfunction. Furthermore, lower consumption of animal products reduces the production of trimethylamine N-oxide (TMAO), a proatherogenic compound. Lipid profiles are also improved, blood pressure is lowered, and insulin sensitivity is increased, which is beneficial for endothelial integrity. From a mechanistic perspective, a plant-based diet reduces monocyte adhesion and endothelial activation - key early steps in atherogenesis. Evidence suggests that such dietary modifications can not only halt but potentially reverse the progression of coronary artery disease (CAD). Overall, switching to a plant-based diet appears to be an effective, evidence-based strategy for protecting endothelial health and preventing coronary heart disease [38]. The quality and nutritional composition of plant-based diets are key factors influencing their anti-inflammatory and endothelial-protective potential [34].

Discussion and Conclusions

The findings show that the nutritional composition of diets plays an important role in modulating endothelial function and vascular health. A ketogenic helps with weight loss and improves certain metabolic parameters; however, it is often associated with dehydration, loss of muscle mass, vitamin and electrolyte deficiencies, and, in some cases, even ketoacidosis. Its low antioxidant content may lead to chronic endothelial inflammation and promote atherosclerotic plaque formation, which increases the risk of cardiovascular diseases development. In contrast, plant-based diets are rich in fiber and antioxidants and low in trans fats, which supports endothelial integrity, reduces inflammation, and lowers the risk of coronary heart disease. Nevertheless, due to potential deficiencies in long-chain omega-3 fatty acids (EPA and DHA), it requires dietary planning and supplementation. Moreover, plant-based diets are likely more effective for long-term endothelial protection because of their anti-inflammatory and antioxidant properties, whereas poorly balanced ketogenic diets may have harmful effects on blood vessels despite short-term benefits. Further research is necessary to better understand the mechanisms linking diet and endothelial physiology. This will enable development of more precise dietary recommendations for the prevention and early treatment of cardiovascular disease.

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