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Review Article

Diet Influence on Inflammation Sabira Sultana, Muhammad Akram

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Abstract

The growing role of chronic inflammation in the major degenerative diseases of modern society has encouraged research in the influence of dietary patterns and inflammatory nutrition. In modern society, there is an increasing number of atherosclerotic diseases due to major lifestyle changes. Increasing body weight, a healthy diet, a sedentary lifestyle, leads to vascular disintegration which increases the risk of thrombotic atherosclerosis. Not surprisingly, Type II diabetes, obesity, and metabolic syndrome have become increasingly common public health problems. A complete diet plan reduces inflammation and heart disease. Low vegetable and fruit intake, as well as physical inactivity, are reported to be the top ten causes of death in developed countries.Because several carcinomas, cardiovascular diseases, and diabetes are combined to make up about 70% of all deaths in the U.S., and high rates of obesity and metabolic syndrome are likely to increase the incidence and spread of many serious illnesses soon. The review contains evidence of epidemiologic and clinical evidence regarding the impact of diet on inflammation. We did literature research using Science direct, Elsevier, Springer Link (Springer), Pub Med and Google Scholar. The search included keywords "diet", "Inflammation", "High fat diet", which are guided by the key words' "inflammation" and "diet". We have included in vitro, in vivo experimental studies and randomized controlled trials.

Key words: diet; inflammation; high fat diet; cytokine; nutrients; metabolic disease; degenerative diseases.

Introduction

Recently, inflammation hasbeen recognized as an important part of the pathophysiology of industrialized societies' age-related ailment and chronic illnesses, including cardiovascular disease, Alzheimer's disease, and many types of cancer. As a bio-chemical marker of inflammation, certain clinical trials and observational studies have used high sensitivity to active protein C (HS-CRP) because it is comparatively stable and easy easily measurable. HS-CRP elevation more reliably predicts future development of diabetes mellitus and hypertension than body mass index (BMI). The effect of a dietary regimen on systemic inflammation should be considered in every attempt to develop a diet for optimum health. Part of this effect, resultingfrom visceral adiposity, because of inflammatory effects of abdominal obesity [1].

It is possible to reduce the risk of heart disease with diet. Adequate consumption of omega-3 fatty acids, avoiding saturated fats and saturated foods, nutritious foods, nuts, vegetables, nuts, whole grains. Whole grains are great dietary strategies. By adopting this method, it can reduce the risk of inflammation. Dietary patterns of refined sugars, carbohydrates and fatty acids are rich in fruits and natural antioxidants, whole grains and vegetables and low omega-3 fatty acids can cause the immune mechanism to start working,most likely because of excessive pro-inflammatory cytokine production associated with decreased anti-inflammatory cytokine production. In particular, the whole diet involves in the reduction of inflammation causing the metabolic syndrome. In order to fight chronic diseases, it is important to select carbohydrates, proteins and fats, which are compatible with daily basis activity and avoid smoking. Inflammation is warmed up by Western dietary habits, while cautious dietary patterns cool it down. Many doctors still consider a low-fat diet (about 30 percent of total calories) to be a safe alternative to all cardiac disease. The unintended effect of diet with low fat can lower high-density lipoprotein cholesterol (HDL-C) and increase level of triglyceride, which increase the body's presentations of insulin resistance syndrome, also known as metabolic syndrome.

Some dietary supplements can help prevent cardiovascular disease [1] increase the intake of Omega-3fatty acid foods in fish or plants 2) replace unsaturated hydrogenated fats instead of saturated fats; and 3) eat foods that are rich in vegetables, nuts, fruits and the whole grains and then lower with refined grains. Diet effect onthe diseases of coronary artery can be mediated by number of biologic ways other than serum lipids, low inflammation, oxidative stress, hypertension, insulin sensitivity, endothelial dysfunction and thrombotic tendency [3]. The objective of the study is to review diet influence on inflammation.

Macronutrients and Inflammation

Oxidative stress and inflammatory responses may be produced by macronutrient consumption. In normal individuals, carbohydrate ingestion is associated with an increase generation of superoxide in leukocytes and mononuclear cells, and with elevated levels and the activity of nuclear factor-

aB (NF-aB), a document that records the activity of at least 125 genes. Two other inflammatory factors, which use protein-1 (AP-1) and Egr-1, are also involved in increasing glucose uptake, the first regulating matrix metalloproteinase secretion and the second regulating tissue factor transcription and plasminogen activator i -inhibitor-1. It has also been shown that a mixed diet from a fast-food chain activates NF-βB, which is associated with the generation of mononuclear cell reactive oxygen species (ROS). Interestingly, superoxide is a catalyst for at least two major inflammatory agents, NF-egB and AP-1, respectively. These results are consistent with previous studies showing an increased ROS production and circulating levels of inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-alpha), interleukin 6 (IL-6), and interleukin 18 (IL). -6), after oral or glucose injection challenges, in both normal studies and patients with type 2 diabetes (IL-18) (Reference).

As shown by the increase in the strength of adhesion molecules VCAM-1 (vascular cell adhesion molecule-1) and ICAM-1 (intercellular adhesion molecule-1) in combination with an increase in plasma concentrations of IL-6 and TNF -alpha, high fat Eating in normal studies causes endothelial function. In addition, circulating levels of IL-18, an anti-inflammatory cytokine that should be involved in plaque use, and the corresponding reduction of adiponectin used, are proteins derived from insulin-sensitizing protein, anti-inflammatory and antiatherogenic properties, which can enhance food intake the same high fat [3].

Effect of Influenced Mediterranean Diet on Inflammation in Crohn's Patients

Inflammation is important for the body's response but chronic inflammation, such as Crohn's disease causes illness, so reducing inflammation will produce significant health benefits, and another way to achieve this through diet. Foods that fight inflammation and use these foods. We look at the development of inflammation and intestinal microbiota. We have produced proven biomarkers, a C-protein and Inflammation micronucleus assay with the effect of a transcriptomic method.

One would hope to find dysbiosis of the gastrointestinal microbiota because Crohn's disease found in the intestinal tract. There is a strong link between diet, intestinal microbiota, genetics and health. In recent decades, the role of germs has played a key role in understanding human health and disease. The prodimenentinhabitant of human gut are six bacterial phylaconsisting of more than 1,000 organisms. Although previously thought to be a static body, it has now emerged that in response to external stimuli such as food, the level of bacterial phyla, and the type of intestine that continues to change. There is also divergence from what is known to be a 'natural'proportion of bacterial phyla in different diseases. Whether the cause of the disease is a change to microbiotais currently unclear (Reference)

Data showed that the known biomarkers of inflammation could be reduced by our six-week diet. However, observed major alternation in gene expression using transcriptomics. The combined effect of minor variation in multiple genesseemed to have a significant effect while no single gene stood out. Data has also shown that our diet has normalizing the microbiota. The study showed Mediterranean-inspired diet improved the health of Crohn's disease affected people. Inflammation markers greatly reduced in study participants and normalize microbiota.

Inflammation and Metabolic Disease

One process whereby an increase in fiber intake in the diet improves metabolic health. This can reduce inflammation and endotoxemia. A review of NHNES data for 1999-2000 reported a negative correlation between higher CRP risk and dietary fiber intake, defined by the American Heart Association as serum concentration> 3.0 mg / L, and a more direct correlation between dietary intake fat and risk of high serum CRP, a potent inflammatory cytokine. In the third and fourth major food quartiles identified as a diet of 13.3-19.5 and 19.5 g / day, respectively, dietary fiber protection effects were observed, which met with a decrease of 28 g / day recommended by the Dietary Guidelines for Americans 2015-2020. The upper quartile of total fat consumption consumed more than 35.1 grams per day. Although the analysis has been around for two decades, research is still in its infancy as fiber consumption has remained the same since 1999-2014. The average fiber consumption reported in 1999-2000 NHANES data was 6.5 g / 1000 kcal compared to 8 g / 1000 kcal in the 2013-2014 NHANES data. This is just an example of the saturated fat and dietary fiber anti-inflammatory effects. As there were no changes in CRP levels when effects of the Mediterranean diet were assessed by treatment in a group of healthy students of university, which might be due to the difficulty in measuring CRP in healthy individuals.

The association between the use of Mediterranean dietary patterns and endotoxemia has been investigated in a prospective cohort study. The authors claim that plasma LPS saturation (measured by ELISA) was adversely affected by Mediterranean dietary patterns and fruit consumption and legumes in adults with atrial fibrillation. LDL cholesterol plasma and negative cardiovascular consequences also have a favorable correlation in LPS plasma concentrations. The study provides an important report on the relationship between the Mediterranean diet and the development of cardio-metabolism through weight loss. To assess whether these links are causal, multidisciplinary intervention trials should be performed. It is also important that ELISA be considered with the same skepticism as the LAL for measuring the concentration of plasma LPS.SCFAs and butyrates have regulatory effects and are known for their ability to disrupt the progression of metabolic syndrome. SCFAs have the ability to regulate the activity of leukocytes in areas of inflammation and production of cytokines such as IL-2, TNF, IL-6, and IL-10, chemokines and eicosanoids. In vitro, butyrate appears to have a greater effect on leukocyte effects than acetate and on the overall anti-inflammatory effect. This may be one way in which butyrate inhibits the development of immune dysfunction. Another murine study reported the ability of butyrate to inhibit insulin resistance in obese mice fed a high-fat diet of 5% wt: wt butyrate. Other murine studies have suggested that the addition of a probiotic that produces sodium butyrate and butyrate, prevents the progression of non-alcoholic fatty liver to nonalcoholic steato hepatitis (Reference).

There are many clinical studies investigating the outcome of the Mediterranean diet on metabolic syndrome. One meta-analysis, report of 8 studies reported a positive effect on the Mediterranean diet. Consumption of the Mediterranean diet reduced waist circumference, increased serum HDL cholesterol, decreased serum Triglycerides, lowered diastolic and systolic blood pressure, and increased blood glucose levels in participants. While shown to improve metabolic health through a Mediterranean dietary pattern, intervention studies that examined the metabolic endotoxemia [5] pathway.

Inflammatory Strength of Food and Risk of Heart Disease

The connection between diet and cardiovascular disease is well recognized. The previously mentioned Mediterranean food ishealthy, and reducesthe CVD risk –that is associated with a33% of reduction in CVS-related mortality [6].

High-Fat Diet-Induced Inflammation

Some of the new features of cancer have been shown to be tumor-promoting inflammation and immune system avoidance. Macrophages, granulocytes and mast cells fasten the development of inflammation-mediated cancer by different cytokines and are inhibited by T cells Myeloid-based suppressor cells are associated with inflammation and cancer that contribute to the suppression of many immune activors and T cells. The onset of prostate cancer can also be induced by inflammation. Macrophage cells participates in regeneration and tissue inflammation. Recently, activated macrophages (M2) reported to have pro-tumor functions. Prostate cancer is associated with anorexia nervosa. High-fat diets include chronic inflammation and obesity. Epidemiologic studies have reported that a high-fat diet is associated with the development and progression of bladder cancer. Researchreported high fat diet also causes tumor progression by adipose-secretory cytokines inmice and within the xenograft mouse model of bladder cancer cell lines. HFD has, however, been stated not to induce tumor progression in a xenograft pattern of tumor tissue taken by a patient using immunodeficient mices. These findings indicate that high fat diet can speed prostate cancer tumor progression by interactions with immune responses like different cytokines.

Although High fat diet raises serum pro - inflammatory cytokines and encourages the development of prostate cancer in mice, whether the growth in the tumor was due to these cytokines remained unclear (Reference).

The research involves high-fiber diet and celebracoxib to indigenous immuno-competent prostate carcinogenic model mice. Development of tumors was tested for both the weight of the tumor and stain Ki67 and flow cytometry was evaluated at twenty-two weeks of age in local immune cells. Cytokines linked to the growth of the tumor and changes in tumor formation, as well as local immune cells, were evaluated in mice following inhibition of cytokine signals. High fat diet stimulated tumor growth and increased suppressor cell fraction derived from the myeloid and the ratio of M2 / M1 macrophages to the mouse model. Celecoxib-suppressed growth in mices suppressing celecoxib and both local Myeloid-derived suppressor cells MDSC and macrophage ratio of M2/M1. HFD-induced growth of the tumor and phosphorylated tumor STAT3 (pSTAT3) positive cells were associated with IL6 secreted from the prostatic macrophages. Suppressed tumor growth and Myeloid-suppressor cell mdSCs cells and cell fragments in HFD-infected mice decreased due to anti-IL6 receptor antibody administration. In a variety of prostatectomy, the number of healthy cells on CD11b was significantly higher than in patients with prostate cancer. HFD increased Myeloid-suppressor cell MDSC cell growth and accelerated the growth of prostate cancer by signing IL6 / pSTAT3 in mice. High-fat diets increase Myeloid-taken suppressor cells and accelerate the growth of cervical cancer by signing IL6 / pSTAT3 in mice. This machine can be present in obese patients with prostate cancer. IL6 joint inflammation is a treatment for bladder cancer [7].

The world's epidemic of obesity is related to chronic low-grade inflammation and secondary comorbidity, primarily influenced by global lifestyle and dietary shifts. Different nutritional strategies for obesity treatment and related metabolic disorders are proposed. The taste of virgin coconut oil, antioxidants and vitamins were nice (VCO). However, VCO contains a significant quantity of saturated fatty acids and is associated with many secondary diseases when eating this fat. In rats fed with ahigh-fat diet, we assess the impact of VCO supplementation on biochemical, infectious and oxidative stress parameters (HFD). The animals have been supplemented by VCO for 30 days after feeding on a high-fat diet for 12 weeks. High fat foods and VCO intake community showed weight gain, low-cholesterol levels of lipoprotein, and aminotransferase, aspartate and ALT levels. These results were complemented by increased lipid profile of hepatic lipid and fat deposition in the liver. High expression of alpha (TNF-α) tumor necrosis factor (TDF-α) in adipose tissue was observed in the HFD+VCO community. These findings show that HFD-related VCO induced major metabolic changes, adipose inflammation and deposition of lipids in rats [8].

Dietary fat intake is one of the major natural factors that contribute to obesity. In mice, a process depending on the in-situ stimulation of inflammation combines the consumption of leptin and insulin anorexigenic signals in thehypothalamus. Since inflammatory signal transduction may contribute to apoptotic transmission pathways. The effect of high fat intake on apoptosis induction of hypothalamic cells was assessed. It was demonstrated that dietary fat intake induces neuronal apoptosis and decreases synaptic input in the arcuate core and side hypertrophy. The effect is based on diet intake and not on calorie consumption, as pair feeding is not enough to minimize apoptotic markers. The TLR4 receptor, protecting cells against more apoptotic signals. TLR4 has a two-fold function in dietary inflammations. On one hand, it activates pro-inflammatory paths which play a major role in building leptin and insulin resistance and, in turn, regulate apoptotic activity and prevent further injury [9]. Consumption of Moraes JC Fructose from added sugar is considered to be the cause of alcohol-free liver. Research has been done to test whether fructose combined with a high-fat diet creates fatty liver, and to determine whether this depends on Fructokinase, a key enzyme for fructose metabolism. Obesity and specific steatosis have been observed in wild mice with a type of fructokinase knockout and there is no evidence of liver inflammation in a high-fat diet compared to a low-fat diet. While acute hepatic steatosis develops in wild-type mice with high fat and high sucrose intake. Low-grade inflammation and fibrosis, elevated CD68, tumor necrosis factor alpha, attractive monocyte chemo protein-attractive 1, alpha - actin muscle smoothness, and collagen I and TIMP1 expression. No changes were observed in fructokinase knockout mice. On the other hand, high-fat sucrose can produce steato hepatitis. Protection in fructokinase knockout mice has been shown to play an important role in fructose in the development of steato hepatitis. Studies show an important role for fructose in fatty liver and nonalcoholic steato hepatitis [10].

High-fat diets (HFD) promote inflammation in systemic organs such as the hypothalamus. This causes obesity and diabetes. The visceral organs are connected to the central nervous system by the vagus nerve. Ghrelin peptide transmits hunger signals to the hypothalamus through the vagal afferent nerve. The inflammatory response in the vagal aferent neurons and hypothalamus in mice after one day of HFD diet was investigated. The number of microglia non-ganglion dosage and hypothalamus is increased. In addition, a single day high-fat diet stimulates Toll-like receptor 4 expression in colon goblet cells and upregulates mRNA expression of pro-inflammatory biomarkers Iba1, Emr1, Il6and Tnfcy in the nodose ganglion and hypothalamus. Lower administration of celiac and ghrelin vagotomy reduced HFD-induced inflammation in the muscles. The HFD diet stimulated the inflammatory process in the gut, there is no ganglion dose, and thereafter in the hypothalamus within 24 hours. The results suggest that associated vagal sensations may transmit inflammatory symptoms from the gut to the hypothalamus by not using the dose ganglion, and that ghrelin may protect against high-fat diets caused by inflammation [11].

Excess sodium use is also associated with an increased risk of heart disease. In recent years, other studies have also shown that high-salt diets (HSDs) can activate Th17 cells and autoimmune diseases. Studies have been performed to evaluate the effects of NaCl-containing diet on colonic mucosa in a stable and inflammatory environment. Studies have shown that the detection of HSD by mice stimulated the intestinal response and production of IL-23, neutrophils and increased the number of cells producing IL-17 3 cell innate lymphoid (ILC3) in the colon. HSD diet induces Colitis which may be oral administration of dextran sodium sulfate or 2,4,6-trinitrobenzenesulfonic acid and the effect is related to the increasing number of RORγt + CD4 + T cells and neutrophils in the colon. Therefore, the results showed that the detection of HSD promoted inflammation of the colon and included chemotherapy in colitis in mice in a way that relied on the production of IL-17 most likely by ILC3 and Th17 cells [12].

Fructose-Enriched Diet Induces Inflammation

Increase fructose consumption especially by worrying about sugary drinks with health problems, as the development of metabolic syndrome (MetS) is a major risk factor for cardiovascular disease and type 2 diabetes. Fructose in use contributes to weight gain, impaired glucose tolerance, visceral adiposity, insulin resistance, dyslipidemia and hypertriglyceridemia, in both animals and humans. Obesity and childhood obesity increased by 47.1 percent between 1980 and 2013. Visceral incontinence is a hallmark of metabolic syndrome, and adipose tissue is considered not just an energy store, but an active endocrine organ that secretes different hormones and adipokines. There are many obesity studies showing obesity associated with chronic inflammation, characterized by elevated levels of cytokines that begin to swell in adipose tissue and blood circulation, which recur after weight loss. The expression of IL-1β genes in adipose tissue is associated with a cycle in the hip. IL-1β and TNFα formulated in adipose tissue promote insulin resistance associated with obesity and type II diabetes. Prolonged administration of IL-1β induces insulin resistance in adipose tissue. In addition, obese mice lacking TNFα genetically protected from deficiency caused by obesity in insulin signaling. Nuclear factor-κB (NF-κB) plays a key role in the documented activation of pro-inflammatory genes including IL-1β and TNFα. In several cells, it contains NF-κB1 (p50) / RelA (p65) heterodimer and remains secreted in the cytoplasm inactive by the I VerB-blocking protein that secretes the local nuclear signal of small organs. NF-κB activation, by pro-inflammatory cytokines, pressure, free radicals, viruses and bacterial agents, reduces phosphorylation and violation of VerB protein, leading to the transfer of NF-κB to -nucleus and genetic function.

Research stated that oxidative stress in adipose tissues involves ~~in~~ insulin resistance and metabolic syndrome. Active oxygen species (ROS) are involved in the activation of NF-κB mediation by the release of inhibitory subunit I VerB from NF-κB.These days, afructose containing diet associatedwith mal functioning of endocrine included the up-regulation of inflammatory markers. In addition, it has been reported that fructose produces a pro-oxidative effect and alters the expression of antioxidant enzymes in mice. The intake of refined food enrich in fructose replaces nutritious foods in children. Meta-analyses and clinical trials showed fructose over intakecauses the development of obesity in children. In addition, gender differences in the metabolism of obese adolescents are suggested to begin in childhood [13].

Conclusion and Recommendations

Dietary patterns of high sugar, starch refining, fatty acids, fruits, vegetables, and whole grains have affected the formation of the reproductive system, with excess production of highly invading cytokines associates with decrease production of the anti-inflammatory cytokines. Inequality leads to the formation of inflammatory markers. This causes endothelial dysfunction at vascular level, and increases risk of metabolic syndrome and cardiovascular in potential individuals. A complete diet plan reduces inflammation and heart disease. Low vegetable and fruit intake, as well as physical inactivity, are reported to be the top ten causes of death in develop countries. Because several carcinomas, cardiovascular diseases, and diabetes are combined to make up about 70% of all deaths in the U.S., and high rates of obesity and metabolic syndrome are likely to increase the incidence and spread of many serious illnesses soon. Adoption of a healthy lifestyle may reduce the risk of chronic illness. Therefore, choosing healthy sources of fats, protein and carbohydrates, to exercise regularly and avoid smoking, so it is important to fight chronic diseases. This is especially important for those with a high risk, such as obesity, type II diabetes and metabolic syndrome, and they are failing to lose weight.

Conflict of Interest

The authors have no conflict of interest.

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