

Diabetes, Obesity and Atherosclerosis: Three **Buds of One Stem MS-X**

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Abstract

Metabolic Syndrome-X (MS-X) represents a medleybolome of several metabolic disorders from the angles of physiological, biochemical, clinical and metabolic anarchy. Diabetes, obesity and hypertension are the major medico-clinical concerns in 21st century both for developed and developing world. A great number of reports in last decades and recent past from various angles to control the pathology of MS-X have been published [1-9]. Exclusively, catching of diabetes in obese subjects is a unique feature in this disturbed metabolome. In human obesity the insulin resistance and type-2 diabetes are most often inter-linked. The feedback regulatory mechanism of hyper leptinemia is failed in human obesity [10,11]. On the other hand, hyperleptinemia develops insulin resistance. The (Figure 1) shows the metabolic interlink and dependency that reels between insulin and leptin.

Keywords: Diabetes; Metabolic syndrome; Insulin; Atherosclerosis

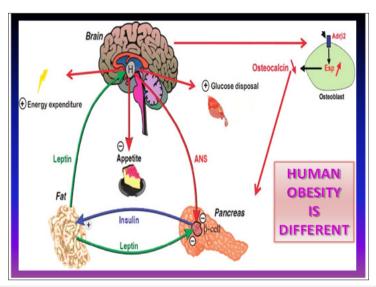
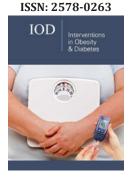


Figure 1: Metabolic interlink and dependency that reels between insulin and leptin.



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Introduction

Ideally insulin and leptin signaling share a common route and ditch each other depending on their surge in their affected physiological state. As a result, insulin and leptin resistances are simultaneous phenomena and no matter to induce other while the one is precipitated. Thus, the preponderance of running obesity and type-2 diabetes hand to hand is deeply expected. The (Figure 2) shows the common signaling road of both the ligands. Report shows that obesity reduces tyrosine phosphorylation of insulin receptor [12]. This affects insulin signaling and hence initiates insulin resistance. On the other hand, report also shows an association of insulin and LDL receptors [12-15] and this association keeps LDL receptor non-functional [12-15] making the system prone to vessel LDL accumulation. Yadav et al. has shown a decrease of LDL receptor mRNA level with increase of leptin concentration as well as two receptor association [14]. Repressed LDL receptor will reduce LDL clearance from blood vessels and the receptor-complex will keep receptors non-functional with concomitant

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rise of LDL in blood vessels. Increase of LDL in blood vessels will initiate hyperlipoproteinemia related atherosclerotic propensity which can also drag hypertension and cardiovascular impairments

[16,17]. Thus, these three metabolic disorders viz. diabetes, obesity and atherosclerosis are the outcomes of impaired metabolic stem.

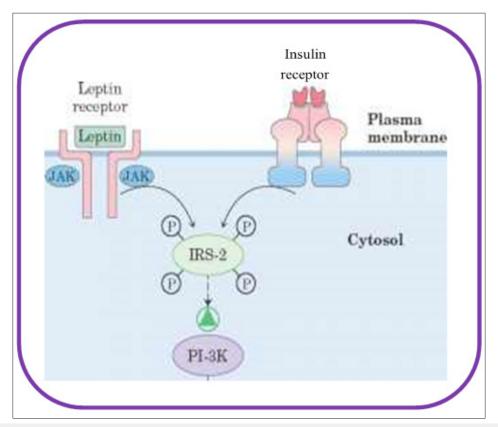


Figure 2: The common signaling road of both the ligands.

Conclusion

All these three metabolic disorders viz. diabetes, obesity and atherosclerosis have an impact on developing stress. This is mediated through another receptor known as oxidized-LDL-receptor-1 or LOX-1. Deposited LDL in the blood vessel will be spontaneously oxidized and the oxLDL (oxidized LDL) generated, will induce a vicious cycle for generating more and more proinflammatory molecules through interaction with its cognate receptor LOX-1 [18,19]. These pro-inflammatory molecules are stress generators and atherosclerotic profounder(s). So, this chain of impairments of multiple pathogenesis with initial induction of one metabolic disorder is a link process to create a medleybolome and ostensibly to call Metabolic-X-disease. So one should keep in mind that prevention is better than cure, once got entrapped by 'Syndrome X'. Among many others, aging is a preponderance factor for precipitating Metabolic-X-syndrome.

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