Heart Disease

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There are many factors that can increase your risk of getting heart disease. Some of these factors are out of your control, but many of them can be avoided by choosing to live a healthy lifestyle. Some of the risk factors you cannot control are: Gender, Age, Family History, Obesity, High Cholesterol, Smoking, High Blood Pressure, Diabetes, Other factors.

Heart disease is the leading cause of death. The causes and prevention of heart disease have been studied for years, and new information is emerging. For the last several decades, saturated fat and cholesterol have been thought to be major contributors to coronary artery disease, and therefore people are typically advised to strictly limit these in their diet. However, recent studies are indicating that it may not be wise to strictly limit the intake of dietary saturated fatty acids or replace them with polyunsaturated fatty acids when taking other health conditions into consideration. Depending on a person's genetics, diet may or may not be an important factor in preventing heart disease. Exercise is also beneficial for everyone in preventing heart disease. When considering human development, including the negative effects of heart disease, humans still have a lot to learn about the human body and the interaction of diet, the environment, and genetics. CVDs, diseases of the heart and blood vessels, are the number one cause of death ecumenically. CVDs include diseases of the blood vessels supplying the heart (coronary heart disease), encephalon (cerebrovascular disease), and extremities (peripheral artery disease), in integration to diseases directly affecting the heart (rheumatic heart disease, congenital heart disease), and diseases involving blood clots in the veins (thrombosis, embolism) World Health Organization. Ischemic heart disease (additionally called coronary heart disease) is ranked as the number one cause of ecumenical mortality and incapacitation). Albeit some individuals with CVDs are identified afore a culminating event, others become vigilant of the disease when affected by a heart attack or stroke, caused by restricted blood flow to the heart (heart attack) or encephalon (stroke). Stroke is currently the second-leading cause of death ecumenically). Environmental factors are kenned to affect CVDs, primarily through their linkages with behavioral factors such as physical activity, salubrious diet, and tobacco use, and exposure to tobacco smoke. As the number one cause of death and incapacitation ecumenically, CVDs demand attention and intervention, including efforts to modify environments to increment risk and embolden salubrious comportments.

Most cardiovascular diseases (CVDs) are age-cognate diseases. The prevalence of cardiovascular risk factors increases with age and cardiovascular aging contributes to the development of CVDs. Mounting evidence has demonstrated that dietary restriction (DR), an alimental anti-aging intervention, confers cardiovascular aegis and may decrease morbidity and mortality of CVDs. The mechanisms underlying the benign cardiovascular effects of DR are multifaceted, but recent investigations reveal that DR triggers an active bulwark replication against stress. At the center of this replication are cardiovascular protective signals, which include the mammalian target of rapamycin, AMP-activated kinase, endothelial nitric oxide synthase, and NAD+-dependent deacetylases called sirtuins. Among them, sirtuins play two consequential roles: epigenomic regulation and post-translational modification. However, epigenomic regulation of the cardiovascular system by DR has not been plenarily demystified. In this chapter, we discuss the molecular mechanisms by which DR confers cardiovascular bulwark and the possible involu-

tion of sirtuins in epigenomic regulation.

Cardiovascular disease is considered to be the leading cause of mortality ecumenical. Atherosclerosis is regarded as the pathological process underlying cardiovascular disease and clinically manifested as coronary disease, stroke or peripheral arterial disease. Epidemiological studies revealed that chronic ingestion of inorganic arsenic has incremented the incidence of sundry cardiovascular diseases. Ischemic heart disease is more prevalent. In arsenic-affected groundwater-contaminated areas in Asian countries, more people are suffering from sundry cardiovascular diseases.

One of the clinical outcomes of cardiovascular disorders is peripheral arterial disease. It causes astringent systemic arteriosclerosis as well as dry gangrene. In highly arsenic groundwater-contaminated areas of West Bengal, India, legs had to be amputated of those arsenicosis patients who had gangrene of feet. A unique peripheral arterial disease kenned as 'Blackfoot disease' was mundane in West Bengal. Some arsenic-affected victims in symptoms kindred to Blackfoot disease. It is now considered that chronic arsenic poisoning is an independent risk factor for cardiovascular disease. It is regarded that arsenic-induced cardiovascular disease to human could be resultant of interaction among genetic, alimental, and environmental factors. Albeit it is found that people living in highly arsenic-contaminated areas and exposed to high dose of arsenic are suffering more from adverse cardiovascular effects, effects of low dose is yet to be established. It is withal considered that cardiovascular effects of long-term chronic arsenic exposure could be irreversible. Involution of chronic arsenic exposure with several subclinical disorders in the circulatory system has been documented. Chronic arsenic exposure through imbibing dihydrogen monoxide in West Bengal withal reported the evidence of dose–response relationship between arsenic in imbibing dihydrogen monoxide and prevalence of carotid atherosclerosis. In a cohort and case control studies done in West Bengal, dose–response relationship was found consequential between level of ingested arsenic and ischemic heart disease. An incremented prevalence of hypertension in an epidemiological study in West Bengal in the endemic Blackfoot disease area showed a dose–response relationship with ingested arsenic. A study of blood pressure in arsenic-affected areas of Bangladesh was withal consistent with the West Bengal report. Hypertension and vascular occlusions are considered to be the jeopardy factors for death from ischemic heart disease or other cardiovascular illness. The possible mechanism for arsenic-cognate cardiovascular disease is fortified by biological mechanism. It is considered that arsenic can engender reactive oxygen like hydrogen peroxide and hydroxyl radicals, and can induce alterations of nitric oxide metabolism and endothelial function. Molecular imaging of cardiovascular diseases is of great clinical interest. A felicitous implement for this challenge are radiopharmaceuticals labeled with the positron-emitter 18F that offer the opportunity to noninvasively investigate the cardiovascular physiology and pathophysiology in vivo with the prominent nuclear medicine technology positron emission tomography (PET).