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Research Article

Anti-Inflammatory Activity of Marine Ovothiol A in an *In Vitro* Model of Endothelial Dysfunction Induced by Hyperglycemia

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Chronic hyperglycemia is associated with oxidative stress and vascular inflammation, both leading to endothelial dysfunction and cardiovascular disease that can be weakened by antioxidant/anti-inflammatory molecules in both healthy and diabetic subjects. Among natural molecules, ovothiol A, produced in sea urchin eggs to protect eggs/embryos from the oxidative burst at fertilization and during development, has been receiving increasing interest for its use as an antioxidant. Here, we evaluated the potential antioxidative/anti-inflammatory effect of purified ovothiol A in an *in vitro* cellular model of hyperglycemia-induced endothelial dysfunction employing human umbilical vein endothelial cells (HUVECs) from women affected by gestational diabetes (GD) and from healthy mothers. Ovothiol A was rapidly taken up by both cellular systems, resulting in increased glutathione values in GD-HUVECs, likely due to the formation of reduced ovothiol A. In tumor necrosis factor-α-stimulated cells, ovothiol A induced a downregulation of adhesion molecule expression and decrease in monocyte-HUVEC interaction. This was associated with a reduction in reactive oxygen and nitrogen species and an increase in nitric oxide bioavailability. These results point to the potential antiatherogenic properties of the natural antioxidant ovothiol A and support its therapeutic potential in pathologies related to cardiovascular diseases associated with oxidative/inflammatory stress and endothelial dysfunction.

1. Introduction

One of the major challenges of the recent research in biomedicine is the discovery of new natural products to develop drugs and dietary supplements that could prevent and relieve pathologies associated with chronic low-grade inflammation and oxidative stress. Among these, diabetes is one of the most widespread. It is associated with oxidative stress and vascular chronic inflammation, alterations underlying the development of cardiovascular disease [1]. In particular, endothelial dysfunction is associated with vascular disease occurrence and is characterized by an increased expression of endothelial adhesion molecules and the recruitment of monocytes to the intima, a pivotal and critical event in promoting atherosclerosis [1, 2]. Nitric oxide (NO), constitutively generated by endothelial cells, plays a key role in the maintenance of vascular homeostasis through the reduction of proinflammatory response that characterizes the early stages of atherosclerosis, especially during chronic hyperglycemia [3]. The preservation of endothelial NO bioavailability, leading to increased vascular cGMP levels, is therefore considered beneficial to endothelial functions and more in general to vascular health.