Participation Of Banana Intolerance in Atherosclerosis Review Of the Literature

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Received: February 16, 2020; Accepted: March 16, 2020; Published: March 31, 2020

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Abstract

The main cause of atherosclerosis is not completely understood. Besides the classical risk factors, food intolerance and its microbiota promote inflammation. IgG antibodies against antigens and microbial genomes have been detected in atherosclerotic lesions.

It has been suggested that fruits despite their benefits in terms of nutrients and anti-oxidative properties may also act as “enemies” provoking diseases through immunity pathways. The current production of fruits involves the use of conservatives, insecticides, fungicides, herbicides, acaricides, surface disinfectants etc. hostile to human body.

Banana contains microorganisms which may use our “ecosystem” to build colonial biofilms called plaques in order to survive. Their metabolites create acidic environment resulting in illnesses and even death.

The primary outcome of this computerized search was that there is a direct and indirect association between banana intolerance and arteriosclerosis. There is limited information about fruit antigens and microbiota and their interactions with the host. Biofilms and immunity complexes could link banana with atherosclerosis. The banana contains nutrients good for us and for our intrinsic “habitants” and microflora which interacts and modulates the host microbiota. Plaques are reconstructed by living “microbes” fighting for home and food.

Extensive studies on the biology and “dietology” of diseases should be conducted to further clarify the “gaps” of this issue.

Key words: banana intolerance, inflammation, atheroma, coronary artery disease, oxidative stress, microbiota, biofilm formation, infection, antigens

Introduction

The predisposing risk factors of atherosclerosis (smoking, genetic disorders of lipid and lipoprotein metabolism, environmental factors, obesity, hypertension, hyperlipidemia, diabetes mellitus, increased plasma homocysteine, ageing) have been recognized[1-9]. However, their pathogenic mechanisms cannot be completely linked to the root-aetiology of atherogenesis. In various studies there were confounding agents and in all likelihood the illness seemed multifactorial[10].

The improvement of diagnostic tools and microscopical mapping of the plaque morphology led to the discovery of the missing ideology of disease expression. The causal mechanisms of atherosclerosis were also defined as infectious and inflammatory[11-24]. Infections and the existence of pathogenic atheromas were screened by laboratory techniques. The implanted alien microbial populations confined in a hostess ecosystem competes for nutrients and settlement[25]. The plaque existence is driven by physicochemical characteristics of living organisms. It is a place of nutrient exchange between the colonizers and the host, a site of quorum sensing between different molecules and cells. The immunological and inflammatory reactions of the host manifest the presence of the foreigners. Microbial ecosystems are universally confronted and implanted. They survive and are affected by our dietary habits in the food chains.

The immunity system attacks exogenous and endogenous antigens by creating antibodies and immune-complexes which are deposited in the vascular wall contributing to atherosclerosis[26]. IgG antibodies against pathogens determine the severity of endothelial damage. Immuno-complexes formed after the exposure to antigens and microbes were found in plaques and in the blood circulation. It is a rescue mechanism of the innate immunity system to destroy microorganisms and intruders[27].

In the effort to solve the global problem of angiology, food was initially studied as a “stranger”-trigger factor of coronary artery disease[28]. The chronic inflammation resulting from food processing the human organization and the destructions caused by the immunological answers of the host are new associated links of the presence of the disease.
In one trial the IgGs against all food categories of patients with coronary artery disease were searched using commercial kits. The most common intolerances detected were in bananas, dairy products and eggs. However, there was a statistically significant difference for banana intolerance and its association with the serious forms of ischemic heart disease[26].

This led to the review of the literature and the analysis of the way’s banana intolerance mediates the atheroma creation. This review deals with the solution of the atherogenesis enigma for purposes of future targeted therapies. It emphasizes the microbial and antigenic infra-structure of banana fruit and its possible association with vascular changes and blockage.

**Aim and Methodology**

A computerized search was performed in Pub Med and Medline regarding banana intolerance and atherosclerosis including results from 1978 until up to 2020. The key words used were: banana, intolerance, inflammation, coronary artery disease, arteriosclerosis, oxidative stress, plaque, microbiota, biofilm, antigens.

We found one meta-analysis of prospective studies and one systematic review.

With the words “banana intolerance” we found 12 articles. Using veil combinations “banana intolerance and inflammation”, “banana intolerance and atherosclerosis”, “banana intolerance and oxidative stress”, “banana intolerance and microbiota”, “banana intolerance and plaque” no articles were noted.

With the key words “banana consumption and inflammation” there were 8 articles. With the words “banana consumption and atherosclerosis” 1 article was written. With “banana consumption and oxidative stress” 7 articles, with “banana consumption and microbiota” 2 articles, with “banana consumption and plaque” 4 articles were announced.

With “banana consumption and coronary artery disease” none was reported.

With the words “banana antigens” there were 209 articles noted but with “banana antigens and atherosclerosis” none.

We included all published articles such as cohort studies, objective screening projects, case reports, cross-sectional surveys, post-hoc analyses, experimental research studies, systematic screening analyses, single-center prospective studies, studies on rats, review articles on sepsis and on food chemical composition, randomized crossover studies about malnutrition and the increased risk of infections, randomized controlled trials, experimental laboratory studies.

One prospective study mentioned the food intolerances in relation to coronary artery disease. The methodologies used for detection of either hypersensitivity or intolerances associated to angio-changes hided some biases. Nutritional support, bioactive food particles, side effects of malnutrition were addressed.

Animal studies emphasized the effect of food consumption on gut microbiota, the deficiency of enzymes in the metabolic pathways and the metabolic disorders as risks of atherosclerosis, the toxicity of diet and drugs were documented as well as the host immunity defenses.

Screening projects in Intensive Care Units were conducted in order to monitor the appropriate nourishment of patients, controlling other comorbidities in an attempt to extend early treatment of diseases and prevention of unfavorable outcomes.

Allergies or intolerances to foods have been documented with other methods e.g. standardized questionnaires, skin tests, hypersensitivity tests, detection of serum antibodies Ig-E, RAST and immunoblotting inhibition analyses. The methods are not 100% predictive of food adverse reactions, meaning that food ingestion and metabolism and its side effects cannot yet clearly confirmed.

The gastrointestinal fermentation of food and the consequences were mentioned. The socio-demographic characteristics, economy and lifestyles (including diet) and their relation to prevention, cure and control of diseases were also addressed among countries.

**Classical and New Mechanisms Of Atherosclerosis**

Atherosclerosis is a chronic inflammatory disease of the vessels and not only the accumulation of lipids and apoptotic cells on the vascular wall[11].

The clinical and epidemiological characteristics of the disease are not fully justified by the multiple classical risk factors. *Cigarette smoking* is hazardous to cardiovascular system through mechanisms of oxidative stress, inflammation and endothelial dysfunction[1, 2]. Genetic disorders of lipid and lipoprotein metabolism and environmental factors influence the expression of atherosclerosis e.g. dietary habits[3]. Obesity which includes abnormality of metabolic pathways, autophagy and alterations of gut microbiota predisposes to cardiovascular diseases[4]. *Hypertension* causes damages to tissues and organs. High blood pressure depends on intrinsic factors (: renin-angiotensin-aldosterone system, the immune system, estrogen and testosterone sex hormones, metabolic disorders) and extrinsic factors (e.g. nutrition). It is implicated in initiating and progressing atherosclerotic lesions[5]. *Hyperlipidemia* means elevated serum cholesterol, triglycerides and Low-Density Lipoprotein levels. Statin therapy and diet rich in flavonoids reduces this cardiovascular risk[6]. *Diabetes mellitus* predisposes to atherosclerosis. It is known that hyperglycemia promotes inflammation, cellular oxidative stress, changes of the gut microbiota and microRNA expression causing plaque generation[7]. *Increased plasma homocysteine* reduces the levels of vascular protein S-nitrosylation and NO which regulate the cardiovascular system[8]. *Ageing* time-related impairment of vascular functions is mostly related to thickening of the vessel walls, oxidation and inflammation[9].

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The Mediterranean lifestyle consisting of low-fat vegetarian diet, less quantity of animal products and moderate exercise prevents or treats cardiovascular diseases[29].

Novel mechanisms of atherosclerosis include infections leading to inflammation and alterations of the lipid metabolism, deposition of cholesterol on the endothelium[12].

Microorganisms such as Chlamydia pneumoniae and Cytophagalivirus (: intracellular pathogens), Helicobacter pylori were detected within the plaques. They activate T cells, involved in the destabilization of the intima[12, 13]. Different microbial populations run simultaneously metabolic procedures having the ability to build neoplastic tissues (: biofilm, plaques, tumors)[14]. Antimicrobial interventions, dissection of human genes, protease inhibitors, vaccinations, acoustic or bioelectrical systems applied to prevent bacterial surface adherence and their bio-membranes are effective in controlling and killing bacteria and biofilm-mediated chronic diseases[15-18].

Microbial antigens, DNA, elevated antibodies, immune complexes and living organisms were found within plaques[18,19]. Helicobacter pylori, Chlamydia pneumoniae DNA were detected in carotid artery plaques by cultures and polymerase chain reaction[19].When endothelial cells are infected, active hydroxyl radicals are formed intracellularly, interleukins (IL-6) are produced, increased adhesion of monocytes and coagulant activity of endothelial cells altogether start atheromatosis[20]. Bacterial and viral infections may cause acute coronary events[21]. Immunological and inflammatory pathways link the endovascular infection with atherothrombosis and atherogenesis[18, 19, 21].

Chronic inflammation resulting from food processing the human body, leads to chronic diseases[22]. Microorganisms of the food enter the oral cavity invoking periodontitis, destruction of teeth and then invade the blood circulation[23, 24].

Periodontitis has been linked to cardiovascular disease, type 2 diabetes, systemic reactions(: platelet aggregation, hyperlipidemia, production of active inflammatory mediators, atheroma creation) [30, 31]. The presence of microorganisms such as Aggregatibacter actinomycetem comitans, Porphyromonas gingivalis, Prevotella intermedia, Prevotella nigrescens and Tannerella forsythia were detected on dental biofilm plaque using the PCR method[23]. The same kinds of bacteria found on dental plaques were also detected in blood cultures and in atherosclerotic plaques[23]. Prevotella nigrescens and Porphyromonas gingivalis were related with carotid atherosclerosis[32]. Periodontal treatment and oral hygiene reduces systemic inflammation, C-reactive protein and atherosclerosis, improves the coagulation status, the biomarkers of endothelial cells activation, endothelial function, arterial blood pressure, and overall health status[31, 33].

Virulent microbes secrete substances and enzymes (e.g. proteases), modulate immunological reactions resulting in the formation of dental plaque which resembles the atherosclerotic one. It is a well-organized community in a uterus-infrastructure made of polysaccharides, proteins and DNA (extracellular space) woven by the bacteria (: dysbiotic microbial biofilm). Periodontitis initiates systemic and chronic inflammatory diseases through the interactions of micro-organisms with the host megalo-organism and “metastasizes” extra-oraly[34].

Polyphenols and Flavonoids isolated from plant species inhibit the substances produced by the bacteria, their growth, the periodontitis and the biofilm (: plaque) formation[35]. When the dental plaque is reduced mechanically or chemically, local and systemic diseases (: intracardiac vegetations, rheumatoid arthritis, chronic obstructive pulmonary diseases, chronic infections, neoplasias, atherosclerosis etc.) are prevented, the oral and overall health is improved and the glycemic index of people with type 2 diabetes mellitus[34,36].

The bacteria enter the bloodstream through the oral cavity or other gates e.g. through lines and dialysates of hemodialysis, migrate to different places and damage the endothelium[37]. They weaken the intima of vessels, proliferate, create their biological colony, and build the plaque. They release endotoxins and participate in pathophysiological procedures, including amyloidosis, malignancies, atherosclerosis, chronic infectious and autoimmunity diseases, endocarditis, mortality. Biofilm formation in combination with an elimating immune response leads to inflammation and cardiovascular diseases[38,39].

A destructive immune-inflammatory response is a modern translation of risk to atherosclerosis besides the classical risk factors. Dietary deficiencies have crucial impact upon periodontal and cardiovascular diseases. Macro and microntrients (vitamins, minerals etc.) regulate the functionality of enzymes during the metabolic processes in human cells and gene transcription factors (e.g. pro-inflammatory and anti-inflammatory nuclear factors) [40]. Enzyme activities can be affected by the food deficiencies causing intolerances and coronary artery disease[26].

Furthermore, bacteria, viruses, fungi, protozoa, parasites and other microorganisms live within the intestinal tract influencing health, growth and development. The microbial communities can be affected by dietary habits, environmental and maternal factors[41].Bacteria can exist in planktonic form or in biofilm that allows them to survive under hostile conditions. Thebacterial colonies cause chronic inflammations including tissue damages, and they contribute in the cross-correlation between local infections and systematic diseases such as atherosclerosis[42]. Biofilms are resistant to antibiotics[39]. The relationship between infections and immune system, bacterial products as activators of the immunity responses, microbial metabolites are considered cardiovascular risk factors[42, 44].

Atheroma is immunological response to exogenous and endogenous antigens, causing inflammatory changes in the arterial wall and cholesterol accumulation. Antigens may be the constituents of food or its microbiobial load[26]. Antibodies, activated macrophages, products of oxidation, biofilms lead to atherosclerosis[27]. Diverse bacterial genomes (H. pylori, N. meningococcal, S. aureus, S. pneumoniae, S. epidermidis and E. coli) and T-cells are present...
in atherosclerotic plaques. T-cells react to bacteria starting endothelial dysfunction, thrombus and atheroma formation[45, 49].

Humoral immunity and the reactions against bacterial antigens can play an important role in the process of atherogenesis. Heat Shock Proteins (HSPs), proteins expressed by cells after exposure to stressful conditions and their cytotoxicity have been identified. Microorganisms produce HSPs mediating the creation of plaque through immunity pathways[46]. Antibodies against Escherichia coli HSP and Chlamydia HSP from patients with atherosclerosis were detected on human endothelia. The immunocomplexes (Ab-Ag) are deposited on tissues and cause inflammatory damages [47].

Pathogenic load defined as the number of positive antibodies and immunoglobulin G against cytomegalovirus, chlamydia pneumonia, helicobacter pylori, hepatitis A virus and herpes simplex virus-1 have been measured. IgG against pathogens is a determinant of severity of endothelial dysfunction [48]. The microbial DNA (especially of Chlamydia pneumonia and the Cytomegalovirus) activates the immune system cells via receptor TLR-9 [49,50]. Lipopolysaccharide receptors of macrophages (involved in microbiological recognition) result in cellular immunological responses to products of microorganisms and to specific antigens of oxidation. Inflammatory factors e.g. cytokines, toll like receptors, accumulation of immune cells exert atherogenic effects[51]. The lipoproteins constitute an endogenous immune system binding and destroying microorganisms with their byproducts through the formation of immunological complexes (Ab-Ag). Such complexes (antibody-antigen) can block the blood supply causing ischemia, destroy vascular cells and create unstable plaque [52].

In summary, antigens and microorganisms found in plaques or blood trigger the immunity reactions: activation of lymphocytes, production of cytokines, antibodies against HSPs or oxidized LDL, activated macrophages and lipoproteins, elevated fibrinogen etc.) connecting inflammation with atherosclerosis [52, 53].

Connection Of Banana Intolerance with Arteriosclerosis: (Immunological and Infectious Mechanisms)

References to banana describe both its usefulness and deleterious actions.

In investigations of hypersensitivity or intolerance reactions as trigger factors for abdominal angioedema attacks they reported tomato, green salad, fish, citrus fruits, apple, onion, garlic, cheese, chili, kiwi, milk, tree nut, peanut, strawberry, pineapple, green salad, fish, citrus fruits, apple, onion, garlic, cheese, chili, kiwi, milk, tree nut, peanut, strawberry, shrimps, bread, banana, leek, chicken, chamomile and alcohol. However, the disease mechanisms remain unknown[54]. Banana, rhubarb, curcumin, peppermint oil and ginger were searched as bioactive foods which could be applied in hospital patients. Malnutrition, infections, gastrointestinal events increase morbidity and mortality [55].

Bananas have been used as components in beverages to augment their value and stimulate the growth of beneficial gut microflora [56]. Lipid transfer protein (LTP) is a plant allergen causing IgE mediated adverse reactions. However, banana seemed safe for the LTP-allergic patients [57]. Antibodies against banana allergens were demonstrated by serological tests, detecting IgE antibodies[58]. In one study banana, cacao, egg, hazelnuts were recognized as foods responsible of the migraine attacks of patients aged from 7 to 18 years. Oligo-antigenic diet had a significant improvement of the migraines to some patients [59].

The existing bibliography promotes banana consumption. It is evaluated for its nutrients and effects on plasma antioxidants, markers of oxidative stress, inflammation and function of the immune system. Especially in the elderly, immunosenescence and malnutrition can contribute to increased risk and severity of infections [60]. Banana, berries, apples consumption increases cellular antioxidant activity and reduces the risk of neoplasias[61]. Banana meal enhances the resistance to oxidative modification of LDL and protects from atherogenesis[62]. Banana contains prebiotic indigestible carbohydrates and increases the bifidobacterial levels of fecal microbiota. Consequently the pathogenic microbiota is downregulated[63]. Although the fruit is benign, oral cariogenic bacteria such as Streptococcus mutans, Streptococcus sorbinus, Lactobacillus spp. and Actinomyces spp can decompose its carbohydrates into acids and create biofilms, lesions and plaques [64]. This fact can be interpreted as follows: the banana is a good fruit, but when it is consumed, “bad” bacteria metabolize it for their needs and for the benefit of their biological colonies. We assume that plaques are biofilm colonial clusters.

Vegan diet prevents cardiovascular diseases[65]. Banana contains phenolics, carotenoids, bioactive compounds and phytosterols, protecting the body against oxidative stresses and chronic diseases [66, 67]. Bananas contain sucrose, fructose, glucose, fibers, tryptophan, and B-complex vitamins. The vitamin B6 regulates blood glucose levels. The consumption of banana products prevents diseases linked to digestion and glucose/insulin metabolism or treats type 2 diabetes[68, 69]. The B6 and B12 vitamins as well as potassium and magnesium minerals found in them neutralizes excessive acidity. It has iron content curing anemia. Its products exert beneficial effects on glycemic control, lipid metabolism, and blood pressure[69]. Banana can limit the risk of death from strokes[70].

Although the above knowledge is true, there is evidence that banana intolerance results in diseases through immunity pathways and secondly the hostile microorganisms carried in or near the fruit may work in a vicious cycle of inflammation. In a recent prospective study, patients with coronary artery disease were checked for food intolerance with the use of commercial kits Food Detective/Golden test. The banana, egg milk wasn’t prevalent. There was a statistical evidence of correlation of banana intolerance with the severe form of atherosclerosis. A percentage of population defends against banana (its microbiota or substances) [26].

A possible explanation of the above study is the fact that some individuals cannot digest banana, opposing to its foreign
Immunity receptors recognize dangerous molecules and antibodies. The microorganisms are associated with food that can build in the gut. Modifying it through the food (e.g., soil-water-air) can influence the expression of genes in the gut microbiome.

Intestinal microbes influence the expression of genes in the enterocytes related to immune function and lipid metabolism.

The intrinsic flora, dysbiosis and a numerical increase of pathogens results to cytokine release, damage of the intestinal barrier and increase of atherogenic metabolites, inflammation through immunity pathways, cardiovascular side effects [78].

Banana contains its microflora stimulating and interacting with oral and gastrointestinal microflora. The microorganisms are necessary for the digestion, metabolism and absorption of food. Diet should strengthen the benign gastrointestinal flora, alter bodily acidity (pH) as well as the incidence of illnesses. Individuals cannot consume all food categories because of intolerances or allergies to their constituents and microbes, lack of metabolic enzymes. Eating banana also means bodily “contamination” with pathogenic intruders. Banana contains its own bacteria that produce metabolites [79]. The microflora of the consumer dissolves banana starch into glucose, fructose, sucrose, and acids are produced [80]. Approximately 164 lactic acid bacteria (e.g., Lactobacillus plantarum, Lactobacillus, Weissella) were isolated from bananas in Taiwan which affect the incidence of banana diseases (e.g., fusarium disease), its sugar content and acidity (sugar to acid ratio) [81]. The banana can become ill depending on the ground, feed and fertilizers used. Modifying the texture of soil (bacterial burden, number of actinomycetes, bacilli numbers, the proportion of bacteria to fungi), we can improve the health of the fruit, its quality and indirectly the health of the consumers [82]. Pesticides pollute grasslands, plantations through the food and water [83]. Bacterial and fungal communities in the soil, organic matter, interactions between bacteria and fungi in the ground and within the fruit correlate with banana diseases and depletion [76].

Generally, the amount and the activity of enzymes a consumer secretes after a meal are influenced by the quality and quantity of food [84]. A cause of banana intolerance is the insufficient metabolism and absorption because of enzymatic deficiency and disturbance. Banana offers water and nutrition to human organisms as well as to its microflora [85]. The good scenario is that it offers water, food and protection to friendly bacteria (e.g., lactobacillus). It contains starch resistant to gastric acid and intestinal amylases [86, 87]. The resistant starch competes the own bacteria that produce metabolites from the consumption of fruits including banana has acidogenic potentiality because they are fermented with either oral or extra oral bacteria to sugars and acids [80]. Fructo-oligosaccharides (oligosaccharides constituted of fructose and glucose chains) present in banana are non-absorbable, having prebiotic effect and stimulating the nonpathogenic intestinal microflora. The banana starch is consumed by intestinal microbiota for anaerobic cellular energy production [85]. Their final metabolites are the carboxylic acids, the lactic acid etc. The chemical composition of plants characterizes their bacteriostatic features. For example, kinds of mint contain essential oils battling the Staphylococcus epidermidis ATCC 35984 which creates bio membranes [88].

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However, banana presents bacterio-feeding and inflammatory characteristics.

Banana fibers are used for the production of surgical sutures because of their powerful inflammatory reactions resulting in healing processes [89]. The fibers reinforce the intestinal bacteria (lactobacilli, bifido-bacteria). Lactobacilli excrete antimicrobial substances and unitete cells. Banana starch along with these lactobacilli decreases pH, increases the production of lactic acid and short chain fatty acids from the intestinal microbiota (: a source of energy for the intestinal cells) and inhibits the population of others e.g. salmonella typhimurium SA2093 [90]. The plant fibers include polysaccharides, oligosaccharides and inulin which are fermented with intestinal microbiota influencing its composition and activity [91].

Dickeyaceae MS1-Erwinia chrysanthemi is a bacterium that forms biofilm and pollutes banana plantations[92]. The biological colonies (: bio-membranes) built by this microorganism are constituted of bacterial cells and extracellular matrix of polysaccharides made by the bacteria. Their colonies, extracellular proteases and toxins harm banana causing its decay and indirectly illnesses of the consumers [93]. The quantity and quality of microbiota and their efforts for survival (=dysbiosis) is the root of diseases. Microorganisms produce useful metabolites (: vitamins, potassium etc.) or dangerous molecules (: alcohol, acids) in the large bowel, serum, blood, fetus, urinary tract, cardiovascular system etc. Disorder of the metabolism of choline by bacteria, involves overproduction of trimethylamine precursors involved in atherosclerosis, increased production of hydrogen sulfide, ammonia and bile acids leading to inflammatory diseases. Microbial production of phenylalanine, tyrosine and tryptophan-indole derivatives contribute to the pathogenesis of chronic liver and kidney disease, cardiovascular diseases, psychiatric and neurological illnesses etc. The knowledge of microbial metabolism-metabolites and dietary changes can prevent diseases [44].

The pathogenesis of banana diseases is explained by the harmful bacteria isolated from the fruit such as Vibrio harveyi, V. ovensii, V. sinaloensis-like, V. campbellii, V. shilonii, Vibrio sp. and Photobacterium damselae. They had virulent genes and whips because of their powerful inflammatory reactions against proteins (: heat shock) and oxidized LDL are involved. Selectines, integrins, immunoglobulins (: adhesion proteins) participate in the interaction between leukocytes and vascular endothelium, play a role in the integrity of tissues, inflammation, the creation and progression of atherosclerotic plaque [97]. Various antigens stimulate immune responses causing inflammation and the development of atherosclerotic plaques. Antigens may be the dietary ingredients or its microflora [26]. Immunomodulating treatments will target those mediators of inflammation, preventing cardiovascular diseases [98].

Bacteria are living in various sites in the form of a biofilm. Disturbance of homeostasis of biofilm and increase in the number of acid-forming bacteria lead to the development of the diseases. The etiopathogenesis of atheroformation is explained by the biofilmic colonization and the metabolic bacterial activities[99].

Besides the classical risk factors, microorganisms and antigens also promote atherogenesis. The inflammatory response against pathogens or antigens of food may have an important role in the creation and stability of atherosclerotic plaque. Banana’s foreign microorganisms enter the host changing the flora of the internal ecosystem (: oral, gastrointestinal, cardiovascular) and thrive for a living. Especially the oral and gut microbiome is associated with the infectious state of the body and diseases. There is a complex relationship between microbiota with the classical risk factors of atherosclerosis, obesity, insulin resistance, fatty infiltration of the liver, blood clotting disorder mechanisms, construction of plaques[100].

Biological battles are observed in nature and in mankind. The health of plants, animals and humans is a matter of balance. We use friendly microorganisms to fight the “evil” ones. The atheroma is not formed only by lipids and cellular debris[101]. The activation of the immune system, inflammation, monocytes, macrophages, cytokines during plaque formation represent procedures against various stimuli. These responses to foreign matters change the endothelium and its function. Elevated monocytes, activated T-lymphocytes, oxidized LDL cholesterol, increased production of peroxides, activated macrophages, cytokines (: IL-1, IL-6, gamma interferon) and Lp (a) lipoproteins have proved to be immune responses while the atherosclerotic plaque is created sub-endothelially. In the process of inflammation the levels of CRP (: acute phase protein), fibrinogen and the erythrocyte sedimentation rate are increased. Medications (statins, aspirin, ACE inhibitors, antibiotics) work as anti-inflammatory drugs. Microorganisms have been detected on atherosclerotic plaque or blood, e.g. Helicobacter pylori or Chlamydia pneumonia. Reactions against proteins (: heat shock) and oxidized LDL are explained by the immunity pathways[53].

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Banana intolerance and consumption to cardiac patients means eating pathogens and antigens. The body produces antibodies which bind to antigens. These complexes are circulating and deposited on the endothelium. Endothelial dysfunction and inflammation is initiated ending in atherogenesis. It also means alteration in total acidity. A megalos organism is better to be alkaline than acidic. Within alkaline environment, no disease is present. PH of blood below 7 equals death. Atheromatic plaque is a form of highly diverse biofilm built by the endogenous microbiome[102]. It is necessary to understand its composition so that we proceed to the appropriate treatment using methods such as enzymatic lyses[103, 104]. Fibrinolytic therapy with tissue plasminogen activator (alteplase) was used in vascular occlusions. The rheological improvement was due to the reduction of plasma viscosity[105].

All plaques are biofilms resistant to antibiotics. Antimicrobial peptides (AMPs made up of 10–50 amino-acid) have been considered crucial in human immunity. They act against Gram-positive and Gram-negative bacteria, fungi, eradicating biomembranes. The anti-biofilm molecules (herbal compounds, lantibiotics etc.) interfere in the quorum sensing pathways, adhesion mechanisms, disruption of extracellular DNA, and the messengers involved in biofilm formation[106, 107].

Dietary changes can fight biofilms and infections of the vascular system. Proanthocyanidins (PACs) from cranberries have properties against Escherichia coli, P. aeruginosa and their biofilms[108]. Diet with stevia, silytul, probiotics, vegan food, mint resulted in less caries and plaque development[109]. The Mediterranean diet with olives, nuts, fruits, vegetables, fish, and minimal consumption of animal products changes our microbiome, displacing the aciduric and acidogenic bacteria[109, 110].

Atherosclerosis affects all organs and fetus in pregnancy. Maternal hypercholesterolemia enhances fetal lesions and postnatal atherogenesis[111]. It equals inflammation and may be associated with banana intolerance leading to immunocomplexes (IgG-Ag) deposited on the endothelium. The microbiota of this fruit may build the arteriosclerotic biofilm.

We need to understand better the food related microbiology and immunology, so that we apply therapeutic nutrition.

Conclusions

All food categories cannot be consumed by all consumers due to intolerances or allergies. Research through the literature gives rationale to the relationship between banana intolerance and consumption with arteriosclerosis. It is necessary to explore banana microflora and constituents for the benefit of immunity system and biological microenvironment without causing metabolic disorders or inflammation. It is also mandatory to search the microbiota of the host and the ways it works for a living.

What we have seen in terms of the atherosclerotic mechanisms and banana suggests that banana intolerance and consumption may mediate atherogenesis and growth of plaques. The host organism attacks the "foreigner - banana" resulting as a boomerang to its own destruction.

Banana diseases depend on the soil and its microbrial populations. The relationship between the soil and banana are not clear. The soil is the "food" source of banana and its soil-borne diseases.

Atherosclerosis is associated with nutrition and its feed-borne diseases. Under circumstances banana's living microorganisms can build the biological colonies of the plaques for their safety or the microorganisms of the host can digest banana producing metabolites and reconstruct colonies for their survival producing plaques and destroy the human body.

Dietary biological “microenvironment” and antigenic content may be the key-translation to atheroma formation. There is limited information about fruit antigens and microbiota and their interactions with the host. Antigens may be the ingredients or microflora of diet. Research focusing on adverse immune and microbiological responses to food could clarify the underlying mechanism of banana-induced atherosclerosis. Biofilms and immune-complexes could link banana with inflammation and atherosclerosis. Plaques are built by living "microbes" fighting for home and food.

The banana nutrients are good for us and for our intrinsic "habitants". Banana is a benign fruit but the host organism (consumer) weaknesses attack and incriminate it.

Extensive studies on the microbiology and dietology of diseases should be conducted to further enlighten the "gaps" of this issue.

Acknowledgement

Many thanks to the doctors Spyros Potamianos (chief staff in the Department of Gastroenterology), Ioannis Skoularigis and Filippos Triposkiadis (chief staff in the Department of Cardiology), Nikolaos Tsilimingas (head director of the Department of Cardiovascular Surgery), Kyriakos Spiliopoulos (thoracic and cardiac surgeon), Marios Karvouniaris and Dimosthenis Makris (medical doctors of Intensive Care Unit), Andrew Xanhtopoulos (cardiologist) all employed in the University Hospital of Larissa at Thessaly / Greece for their cooperation.

Author contributions

Angeliki or Aggeliki Tsantsaridou had the idea and wrote this particular article. George Valsamakis revised it and corrected it. The rest of the authors also contributed to drafting the manuscript.
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Citation: Georgios V, Angeliki T, Olga T, Maria A, et al. (2020) Participation Of Banana Intolerance in Atherosclerosis Review Of the Literature. J Nutrition Health Food Sci 8(1):1-10. DOI: 10.15226/jnhfs.2020.001173
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