Pancreat Disord Ther 2017, 7:4(Suppl)
DOI: 10.4172/2165-7092-C1-009

MECHANISM OF VASCULAR ADIPONECTIN RESISTANCE IN PRE-RECEPTOR LEVEL IN EARLY PHASE OF HYPERLIPIDEMIA

Rong Li[a]
[a]Fourth Military Medical University, China

With the recent research, people discovered there are reduced protective effects of APN though raised APN level in metabolic syndrome which is called APN resistance. APN resistance exists in different tissues that blunted protective effects of APN, We still know little about the mechanism of the progress of APN resistance. Our group found that in early phase of hyperlipidemia the phosphorylation level of AMPK did not rise to exert cardiovascular protective effect as APN content was significantly increased, indicating that APN resistance exist in hyperlipidemia. Further research showed that hyperlipidemic plasma inhibited the ability of APN in phosphorylation of AMPK and activation of eNOS. We assume that some vascular active factors in hyperlipidemia plasm could depress APN activity and lead to APN resistance. To illuminate which vascular active factor could inhibit APN activity would lead us to understand the mechanism of APN resistance and to improve endothelial function in a better approach. Twenty male Sprague Dawley rats were divided equally into two groups at random, receive regular diet or high-fat diet for 8 weeks, then circulating triglyceride, total cholesterol, high-density lipoprotein, low-density lipoprotein was determined. Plasma from regular diet and high-fat diet rats was applied co-immunoprecipitation with anti-APN antibody then followed with mass spectrometric analysis to determine which vascular activity factors can combine with APN. Pre-incubated rAPN with vascular activity factors which discovered in former step and observed the AMPK activation effect of rAPN in HUVECs. Compared with rats fed with regular diet animals fed with high-fat diet circulating triglyceride, total cholesterol, high-density lipoprotein, low-density lipoprotein and APN levels obviously raised after 8 weeks. Discovered APOA1, APOC1, PON1 along with other fifteen vascular activity factors can combine APN and significantly increased in hyperlipidemia plasma. Among the eighteen vascular activity factors APOA1 significantly increased in hyperlipidemia plasma, APOA1 and APOC1 can significantly inhibit the ability of rAPN to phosphorylate AMPK in HUVECs. Increased apolipoprotein that can combine and inhibit the activity of APN in hyperlipidemic plasma are likely responsible for vascular APN resistance in early phase hyperlipidemia.