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Perivascular Adipose Tissue can be Considered a Risk Factor for Atherosclerosis?

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Introduction

Perivascular adipose tissue (PVAT) is an ectopic deposition of adipose tissue surrounding the vasculature and your influence on the vasculature changes with increasing adiposity. PVAT involves coronary arteries, aorta, mesenteric, and small arteries in the body, and its likely function differs in each of these anatomical regions [1-4].

PVAT secretes a wide variety of adipocytokines and other substances, including hormones, cytokines, chemokines, oxygen radicals, angiotensinogen, leptin, resistin and fatty acids [5]. The rate of secretion of various adipocytokines varies in different places in the vascular tree, adipocytokines as TNF-α, IL-6 and others. Adiponectin can affect insulin sensitivity, inflammatory responses, hemostasis, appetite and atherosclerosis [6-10]. The factors secreted by PVAT that act in the regulation of vascular function are presented in Table 1.

Cytokines/Chemokines	Vasoactive agents	Hormones and Fatty acids
IL-6 IL-8	H_2S C_3	Adiponectin Visfatin
IL-10 IL-1β	ADRF NO	Leptin Oestrogen
IL-1β MCP-1	Ang (1–7) Ang II	FFA Androgen
TNFα MIP-1α	ROS H_2O_2	Resistin HGF
MIF RANTES	Angiotensinogen	FABP4 Adrenomedullin Glucocorticoids
PAI-1 HB-EGF	Methyl-palmitate	

Table 1: Product of PVAT involved in the regulation of vascular function.
IL: interleukin-; MCP-1: Monocyte Chemoattractant Protein-1; MIP-1α: Macrophage Inflammatory Protein-1α; ROS: Reactive Oxygen Species; NO: Nitric Oxide; TNFα: Tumor Necrosis Factor α; Ang II: Angiotensin II; Ang (1–7): Angiotensin (1–7); FABP4: Fatty Acid-Binding Protein 4; H_2S: Hydrogen Sulphate; FFA: Free Fatty Acids; MIF: Macrophage Migration Inhibitory Factor; HB-EGF: heparin-Binding Epidermal Growth Factor-Like Growth Factor; ROS: Reactive Oxygen Species; C3: Complement 3; HGF: Hepatic Growth Factor; H_2O_2: Hydrogen Peroxide; ADRF: Adipocyte-Derived Relaxing; RANTES: Regulated on Activation, Normal T Cell Expressed and Secreted.

PVAT is related to the vascular contractility, endothelial dysfunction, neointima formation, arterial stiffness, aneurysm formation, and produce substances that can interfere in the process of atherosclerosis and contribute to the pathogenesis of type 2 diabetes and cardiovascular diseases [11-16].

An understanding of the pathophysiology of PVAT and its potential role in cardiovascular morbidity and mortality can be significant in preventing and treating of atherosclerosis.

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