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Review Article

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How Antioxidant Have an Effect on Scavenger Receptors Action

Sepideh Farahiniya

Intensive English Language Institute, University of north Texas

***Corresponding author**

Sepideh Farahiniya, Intensive English Language Institute, University of north Texas.

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Abstract

Today's lifestyle has changed in comparison with the past. Using junk foods and unhealthy foods leads to increasing the risk of heart attacks. Atherosclerosis is an inflammation disease made by many factors that lead to cardiovascular diseases (CVD). Macrophages, endothelial cells, OX-LDL, and some oxidants such as reactive oxygen species (ROS) are the reasons for this transformation, and they have an important role in causing cardiovascular disease. Hyperlipidemia is a major reason for atherosclerosis. Increase in the use of fatty food and fast food can lead to a rise in the risk of atherosclerosis. Macrophages are special cells derived from monocytes and can make plaque which led to increased risk of atherosclerosis. Curcumin is one of important ant inflammation can reduce the risk of CVD. Some studies illustrate that a class SRs, SR-AI and SR-AII, and 2 members of the B class, CD36 and SR-BI, involved in atherosclerosis process. Some studies strongly suggest that oral antioxidants are effective in atherosclerosis treatment, most of them have been unsuccessful except probucol. The aim of this study is illustrating that how curcumin as antioxidant can effect on atherosclerosis proses and scavenger receptor's activation.

Keywords: Atherosclerosis, Macrophages, Curcumin, Scavenger Receptors, Antioxidant, CD36, SR-BI

Today's lifestyle has changed in comparison with the past. Using junk foods and unhealthy foods leads to increasing the risk of heart attacks. Most people suffer from heart diseases. Heart diseases have been big problems since half a century ago, and most people have suffered around the world, including UK, northern Europe and North America [1]. Atherosclerosis is an inflammation disease made by many factors that lead to cardiovascular diseases (CVD). These inflammation diseases are important issues to injure heart vessels in humans [2].

These diseases are also known as low elasticity in all types of arthritis caused by atherosclerosis plaque, and the plaque leads to making hard walls in endothelial cells [3]. Lipids accumulation in endothelial cells can increase the risk of atherosclerosis because it helps to make plaque as a result of risk factors called foam cells. Thus, the development of foam cells leads to an increased heart attack. Foam cell formation is caused by Oxidized low-density lipoprotein (OX-LDL), and its interaction with macrophages. The transformation of macrophages leads to making phagocytes called macrophages. During this process, monocytes pass the wall of the blood vessels, and they enter the tissue. Although atherosclerosis is an inflammation disease, some evidence shows that high level of OX-LDL or hyperlipidemia, ROS, macrophages, foam cells formation, and antioxidants have direct effects on the risk of plaque formation and atherosclerosis.

Macrophages, endothelial cells, OX-LDL, and some oxidants such as reactive oxygen species (ROS) are the reasons for this transformation, and they have an important role in causing cardiovascular disease [2]. Inflammation diseases are usually the reason for this development. Macrophages, endothelial cells, OX-LDL, and some oxidants such as reactive oxygen species (ROS) are the causes of this transformation.

Hyperlipidemia

Hyperlipidemia is a major reason for atherosclerosis. Increase in the use of fatty food and fast food can lead to a rise in the risk of atherosclerosis. Hyperlipidemia usually happens when the level of cholesterols increases in plasma [4]. According to Bergheanu, increasing measure of cholesterols in plasma not only will lead to modification of vessels walls but also can modify the percentage of penetrance in endothelial cells, in most cases LDL-C particles have involved in this process.

Low-density lipoprotein (LDL) is a type of cholesterol called bad cholesterol. This type of cholesterol is the main reason for fatty

deposits that lead to making plaques and increasing the risk of heart attacks (Figure 1).

Presence of LDL in plasma causes expression of some adherence molecules such as vascular adhesion molecule-1 (VCAM-1) and selectins. These molecules can modify the act of monocytes, and they will increase diapedeses. During this process, monocytes by rolling in vessels and in the presence of lipid particles try to move and spread in sub endothelial walls to activate VCAM-1 and selectins. Finally, in this place they will be changed into macrophages. In other words, after leaving the vessels and interring into tissues, monocytes will be changed into macrophages. After interring into sub endothelial layers, LDL particles oxidize, and they will be enormously powerful chemoattractant [4].

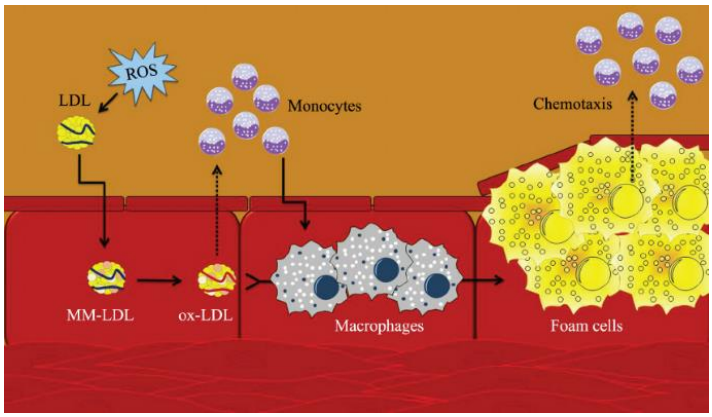

Figure 1: Low-density lipoprotein (LDL) formation [5].

In atherosclerosis process Oxidized low-density lipoproteins (OX-LDL) are a motivation to active macrophages. Activated macrophages lead to starting several signaling cascades that increase the level of some cell products, including proinflammatory cytokines, proteases, and cytotoxic oxygen and nitrogen-free radical molecules. These factors are the major factors in heart attacks [6].

Oxidative stress is main reason for LDL modification. Oxidative stress, which is an extra type of reactive oxygen species (ROS), has basic role in atherosclerosis process, and it can change LDL to make ox-LDL. The main reason for oxidative stress is differentiation of the levels of radical products such as the reactive oxygen and scavenging process [2].

The other type of cholesterol is High-density lipoprotein (HDL) or good cholesterol, which our bodies can remove by the liver. HDL has improved people's health. In addition, it can reduce the risk of heart attacks. The levels of HDL and LDL depend on lifestyle habits. Routine blood tests can control them. As a result, high levels of cholesterol and triglyceride in vessels with different ranges of HDL, LDL, lipoprotein, and their ratio in plasma can lead to heart attacks also [3].

ROS

Some activities usually happen in all cells. Some of them are Chemical and biological activities. Chemical and biological activities and all organisms are effected by oxygen as a known molecule on all events in the world. The process of O2 is necessary for all actions and reactions in both biological situations and decomposition of materials [7].

Reactive O_2 species called (ROS) are important molecules can effect on all chemical and biological activities. Reactive O_2 species (ROS) have high-capacity oxidants. Some studies have shown that the onset of tissue damages Ox-LDL in macrophages and endothelial cells leads to manufacture of ROS and reactive nitrogen species (RNS) [6]. In addition, there are some factors that lead to changing levels of ROS. If white blood cells and platelets are present in blood, blood oxidative stress (BLOS) can lead to raising the levels of ROS [8].

These studies have shown the important effect of ROS on all type of cells. Although high levels of ROS or their presence in the tissue leads to tissue hurt, reactive oxygen species (ROS) are essential parts of various cellular process [9]. ROS can have an effect on cell immunity, and different levels of ROS will cause different levels of immune cell reactions.

Another important molecule is Nuclear factor-κB (NF-κB). Nuclear factor-κB (NF-κB) transcriptional activity is controlled by anti-microbial effectors and signaling molecules made in immunity processes under the influence reaction of ROS in cells [9].

In addition, one organelle that has key role in this process is Mitochondria. Mitochondria and NADPH oxidases (NOXs) are two main origins of cellular ROS. Furthermore, cells react to motivation by making cytokines, chemokines, reactive oxygen species (ROS), and they change the structure of kinds of matter with uniform properties [10]. Some studies have shown that the elimination of antioxidant systems in ApoE−/− mice has an important function for mitochondrial ROS in atherogenic structures [10]. On the other hand, ROS surfaces have controlled by releasing of Nuclear factor-κB (NF-κB) in cell signaling cascades, which are motivated by both antioxidant and prooxidant proteins [10]. The increased cellular levels of antioxidant enzymes by ROS in NF-κB cascade will keep cells safe from harm or injury. In addition, high levels of NF-κB increase the ROS generation in inflammation disease [10]. As a result, atherosclerosis is usually effected by reactive oxygen species (ROS), and both free radical and non-free radical oxygen are part of ROS which have had an important role in atherosclerosis process in abnormality endothelial cells. Furthermore, OX-LDL, and inflammations acts have key role to produce atherosclerosis damage [11].

Macrophages

Macrophages are special cells derived from monocytes. These massive cells have one nucleus, and they are high quality in phagocyte. The exit of monocytes from blood vessels and entering in tissues such as phagocytic reticular cells and histiocytes lead to macrophage formation. Macrophages are part of the reticuloendothelial system. Bone marrow is the main source of macrophages, and the process of making macrophages has three steps. In first step, monoblasts are derived from bone marrow, and in second step promonocytes are derived from monoblasts; eventually promonocytes improve to monocytes. On the other hand, inflammations are motivation for inactive macrophages

to become active macrophages. The macrophage's tasks include phagocytosis, pinocytosis, carrying antigens to deliver T and B cells, and making some products including enzymes, some parts of coagulation factors, prostaglandins, leukotrienes, and some regulative molecules. In addition, inflammation in blood vessels, even in high level decrease of cholesterol, is assumed as an important factor in repeated atherothrombotic attacks. Macrophages have been demonstrated to have key-role in stable inflammatory problems. ASCVD demonstrates lack of success to remove of the inflammatory responses, if apoB (apolipoprotein B), which has included lipoproteins, is maintained in the arterial walls [12]. Organization of atherosclerotic trauma usually is performed at a moderately slow rate during the years, and eventually it will break down blood movement, leading to the clinical appearance of permanent angina or claudication. Studies have emphasized that presence of plaques and lipid-rich lesions for lang time increase levels of all types of CVD. Barrett mentions that lipid accumulation leads to immune system activation and lipid localization, and it improves organization of lipid-rich lesions (2019). These lipid-rich lesions will organize plaques which increases the risk of atherosclerosis. According to Barrett (2019), plaques can appear in any number in vessels walls, and they lead to ASCVD or all types of artery disease.

Scavenger Receptors

Cell surface receptors appear in all type of cell, and macrophages are no exception. There are some receptors called scavenger receptors derived from a large superfamily. These receptors recognize antigens or inflammation factors and leads to increasing macrophages function. Depending on the type of antigens, specific receptors are activated. Scavenger receptors recognize low density lipoproteins (LDL), and they have the capability to prevent and remove them. There are several types and classifications of scavenger receptors, including class A, class B, class E, class F, and class G (Figure 2).

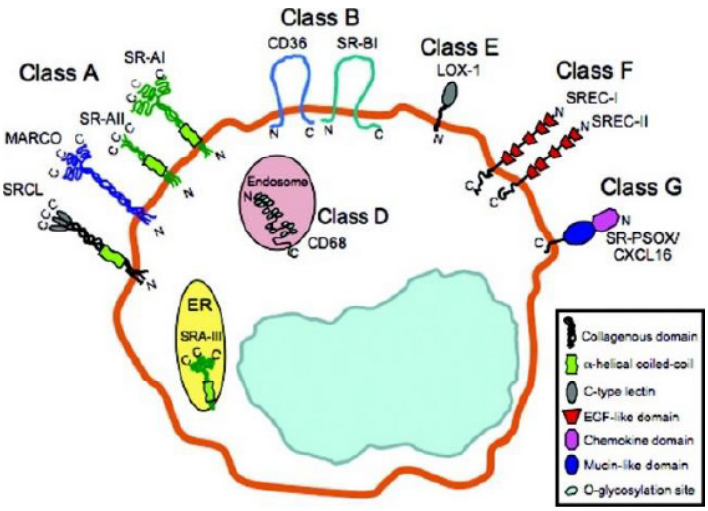

Figure 2: Schematic Representation of Large Family's Scavenger Receptors [13].

Many actions are affected by scavenger receptors such as apoptosis, pathogen clearance, inflammatory diseases and homeostasis. According to Moore (2006), some studies illustrate that a class SRs, SR-AI and SR-AII, and 2 members of the B class, CD36 and SR-BI, involved in atherosclerosis process. Although these arrangements are considered according to SRs ability to join moderate LDL, no research has shown that SRs have not direct effects in atherosclerosis improvement in vivo [13]. As a result, macrophages and scavenger receptors have an important for OX-LDL transportation that leads to heart attack's improvement.

The article reports that "Scavenger receptors expressed on myeloid cells are activated by apoptotic cell debris, TAGs, lipids etc upon activation of these receptors, they induce pro-inflammatory signaling in myeloid cells polarizing anti-inflammatory tissue-resident macrophages to pro-inflammatory phenotype. Constant activation leads to a chronic low-grade inflammation and ultimately to fibrosis" [14]. As a result, scavenger receptors have key role in cholesterol's transport in the atherosclerosis process.

Antioxidants

Although there are some treatments for atherosclerosis, it is necessary that researchers improve alternative treatment strategy for atherosclerosis. The most resent experimental studies have illustrated that organic food and herbal medicine, which have had high levels of antioxidants, have positive effects for atherosclerosis treatments. Although some studies strongly suggest that oral antioxidants are effective in atherosclerosis treatment, most of them have been unsuccessful except probucol [15].

According to the Levonen (2008) "One of the best-characterized protective genes proven to be effective in ameliorating cardiovascular problems associated with increased oxidative stress is heme oxygenase-1 (HO-1). HO-1 is a stress-inducible enzyme that degrades heme to yield biliverdin further metabolized to bilirubin, carbon monoxide (CO), and ferrous iron".

Furthermore, some herbal medicine used as full antioxidant food such as curcumin, niacin, etc. Curcumin is a type of polyphenol and a basic ingredient of natural turmeric Curcuma longa Linn, Turmeric. Curcumin has long been used as spice, but Chinese people have used it as herbal medicine for a long time. Some countries such as Japan have used curcumin to make color for food and sweetie. In addition, Food and Drug Administration (FDA) in United States approve curcumin as a reliable supplement [16]. Some studies illustrated that if people use both medicine and high-level antioxidant supplements, they can control atherosclerosis and heart attacks [17, 18].

Conclusion

Atherosclerosis as an inflammation disease is affected by some risk factors. These factors include high level of OX-LDL or hyperlipidemia, ROS, macrophages, foam cell formation, antioxidants, and plaque formation. Researchers are constantly looking for a way to reduce the range of CVD. It seems that some kinds of herbal medicine, which have had high percentage of oral antioxidant, influence the atherosclerosis process. Some oxidants such as ROS have effects on scavenger receptors, they can change the SRs actions and increase the levels of foam cell formation. The high levels of foam cells in the presence of modified LDL (OX-LDL) lead to raising the chance of atherosclerosis. The use of herbal medicine can be an easy way to reduce the chance of CVD.

It means that antioxidants can control the scavenger receptors in the cell membranes of macrophages. It shows that reduced scavenger receptors activation has positive effects in CVD. It is important that if people want to have health lives, they should use organic food with high levels of antioxidants.

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