
Abstract
Compounds of the trace element vanadium exert various insulin-like effects in *in vitro* and *in vivo* systems. These include their ability to improve glucose homeostasis and insulin resistance in animal models of Type 1 and Type 2 diabetes mellitus. In addition to animal studies, several reports have documented improvements in liver and muscle insulin sensitivity in a limited number of patients with Type 2 diabetes. These effects are, however, not as dramatic as those observed in animal experiments, probably because lower doses of vanadium were used and the duration of therapy was short in human studies as compared with animal work. The ability of these compounds to stimulate glucose uptake, glycogen and lipid synthesis in muscle, adipose and hepatic tissues and to inhibit gluconeogenesis, and the activities of the gluconeogenic enzymes: phosphoenol pyruvate carboxykinase and glucose-6-phosphatase in the liver and kidney as well as lipolysis in fat cells contributes as potential mechanisms to their antidiabetic insulin-like effects. At the cellular level, vanadium activates several key elements of the insulin signal transduction pathway, such as the tyrosine phosphorylation of insulin receptor substrate-1, and extracellular signal-regulated kinase 1 and 2, phosphatidylinositol 3-kinase and protein kinase B activation. These pathways are believed to mediate the metabolic actions of insulin. Because protein tyrosine phosphatases (PTPases) are considered to be negative regulators of the insulin-signalling pathway, it is suggested that vanadium can enhance insulin signalling and action by virtue of its capacity to inhibit PTPase activity and increase tyrosine phosphorylation of substrate proteins. There are some concerns about the potential toxicity of available inorganic vanadium salts at higher doses and during long-term therapy. Therefore, new organovanadium compounds with higher potency and less toxicity need to be evaluated for their efficacy as potential treatment of human diabetes.