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Editorial Open Access

The Dispute of Statins and Cholesterol: Can Statins Save Your Life by Abating the Risk of Heart Disease?

Md. Sahab Uddin[1*] **and Muniruddin Ahmed**[2]

[1]*Department of Pharmacy, Southeast University, Dhaka, Bangladesh*

[2]*Department of Clinical Pharmacy and Pharmacology, Faculty of Pharmacy, University of Dhaka, Dhaka, Bangladesh*

[*]**Corresponding author:** Md. Sahab Uddin, Department of Pharmacy, Southeast University, Dhaka, Bangladesh, Tel: +880 1710220110; E-mail: msu_neuropharma@hotmail.com / msu-neuropharma@hotmail.com

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Editorial

Statins are a class of lipid-lowering medications that inhibit a step used by the liver to synthesize cholesterol [1,2]. Statins abate the health perils linked to atherosclerosis by lowering the level of cholesterol. Atherosclerosis is a cardiovascular disease (CVD) in which cholesterol-loaded plaques build up inside arteries and affecting the blood supply to critical organs [3]. Statins have been found to abate CVD and mortality in persons who are at great menace. The proofs are strong that statins are persuasive for the treatment of CVD in the initial stages of the disease (i.e. secondary prevention) as well as in individuals at augmented risk but without CVD (i.e., primary prevention) [4].

Nowadays a number of statins are available on the market such as atorvastatin, fluvastatin, lovastatin, pitavastatin, pravastatin, rosuvastatin and simvastatin including combination preparations such as ezetimibe/simvastatin [5,6]. Amid statins, atorvastatin also known as Lipitor® manufactured by Pfizer is the best-selling statin. In 2008, Pfizer reported sales of US$12.4 billion making it the topmost-selling branded pharmaceutical in the world [7]. Currently, copious statins became accessible as less costly generic pharmaceuticals, owing to patent expirations.

Generally, elevated low-density lipoprotein (LDL) cholesterol is an accepted peril factor for CVD and the drive of cholesterol-lowering drugs is simply to prevent the incidence of heart disease [8,9]. Statins reduce cholesterol levels by blocking 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase that is involved in the biosynthesis of cholesterol in the liver [10]. Ominously, this is the similar alley in which the body usages to engender coenzyme Q10 (CoQ10). CoQ10 plays a significant role in boosting energy, augments the immune system and acts as significant lipid antioxidants [11]. Studies demonstrated that the blood levels of CoQ10 abated by 40 to 50 percent owing to 1 or 2 months statin therapy and this decline harmfully affects entire system of the body [12]. Greatly impaired organs are those with the utmost energy requirements, comprising the heart. In fact, low level of CoQ10 serves as an inconspicuous clairvoyant of death in patients with CVD [13].

In 2003, Pfizer ran a campaign with print ads that used pictures of a labeled toe of a dead body in France and Canada, [14] given in Figure 1. Pfizer also advertised on television, that owing to a heart attack a young and fit man died hastily, leaving his family mystified with angst. Pfizer must have expended countless millions of dollars on these ads. The note of these two ads was that a person will not expire until the blood cholesterol is under control and if the blood cholesterol is out of control, it will be best to take the statin. But, this was and is a fib, based on existing studies.

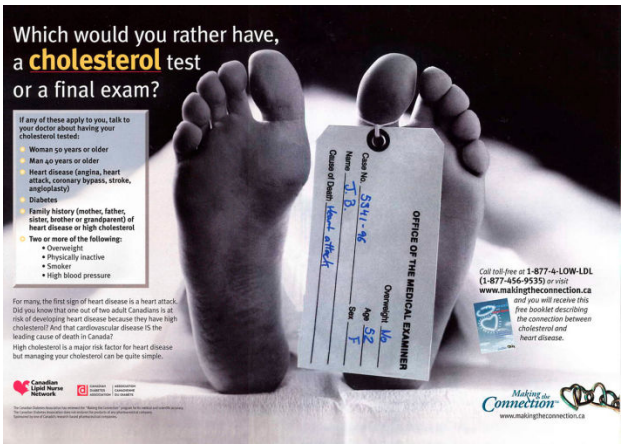

Figure 1: The campaign of heart disease-awareness sponsored by Pfizer, the manufacturer of Lipitor®. The message is clear that the testing of cholesterol, as well as treatment could inhibit premature death from heart attacks in typical people and you will not die as long as your blood cholesterol is low and if it isn't, you had better to take a statin [15].

In 2003 a meta-analysis of cholesterol-lowering drugs (i.e., drug name: atorvastatin, trade name: Lipitor®; drug name: pravastatin, trade name: Pravachol®; drug name: lovastatin, trade name: Mevacor®) in primary prevention reported no variance in mortality amid drug and placebo [16]. Therefore, statins have not been revealed to offer a complete health benefit in primary prevention trials.

Okuyama et al. represented a perspective that statins stimulate atherosclerosis and heart failure [17]. The researchers said that statins stop the biosynthesis of vitamin K2 that serves as a cofactor for the activation of matrix Gla-protein. But to shield arteries from calcification, matrix Gla-protein activation is crucial. The calcification of calcium in arteries is accountable for the inflammation that leads to cholesterol attempts to patch up. Thus cholesterol takes the guilt for blocked arteries caused by statin. Furthermore, statin stops the genesis of selenoprotein such as glutathione peroxidase that serves to prevent peroxidative stress. Disruption in the synthesis of selenium-containing proteins may be a factor in congestive heart failure.

Sultan and Hynes in a comprehensive appraisal stated that there is a scarcity of proof to support the use of statin therapy in primary cardiovascular protection [18]. Moreover, there is abundant proof to display that statins really boost cardiovascular peril in women, patients

with diabetes mellitus and in the young. In fact, statins are linked with triple the peril of coronary artery as well as aortic artery calcification.

DuBroff and de Lorgeril based on critical analysis of existing data related to statins and the Mediterranean diet avowed that the chance that coronary heart disease (CHD) could be stopped by basically decreasing cholesterol seems speculative [19].

The effect of blood cholesterol levels in cardiac mortality and the factual consequence of cholesterol-lowering statin drugs are greatly doubtful. Based on the aforementioned evidences, whether statins really abate CVD and raise life expectancy is exactly contentious. But the epidemic of heart failure and atherosclerosis that plagues the modern world may absurdly be infuriated by the omnipresent use of statins. Therefore, existing statin treatment plans be cautiously reassessed and further studies are prerequisite.

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Competing Interests

The authors proclaim no competing interests.

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