

Abstract

Glycolysis in bloodstream *T. brucei* is the sole source of energy and remains a favourable chemotherapeutic target. In furtherance of this, an attempt has been made to understand better the contribution of glucose, fructose, mannose and glycerol to the energy charge of these parasites incubated in the presence of oligomycin, salicyhydroxamic acid (SHAM) and digitonin. Their cellular energy charge, when catabolizing glucose was 0.860, and under inhibition by oligomycin (10 microg), SHAM (2 mM) or oligomycin plus SHAM, 0.800, 0.444 and 0.405, respectively. Oligomycin inhibited the rate of catabolism of glucose, mannose and fructose up to 80%. The inhibition could not be alleviated by uncouplers, such as 2,4-dinitrophenol or permeabilization of the membranes by digitonin. Glucose-6-phosphate and other phosphorylated glycolytic intermediates, such as fructose-6-phosphate were catabolized by the permeabilized parasites in the presence of oligomycin, implying that except hexokinase, all the other glycolytic enzymes were active. Glucose oxidation was stimulated by low concentrations of digitonin (up to 4 microg), but at higher concentrations, it was significantly inhibited (up to 90% inhibition at 10 microg). Apparently, the inhibitory effects of oligomycin and digitonin were confined to glucose uptake and hexokinase catalysis. The above observations suggest that the hexose transporter and the enzyme hexokinase might be functionally-linked in the glycosomal membrane and oligomycin inhibits the linkage, by using a mechanism not linked to the energy charge of the cell. Digitonin at concentrations higher than 4 microg disrupted the membrane, rendering the complex in-operative. A hexokinase/hexose transporter complex in the glycosomal membrane is envisaged.