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## **Essential Hypertension in Immunoreactive Leptin Level**

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## **Commentary**

Insulin resistance, the foremost important think about metabolic syndrome X, has been considered to boost vital sign. Recently it was reported that insulin resistance was related to an elevated plasma level of leptin, which is an adipocyte specific of gene product and which plays a role in food intake suppression, thermogenesis, and energy expenditure through the activation of the hypothalamus. However there are no reports that deal with the relationship of insulin resistance to plasma leptin and blood pressure.

Metabolic syndrome X1 is the theory that glucose intolerance, hyper insulinemia, increased very low density lipoprotein triglyceride level, decreased high density lipoprotein cholesterol level, and hypertension are proposed consequences of insulin resistance. These metabolic disturbances have been shown to increase the risk of coronary artery disease. In this theory, insulin resistance and therefore the resultant hyperinsulinemia are considered to boost vital sign through sympathetic system nervous activation, renal sodium retention, renin-angiotensin system stimulation, and intracellular calcium accumulation in vascular smooth muscle. Indeed, metabolic disturbance and insulin resistance are acknowledged in essential hypertensive. Leptin is a recently discovered hormone produced by an adipocyte-specific of gene that contributes to the regulation of energy balance by informing the hypothalamus of the amount of adipose tissue in the body. As a result, the hypothalamus adjusts food intake, thermogenesis, and energy expenditure appropriately.

Thus, leptin could play a role in the pathophysiology of insulin-resistant hypertension, although there is no report concerning its relationship with

plasma leptin and blood pressure. In this study, we attempted to clarify the relationship between insulin resistance, hypertension, and plasma leptin level.

It is known that plasma leptin levels are highly correlated with BMI and that its levels are significantly higher in women than in men. The present study demonstrated that p-leptin is higher in essential hypertensive than in normotensive controls, even when age, gender, and BMI were not significantly different between the two groups. Furthermore, p-leptin was well correlated with vital sign, although the chronic effect of leptin itself on vital sign in humans remains unknown. The initial aim of this study was to clarify the connection between insulin resistance, hypertension, and p-leptin, because insulin resistance, the key factor of metabolic syndrome X, 1 has been reported to be closely associated with hyperleptinemia. Obese subjects, among whom prevalence of hypertension is higher than in the lean population, were insulin resistant and revealed to be hyperleptinemia. Therefore, we hypothesized that hyperleptinemia might be related to insulin resistance, resulting in blood pressure elevation.

A limitation of our study is that we did not measure percent body fat in the subjects even though BMI were carefully matched. Because plasma leptin levels are closely correlated with percent body fat, further study of percent body fat, subcutaneous and visceral fat ratio, and body fat composition in normotensives and hypertensive is required. In summary, essential hypertensive was found to be not only insulin resistant, but also hyperleptinemia. Furthermore, plasma immunoreactive leptin was correlated to blood pressure. From these results, it is suggested that leptin may play a role in a pathophysiology of essential hypertension.

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