

RESEARCH ARTICLE

The Effect of Vitamin C towards Endothelial Dysfunction in CdCl₂-induced HUVEC Culture

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ABSTRACT:

Cardiovascular diseases are the leading cause of death globally which usually begins with endothelial dysfunction. This vascular abnormality is potentially caused by one kind of heavy metal called cadmium (Cd). Vitamin C, as an antioxidant, can serve as the free radical scavenger. This research aimed to determine the effect of vitamin C on the viability of endothelial cells and endothelial dysfunction through nitric oxide (NO) levels and malondialdehyde (MDA) in human umbilical vein endothelial cells (HUVEC) induced with CdCl₂. HUVEC was induced by 24.154 µg/L CdCl₂. Three treatment groups of CdCl₂-induced HUVEC were employed, each given vitamin C concentrations of 50, 100, and 200 µM, respectively. Cell viability was assessed by MTT assay. Levels of NO and MDA were determined by with the calorimetric test. Cell viability was increased significantly in treatment group receiving vitamin C 100 and 200 µM. However, there were no significant differences on NO and MDA levels in vitamin C treatment groups compared to control. In conclusion, vitamin C increases endothelial cell viability and NO level, and reduces MDA levels in CdCl₂-induced HUVEC. This study implies the importance of vitamin C supplementation to prevent cardiovascular diseases.

KEYWORDS: HUVEC, cadmium, vitamin C, cell viability, NO, MDA.

INTRODUCTION:

Cardiovascular disease is the prominent contributor of deaths worldwide. A cohort study in 2013 discovered the relationship between increasing cadmium (Cd) levels in human body and cardiovascular disease¹. In Sweden, heart failures were also associated with low dose Cd exposure². An average Cd dose of 1.53 µg/L in blood is potentially capable to cause myocardial heart disease and hypertension³. Cd is a toxic heavy metal substance that can easily contaminate human body and environment⁴.

This toxic metal substance enters the human body through inhalation or oral route then accumulated in human body⁵ and causes long term health problems^{6,7}.

Cd indirectly triggers reactive oxygen species (ROS) production by releasing active redox metal, which causes oxidative stress through the fenton reaction^{8,9}.

Vascular disorders are often initiated by endothelial dysfunction. This symptom is characterized with impaired endothelial cell homeostasis, decreased antioxidant levels, and increased antithrombotic and adhesion molecules. Typical signs of endothelial dysfunction are decreased levels of nitric oxide (NO)¹⁰ and increased lipid concentration¹¹.

Malondealdehyde (MDA) is used in this study as the indicator of the lipids peroxydation¹² which affected toward cell and membrant damages¹³ Cd toxicity due to ROS production and oxidant damage generates several