Abstract

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Insulin and the vasculature--old actors, new roles.

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BACKGROUND: I have presented data to support an interaction of insulin with the vascular endothelium to cause the release of NO and vasodilation. In effect, insulin-mediated vasodilation can be viewed as a novel test of endothelial function. The physiologic significance of insulin-mediated, endothelium-dependent vasodilation is not fully understood, but some insight has been gained.

DISCUSSION: Insulin-mediated vasodilation in skeletal muscle appears to be an important mechanism to amplify insulin's overall action to stimulate skeletal muscle glucose uptake in insulin-sensitive man. Insulin-resistant states of obesity, hypertension, and NIDDM exhibit blunted insulin-mediated vasodilation and impaired endothelium-dependent vasodilation.

SUMMARY: Together, the data suggest that endothelial dysfunction is an integral aspect of the syndrome of insulin resistance, independent of hyperglycemia, and as such may contribute to worsen insulin resistance, increase vascular reactivity, 32 and predispose to macrovascular disease. Thus, the insulin/EDNO interaction provides a potential mechanism (but certainly not the only one) linking insulin resistance, hypertension, thrombosis, and atherosclerosis-key features of the insulin resistance syndrome (Figure 7).

CONCLUSION: Future research into the signal transduction steps leading to insulin-mediated, endothelium-derived NO production and its dysregulation in insulin-resistant states will be extremely useful to elucidate the mechanisms underlying the increased macrovascular risk in insulin-resistant man.

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