Obesity and Relative Significant Risk Factor for Atherosclerosis

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and adiponectin, govern fat metabolism, energy balance, and insulin sensitivity, and hence in uence obesity-related metabolic diseases. Because of their in uence on the activity of endothelial cells, arterial smooth muscle cells, and macrophages in vessel walls, several adipokines have lately been viewed as direct connections between obesity and atherosclerosis, regardless of their e ects on glucose and fat metabolism. e discovery of a novel adipokine that controls the atherosclerotic process might open up new avenues for creating more e ective cardiovascular disease prevention strategies [1]. Adipokines that mediate obesity and atherosclerosis, such as adiponectin, resistin, adipocyte fatty acid binding protein (A-FABP), omentin-1, and chemerin, will be discussed in this study.

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S e c a a :

As people gain weight and their adipocytes get larger, the adipose tissue undergoes molecular and cellular changes that impact systemic metabolism. To begin, macrophages build up in fat tissue, causing local in ammation. As obesity rises, adipose tissue produces a number of proin ammatory mediators. Obese people's adipose tissue expresses more proin ammatory proteins, such as TNF- and IL-6, than lean people's adipose tissue. Obesity increases the amount of macrophages in adipose tissue, which appear to serve as scavengers of apoptotic adipocytes. Obese people have also been shown to have a signi cant increase in these scavengers [2, 3]. Numerous metabolic dysfunctions associated with obesity, such as systemic in ammation and atherosclerosis, are thought to be caused by macrophage accumulation and consequent local in ammation.

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