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Surveillance utilizes multi-omics in cardiovascular disease: Diet and its potentiality in Preventive index

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Abstract

Cardiovascular Disease (CVD) is characterized by multidimensional risks including drug, diet, lifestyle, stress, and metabolomics diseases which cause mortality and morbidity depending on age and status of chronic diseases. However, emerging evidence indicated it is preventable health complications that depend on risk management along with lifestyle change, and personalized medication that include alternative measures like Diet use following molecular diagnostic and imaging analysis. CVD is mainly attributed to the narrowing of blood vessels through atherosclerotic lesions and/or thrombosis. Hypertension, obesity, and hyperlipidemia are major risk factors for the development of CVD and treating these diseases is essential in slowing down progression of CVD. Inflammation appears to play a pivotal role in CVD and can be measured through a simple blood assay (CRP). Multi-omics approaches have been essential in the development of treatments for CVD, in the prevention of CVD, and in the diagnosis of CVD. There are many outcomes available to help with diagnosing CVD and omics platforms have helped scientists and clinician develop these diagnostic tools. Radiomics has played a key part in the diagnosis of CVD as being able to view the diseased heart is essential in determining CVD progression and the treatment options suitable for that secondary disease related. Nutrigenomics is emerging as the future of medicine such as utilizing treatment strategy innovation instead of medications, but it is still in its infancy. Nutrigenomics will open the doors to different therapeutic drug targets and allow us the ability to be more specific in our treatment options. There are only a few gene-diet interactions documented that increase a person's chances of developing CVD. Curating an individual diet and treatment plan based on somebody's genetic disposition or skewed immune responses following personalized diagnosis will be essential in the survival of these severe CVD patients. Key issues referring to risk surveillance and prevention is a distant approach which reflects several factors: for example, what type of tools can be used to conduct diagnosis, molecular diagnostic tools detect what type of biomarkers are present prior to prescribing the personalized diet and to ensure diagnostic accuracy. Recently, increasing findings emphasize dual aspects of diet such as immune enhancers and modulators in which gut microbiota has been proven to play a major factor in development of CVD. The future direction of omics studies will foster the ability to test the impact of gut microbiome of a patient with CVD following diet driven organ protection as well as prescribe essential components of the diet that can be adjusted with proper probiotic medication. Proper diet adjustments can correct the organ dysfunction that occurred due to interaction between molecular mismatch and cellular damage following stress-mediated damage or chronic disease. Further micro-scale assays and molecular diagnostic techniques following nutrigenomics application to the patient could be beneficial to allow patient' care shift from physician driven and clinic based to self-management with knowledge based at home treatment programs that work by envisioning molecular reprogramming and rejuvenation of damaged organ. These at home treatments can be utilized with development of radiological data with innovation of software. The aim of the short review is to visualize the current role of nutrigenomics and diet formulation for integrative care (e.g., diagnosis, prevention, and treatment of CVD) which would take advantage of earlier prevention synchronized with current medical tests, imaging techniques. Health economy like management can reduce medical cost with disease prevention disease and could modulate the following: enhance knowledge-based interaction between body and diet, discuss cognitive enhancement how sensing with molecular

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behavior under image-management platform, monitor drug surveillance of current treatment options in CVD and the pitfalls of current omics application and data transformation needs for patient care in the future.

Keywords: Cardiovascular disease; Diet; Molecular target; Metabolomics; Nutrigenomics; Molecular imaging; Radiomics

1. Introduction

Cardiovascular disease (CVD) is a big public health concern with excessive health cost, susceptibility to lifestyle factors, stress related morbidities, and environmental factors that correlate with climate change. It is the number one leading cause of death worldwide with an annual death toll of over 17 million people [1]. In the United States, CVD attributes to 39 percent of all deaths [2]. Ischemic coronary artery disease (CAD) and stroke are main contributors to high CVD mortality. Ischemic CAD is caused by the accumulation of fat inside the arteries (atherosclerosis) and this accumulation of fat leads to plaque which narrows the blood vessels, restricting blood flow. CVD has been linked to obesity as the increased fat intake and a high processed food diet leads to atherosclerosis. The rupture of these atherosclerotic arteries and/or movement of blood clots are the leading cause of myocardial infarction (MI), heart failure, and stroke [1].

Hyperlipidemia and type 2 diabetes are well studied risk factors for CVD and can be directly linked to CVD development. According to a study done in 2017 a patient is 26% more likely to develop CVD if they suffer from hyperlipidemia [3]. These risk factors are easily diagnosed through simple laboratory blood tests. In previous studies, Data suggests that CVD is a preventable disease that can be curbed with a good diet (or nutritional therapy), exercise, and stress-control techniques in a cardiac rehabilitation program [4]. CVD does have a strong heritable component and certain genetic factors can attribute to a higher probability of developing the disease. Preventative medicine has gained some traction in the past few years and multiple educational programs have been implemented. Public health programs focus on preventative care and risk management for CVD.

Recent studies have shown that gut microbial composition is linked to CVD as the microflora affects multiple metabolic pathways. Multi-omics has emerged has an invaluable tool to understand the modifications in cell and molecular signaling pathways that reflect diversity of microflora and interaction with variety of metabolic syndromes that promote CVD disease development. Omics provides molecular indicators for diagnostic use and therapeutic direction with treatment strategies by enhancing the role of identifying biomarkers in cellular and molecular pathogenesis and discovery of new therapeutic drug targets [1, 5].

There are several lines of evidence that support how stress affects CVD vulnerability regarding differences based on genomics data (e.g., GWAS) among sex and molecular alteration. For example, A study led by Albert et al. reported that cumulative psychological stress could associate the outcomes such as primary of CVD related pathological risks (i.e., myocardial infarction, stroke, coronary revascularization, and death) and secondary CVD related societal and behavioral risks (i.e., sleep, anger, cynical hostility, depression, anxiety, social support, intimate partner relations, and volunteer and social activities) [6]. The study suggests that variable factors including younger age, black race/ethnicity, divorced or separated marital status, increased prevalence of obesity, smoking, diabetes, depression and anxiety resulted from stress which deemed to be susceptible in CVD interventions. For aging process, health disparity regarding CVD risk among women could be ensued due to cumulative psychological stress-driven oxidative stress interacted with genetic and environmental factors thereby pathological status exacerbated organ damage (i.e., myocardial calcium handling, arrhythmia, and cardiac remodeling, formation of atherosclerotic plaque) influenced by malfunction of cellular and molecular function with chronic diseases ended up CVD risks: coronary artery disease, congestive heart failure, arterial hypertension, pulmonary arterial hypertension, peripheral arterial disease, myocardial ischemia/reperfusion-related injury, stroke, cardiac arrhythmia and venous thrombosis attributed by MPO molecular malfunction (i.e., Myeloperoxidase, MPO) or sirtuin (SIRT, SIRT1 and SIRT6) which regulate inflammation and oxidative stress, vascular resistance to aging [6-9].

Interestingly, it appeared molecular crosstalk could be a determinant as a surveillance clue between diet and CVD risks. Also insulin resistance could be intricated with molecular intervention caused by chronic metabolic disorders. With respect to molecular interaction to CVD risk, Naghipour et al. (2021) reported evaluation of trimethylamine N-oxide (TMAO) variance to CVD risk such as insensitivity, ranged from cellular levels (i.e., inflammation, oxidative stress, scavenger receptor up-regulation, reverse cholesterol transport (RCT) inhibition) to organ damage (i.e., cardiovascular dysfunction), focus on the cellular and metabolism level which reflects effect of metabolites following diet on gut microbiota [10]. In addition, another study investigated the connection between metabolic networks in cardiac metabolism dysfunction and CVD risk following alteration of glucose metabolism (so called chronic hyperglycemia) or

lipid metabolism (I.e., dyslipidemia and formation of atherosclerotic plaque) cause insulin resistance result in endothelial cell dysfunction which lead to myocardial malfunction [10, 11].

This short review provides some insight on molecular dynamics which shed light on key determinants of risk assessment and preventive indicators.

2. Diagnosing CVD with molecular assessment tool

Hypertension is a highly treatable CVD risk factor and there are many treatment options available. Hypertension can be measured directly with a blood pressure cuff at the doctor's office. Using a stethoscope and blood pressure cuff this risk factor should be measured on a regular basis to ensure proper control. High blood pressure increases the chance of stroke significantly and can lead to arterial damage leading to crucial organs. The relationship between blood pressure and CVD is continuous [12].

Hyperlipidemia is a major risk factor for CVD and is the number one cause of atherosclerosis. Blood tests are the current gold standard for testing for hypercholesteremia and a lipid panel can show disease progression or treatment success. A lipid panel measures four biomarkers: low-density lipoproteins (LDL), high-density lipoproteins (HDL), total cholesterol, and triglycerides. There are well known associations between elevated total cholesterol levels and CVD [12].

Inflammation appears to play a preconditional role in the development of atherosclerotic cardiovascular disease and can be measured directly with altering of macrophage molecular features in blood assays [13]. The C-reactive protein (CRP) is a measurable biomarker that increases with inflammation and can be tested in most laboratories. Elevated CRP levels are concomitant in patients with recent myocardial infarctions and directly correlates with tissue damage [14]. Recent developments of high-sensitivity assays have made it possible to measure CRP levels in the blood, even at micro concentrations. Furthermore, it was suggested that infection attributed to immune network and crosstalk machinery depend on pathogenic type and cellular and humoral response with pattern of cytokine in the lesion of cell and tissues. For example, severe atherothrombosis following provoked microbial infection could intervene chain of myocardial oxygen supply which cause organ dysfunction associated with myocardial ischemia concomitant with hypotension, hypoxemia, fever, tachycardia [15]. Recently, a study illustrated there are two different modes of infection which could affect CVD pathogenesis. The study detected fifty species of microorganisms by directly measuring the gut cavity and the plaque of atherosclerotic vessels and indirectly through production of inflammatory cytokines following alterations of immune tolerance by interacting with innate immune molecules (i.e., Toll like receptors, TLRs) or generation of metabolite as outcomes of pathogenic byproduct (i.e., trimethylamine (TMA) vs TMAO oxidized form by flavin monooxidases 3) which could accelerate CVD pathogenesis due to molecular alteration of platelets.

Homocysteine is a naturally occurring amino acid that is found in blood plasma. An analysis of 23 studies on ischemic heart disease showed a highly significant association between homocysteine levels and ischemic heart disease [16, 18]. Elevated homocysteine levels have been observed as a possible cause of CVD, stroke, and deep vein thrombosis (DVT). This observation was first seen in children with genetic homocystinuria where most died of premature vascular disease [12]. Total homocysteine can be measured with a blood assay and results can help determine preconditional factors of CVD and stroke.

Interestingly, the relationship between gut microorganism and CVD risk was demonstrated using fecal samples of 405 Chinese subjects that were diagnosed with atherosclerotic cardiovascular disease (ACVD). The study indicated that Trimethylamine N-Oxide (TMAO), metabolite by hepatic flavin-containing monooxygenases (FMOs), increases atherosclerosis and leads to accumulation of cholesterol that interacts with surface molecules within immune cells. This process is exacerbated by existing chronic diseases such as coronary artery disorder. Different profiles of gut microorganisms were studied in healthy individuals with varying diets and lifestyles, this factored into gut metabolism and reflects disease pathogenicity and immunogenicity in healthy people [17].

The metabolomic study led by Karlsson et al. illuminated that atherosclerotic cardiovascular disease (ACVD) could be influenced by different types of gut microorganisms which were detected in patients (the genus *Collinsella*) and in healthy control participants (e.g., the species *Roseburia* and *Eubacterium*) differently. It suggests that diet could be considered as a potential trigger and preventive option for patients who are suffering from chronic diseases such as CVD [19].

There was correlation between homocysteine and extracranial carotid-artery stenosis in cases of patients with high Homocysteine who had vulnerable blood vessels and damage due to a low vitamin B6, 9 and 12 level. There is a strong

association between an elevated level of homocysteine (>10) and a high risk to damaging the cardiovascular system. Which includes injury of artery wall, fatty acid accumulation, plaque formation in vessel lining, and a 2.5-fold increase in the risk of Alzheimer disease. Elevated levels of homocysteine can be caused by a diet of high carbohydrates, drinking alcohol and smoking tobacco, a deficiency of vitamin B (vitamin B6, B9, and B12) and magnesium levels. Depression of the pituitary gland and genetic alterations can influence homocysteine [20]. Further study needs to be done to clarify the impact of diet on CVD by utilizing nutrigenomics and metabolomics. This can be done by visualizing genetic determinants based on the patient's genomic data regarding homocysteine metabolism and gut microbiota activity.

Radiomics is a necessary tool in assessing CVD disease and there are multiple modalities to assess for disease like traditional imaging to detect molecular behavior upon disease status with or without treatment options. There are several different imaging techniques are listed as follows: coronary artery calcium (CAC) by computed tomography (CT) imaging, carotid artery ultrasound, and abdominal aorta ultrasound (AOC) [12]. These imaging techniques can assess for carotid intima-media thickness which reflects hypertensive medial hypertrophy and is a major causative agent of stroke. CAC scores predict CVD events in asymptomatic adults and is an important tool in assessing CVD risk. New radiomics' tools have advanced to molecular imaging which show risk factors that are invisible to traditional structural diagnostic imaging techniques. Carotid plague FTD PET (Positron Emission Tomography) imaging is a new image technique that uses FDG (radiolabeled glucose analog) and PET imaging. Radiolabeled FDG concentrates in metabolically active cells and can be detected using PET imaging [21]. In atherosclerosis the FDG accumulation correlates to plaque hypoxia and macrophage infiltration. This radiolabeled FDG accumulates in the atherosclerotic arteries and the marked inflammation is visible on a PET scan. The only setback to this is that only the aorta and other larger arterial beds are visible on the PET scan.

Imaging skills implemented with nutrigenomics and metabolomics are critical in monitoring organ health aspects such as elasticity, endurability of muscle, and status of blood vessel health. Predictability of scale is variable depending on age, race, education level, and household income. However, mental health and stress management are critical factors other than diet that can prevent development of CVD. For instance, the impact of diet in prevention of CVD is associated with systematic resistance of organ damage, which is dependent on the number of chronic diseases, variety of medications, along with ability to regenerate healthy tissue through exercise and healthy lifestyle choices.

Recently, a molecular sensor was able to detect metabolic connectivity to CVD pathogenesis through gut microbiota and was associated with development of cardiovascular diseases (i.e., coronary artery disease, Chronic heart failure, Atrial fibrillation) and risk of chronic diseases (i.e., Diabetes mellitus, hypertension, obesity, and dyslipidemia) by modulating molecules that attribute to functionality of each tissue and organ through specific metabolites (e.g., TMAO) and immune molecules [22]. Further study needs to confirm the association of CVD with this molecular network.

Proteomics have been essential in monitoring and diagnosing different CVDs. Regarding heart failure the biomarker, B-type natriuretic peptide (BNP) can be tested to determine if the heart muscle is under excess stress. BNP is released into the blood stream when the myocardium wall is under stress, and this is a common occurrence during heart failure. BNP can also be present during other pathologies, and therefore the specificity for this biomarker low. Another proteomic biomarker used in clinical diagnosis of ischemic heart disease (IHD) is creatinine kinase (CK) and myoglobin. CK and myoglobin blood tests both allow for the detection of IHD. CK is released from myocytes that have been injured during a MI and can be of great clinical significance for early detection of MI. The levels of CK in the blood rise rapidly during the onset of myocyte stress and this test is essential for inpatient heart failure patients [23].

The ultimate goal of increasing the number of novel protein or peptide biomarkers is to help assess and diagnose cardiomyopathies before they cause damage to the myocardial cells and death occurs. These biomarker assays must be investigated during clinical trials and pass through three different stages: identification, validation, and application. Proteomics will be essential in diagnosis and determining disease progression of CVD.

3. CVD Prevention structured with molecular value

Nutrigenomics will become a very important prevention and treatment in cardiovascular medicine. Nutrigenomics is a suitable approach for the prevention of cardiomyopathies and atherosclerosis. It has been proven that diet plays a crucial role in the development of CVD and certain nutritional requirements must be met in order for the heart to pump efficiently. High-fat processed meals have been shown to increase the risk of CVD and in a large observational prospective study patients that consumed excessive ultra-processed foods saw a 10% increase in CVD occurrence [24, 25]. More studies need to be done to confirm this in all populations.

Diet is not a universal type of prevention as different individuals have different nutritional requirements and need specialized diets. To create a "personalized" diet, gene-diet interactions need to be better understood. Gene-diet interactions have been documented in multiple studies, but the problem is reproducing these events for another study. Replicating these gene-diet interactions in different populations remains a major hurdle in nutrigenomics [25]. Nutrigenomics is in its infancy and more research needs to be done on gene-diet interactions. The latest genome-wide association studies (GWAS) have recently reinvigorated efforts to understand the genetics behind CVD. The GWAS has confirmed that inflammation and lipid metabolism are major contributors to CVD development.

Diets rich in w3 polyunsaturated fatty acids (w3-PUFAs) such as alpha-linolenic acid have been associated with a decreased incidence and severity of CVD. Some of these beneficial effects come from metabolites such as prostaglandins, thromboxane, resolvins, and leukotrienes [26]. The biological response to these metabolites is moderated by cognate receptors that regulate gene expression and affect the metabolic and signaling pathways that are associated with CVD. There are certain single-nucleotide polymorphisms (SNPs) in key enzymes that affect the metabolism and response to w3-PUFAs. Most notable is the FADS1-rs17457 polymorphism that presented with significantly less omega-3 PUFA concentrations in the patient's blood samples [27]. This is just one example of a gene-diet interaction.

Many diet initiatives have been created by national and international organizations in the past few decades, but they have not been successful. The challenge lies in the ability to change a person's behavior, the presence of mass media pressure, sedentarism, and lack of education. Public health focuses on prevention of disease and aims to increase the quality of life for all population and community. No matter how much education and intervention an organization implements on a population it is still up to that individual lifestyle and behavioral approach to adopt the recommended changes.

4. Treatment of CVD with molecular assessment of functional outcome in Omics

Early detection of CVD is necessary to perform interventions to treat the disease. CVD can be treated with interventional medicine such as surgery and surgical interventions are usually done following a cardiac event. CVD treatment is mostly about controlling the symptoms and keeping the myocardial cells from being overly stressed and then dying. MI's can lead to heart necrosis where some of the muscles in the heart die following the cardiac event. Hypertensives are the first line of defense and should be prescribed first as this keeps blood pumping through the vessels at proper pressures, without further damaging vessels that lead to crucial organs.

Genomics plays a key role in treatment of CVD as there are certain genetic predispositions that increase the probability of developing CVD. Several studies have amplified the role that genetics plays in CVD documenting that CVD has a high degree of heritability, especially in male subjects [1]. There are more than 300 genetic variants associated with CVD and a further study targeted a metabolomic approach to show that carbamoyl-phosphate synthase 1 (CPS1) was associated with CVD. CPS1 was shown to decrease the risk of CVD in female patients [1].

The gut microbiome has recently emerged as a significant regulator of cardiovascular health and risk of disease. Recent advancements in metabolomics have made available some assays that can test the gut microbiome to determine the status of the gut and where the problem may lie. In 2008 the Human Microbiome Project was created to determine if the gut microbiome was a causative agent in certain pathologies. Since the initiation of this project various bioinformatic and biostatic tools have been developed and applied to these microbiome studies. The gut microbiome unveil its potentiality as a therapeutic target for CVD is in its infancy level and there are a few proven alternative pathway between gut microbiota and host that currently exist several chronic diseases (i.e., hypertension, chronic kidney disease, and atherosclerosis) [28-31, 36]. Probiotics and fecal microbiota transplantation have shown some potential in the reduction of CVD risk but there is not currently enough data to prove that this is a sufficient treatment [32]. "There is evidence that dietary interventions may improve cardiovascular health, and this has been confirmed through the application of multi-omics approaches." [1]. For example, a diet high in fiber was shown to elicit changes in the gut microbiome that lowered the risk of CVD development. The most notable change was the increased generation and shuttling of acetate, which is a molecule associated with improved cardiovascular function. The transcriptome of the high fiber diet revealed an upregulation in certain genes called *Tcap* and *Timp4.* These genes are considered to have a preventative role in heart disease, for example hypertrophic cardiomyopathy and dilated cardiomyopathy [1, 33, 34].

Although we currently have many pharmaceutical and interventional treatments for CVD, we are still a distance away from developing gene-specific treatments. By exploring the human genome, we have discovered multiple genes that are associated with the development of CVD, and these will play a crucial role in treatment for the future. Developing personalized treatments is the future of medicine and the key to some patient's survival.

5. Impact of Diet on CVD prevention

Prevention of CVD contains a multidimensional process that utilizes molecular sensors that capture signaling cascades and detect potential disease-causing molecules. Advent of digital medicine and quantitation of molecular genomics allow us to optimize potential benefit for the patient by mitigating risk of CVD progression with one's lifestyle that includes patterns of stress, and diet. CVD progression is linked to the aging process due to the increased level of oxidative stress in the elderly. Eating behaviors and diet choices can affect metabolism by altering the type of endocrine hormones released during exposures to environmental stress. A scientific report led by Ginty A. et al. (2017) demonstrated that a potential neuronal metabolic network was associated with CVD. Accumulated psychological stress and CVD risk rely on a two-step cascade which includes stressor-evoked cardiovascular reactivity through visceromotor and viscerosensory mechanisms and are metabolically dysregulated (or extreme stressor-evoked cardiovascular reactions) [35].

There are several lines of evidence that support CVD risk is mitigated with management of a functional diet and healthy gut microbiota management. In a previous epidemiological study clostridia was treated with a regimen of plant derived flavonoids and was shown to reduce CVD risk parameters (e.g., BMI/waist circumference) along with inflammatory cytokine levels (e.g., TNF-alpha) [36]. Moreover, functional diet categorized by several patterns of bioactive compounds (e.g., fruits and vegetables, whole grains, dairy products including fermented products, legumes, nuts, green tea, spices, olive oil, seafood, red wine, herbs, and spices) include dietary proteins that could change cardiometabolic risk factors. For example, low-density lipoprotein cholesterol (LDL-C) was altered by a plant derived diet style and insulin sensitivity was increased by polyunsaturated fat (PUFA) [37-39].

Psychological stress is integral in the pattern of gut microbiota where they play a role in disease management (e.g., chronic kidney disease, atherosclerosis, hypertension, and CVD), health risk assessment, and prevention. Following a functional diet and cultivation of a good microenvironment for gut microbiota, they interact with host immune surveillance systems which reduces risk factors for CVD by reducing inflammation by anti-inflammation driven metabolites, immune boosting molecules, polyunsaturated fats (PUFA), and short-chain fatty acids [40].

In the study led by Kalea A. et al, they illustrated a mode of action in bioactive compounds following intake of a functional diet contributed to epigenetic modification of molecules such as histone acetylation, histone deacetylases or acetyltransferases (e.g., activate deacetylases Sirtuins (SIRTs)), DNA methylation which represented increased CVD risk during metabolic syndromes and disease pathogenesis (i.e., adiposity, inflammation following oxidative stress, atherosclerosis) [41]. It suggests that future direction of risk assessment in CVD could evolve from molecular based evaluation which outcomes may be visualized quantitatively and qualitatively. Impact of diet can be categorized by bioactive compounds and the pattern of nutrients on CVD risk and health promotion. By empowering the preventive effect of diet on personalized care utilizing nutri-genomics, nutri-metaolomics, nutri-epidegenetic, and epigenomics combined with measurement tools in advanced analytical chemistry better treatments and diagnostic tools can be developed.

6. Conclusion

The complexity of CVD heterogeneity suggests that personalized treatments will be required because several risk factors depend on the cause of CVD. As disease determinants, hypertension and hyperlipidemia are two very strong underlying factors that can increase the chances of CVD development. Omics can help develop biomarkers and can be used to develop new targets for drugs. Although, molecular diagnostic depends on the biochemical analysis and traditional imaging techniques, they work to ensure treatment options for patients who are suffering from CVD pathogenesis. Early detection of CVD cannot be clearly measured due to a lack of available biomarkers. Further genomic testing and nutrigenomics will need to be studied in order to develop personalized tests and treatment plans for different genetic predispositions. Differential genes that are altered by epigenetic modification and protein structure conformation can be sequenced from next generation gene sequencers. Protein structure images will unveil interactions between a variety of environmental stressors that are risk factors of interest that could potentially lead to organ damage and CVD progression. Drugs are currently being developed to correct these metabolic pathways as positive endpoints. However, emerging data of diets suggest that it could play a role in systematic modulation to reduce metabolic engagement and detoxification of chronic inflammation.

Perspectives in Public Health

Recent advancements in gut microbial engagement to immune and inflammation have allowed scientists to develop a new avenue of therapeutic value and promising treatments for CVD using personalized diets for prevention and

treatment. Although these alternative treatments have not been proven to work in the short-term, they do show promising results thus far without any side effects. CVD is a public health threat and enhance mortality depend on chronic diseases. When working with genetic information it is important to always protect the privacy of the participants. There should be development a mothod of standardization along with guidance for the elderly population suffering from CVD, who have a different metabolic profile. Lifestyle change along with diet is a form of alternative care for CVD. Through preventative measures such as personalized treatments, specialized assessments, monitoring through nutrigenomics, and sharing with patients through personal devices to support self-management, a new form of prevention and treatment can be devised.

Compliance with ethical standards

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No Conflict of interest.

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