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Fatty Acid Binding Protein 4 And Fat Metabolic Markers Reacted To Human Catecholamines

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Introduction

Fatty acid binding protein 4 (FABP4, also referred to as adipocyte FABP or aP2) may be a member of the cytosolic carboxylic acid binding protein family and highly expressed in adipocytes and macrophages [1]. FABPs bind to hydrophobic ligands like long chain fatty acids (FA) with high affinity. it's been proposed that biological function of FABPs is trafficking of FA to subcellular compartments. Clinical and animal-based studies have demonstrated that FABP4 has a crucial role in obesity-related metabolic diseases like insulin resistance, type 2 diabetes, hepatosteatosis and atherosclerosis. Recently, FABP4 has also been introduced as a fat-derived circulating protein. Serum FABP4 levels are strongly correlated with adiposity while FABP4 is eliminated from the circulation mainly by renal clearance, Secretion of FABP4 from adipocytes is enhanced by lipolysis, which is especially activated by catecholamines during activation of sympathetic systema nervosum (SNS). Catecholamines stimulate β1/2/3-adrenergic receptor-mediated adenyl cyclase-protein kinase A (βAR/AC/PKA) pathway, [2,3]which successively promotes lipolysis via activation of several cytosolic lipases . Emerging evidence has demonstrated that serum FABP4 levels are positively correlated with markers of the metabolic syndrome and vascular diseases which a rise in serum levels of FABP4 at baseline predicts the danger for metabolic and vascular morbidity and mortality These findings suggest that circulating FABP4 derived from adipocytes may be a useful biomarker to estimate current status of cardiometabolic diseases and predict their incidence within the future. We recently found that serum FABP4 concentrations also are dynamically regulated during early phase of acute myocardial infarct (AMI). In patients with AMI, serum FABP4 concentrations peaked on admission or simply after percutaneous coronary intervention and declined thereafter. Of note, FABP4 concentrations were particularly elevated in patients with AMI resuscitated from [4], out-of-hospital asystole (median 130.2 ng/ml, interquartile range compared with those without (median 26.1 ng/ml, interquartile range., along side previous notion that ischemia and lethal arrhythmia are strongly related to activation of SNS via elevated levels of epinephrine and norepinephrine, these findings allowed us to presume a scenario that severe acute cardiac events induce robust activation of SNS which successively promotes lipolysis of TG in adipocytes, resulting in rapid and dynamic secretion of FABP4 into circulation.[5,6] To our knowledge, however, the hypothesis of direct link between dynamic change of circulating FABP4 and activation of SNS in physiological situations has not been investigated a minimum of in human populations

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