A novel insulin accelerant

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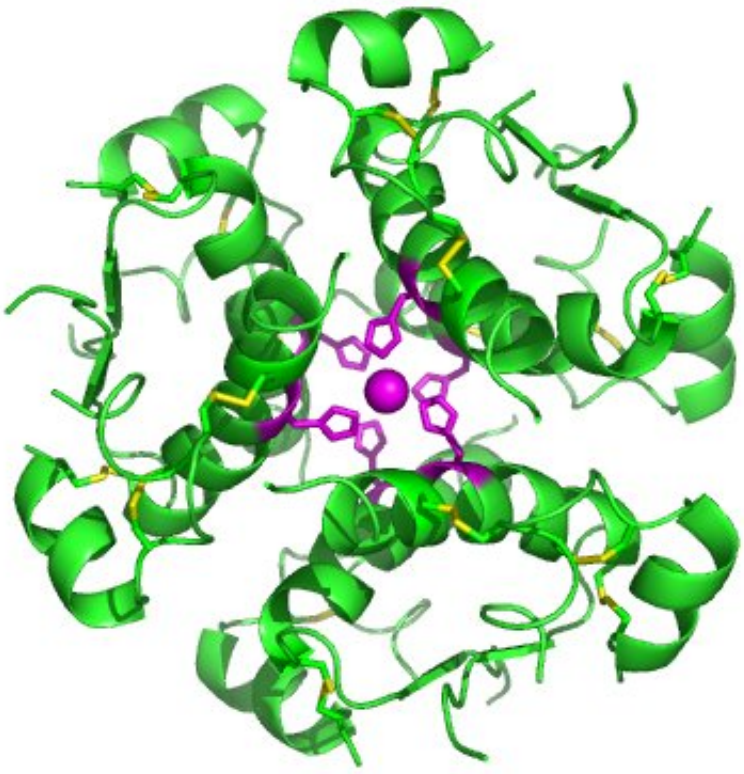

High-resolution model of six insulin molecules assembled in a hexamer. Credit: Isaac Yonemoto/Wikipedia

Insulin levels rise after eating a meal, signaling uptake of circulating glucose by skeletal muscle. In individuals with diabetes this process is often impaired—a condition known as insulin resistance.

Insulin must travel across the endothelium or inner lining of small blood vessels to reach skeletal muscle and induce glucose uptake. Nitric oxide (NO) is a key regulator of endothelial function. It stimulates arterial vasodilation, which increases the surface area available for insulin exchange.

This month in the journal *Diabetes*, David Wasserman, Ph.D., Ian Williams, Ph.D., and colleagues report that when they reduced NO levels in mice by blocking the enzyme NO synthase with a compound called L-NAME, the movement of insulin across the endothelium accelerated. So did insulin-stimulated lowering of blood glucose.

They concluded that acute pharmacological inhibition of NO synthase increases the permeability of capillaries to insulin and accelerates insulin action, counteracting other acute effects that decrease capillary blood flow. It thus may represent a new mechanism to treat insulin resistance.

More information: Ian M. Williams et al. Acute Nitric Oxide Synthase Inhibition Accelerates Transendothelial Insulin Efflux In Vivo, *Diabetes* (2018). DOI: 10.2337/db18-0288

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