PHOSPHOFRUCTOKINASE AND THE PASTEUR EFFECT*

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Several recent studies, based on measurement of substrate levels, indicate that the enzyme reaction primarily responsible for the Pasteur effect is phosphofructokinase (PFK) in yeast (1), ascites tumor cells (2), heart (3), and diaphragm (4). The last confirms earlier less clearcut evidence for skeletal muscle (5). The rapid increase in fructose diphosphate (FDP) following onset of ischemia in brain (6,7) may be interpreted in the same way, and this is substantiated by in vitro studies with supernatant fluid from brain homogenates supplemented with liver mitochondria (8,9).

Bücher (10) concludes that in insect wing muscles PFK is activated when the metabolism is increased during muscular activity. He stated the PFK could be activated in vitro by changes in concentrations of ATP, Mg and fructose-6-P (F6P).

Mansour and Menard found that glycolysis in liver flukes is controlled by PFK (11). When glycolysis was stimulated by serotonin or 3',5'-cycloadenylate (3',5'-AMP), the concentrations of glucose-6-P (G6P) and F6P fell and FDP rose (12). In addition it was shown that partially purified PFK could be activated in vitro by ATP, Mg and 3',5'-AMP, and that activation was characterized by decrease in the concentration of F6P required for activity in the presence of high levels of ATP (13). Dr. Mansour has recently found a similar phenomenon in heart muscle (personal communication).

In studies to be reported elsewhere, every member of the glycolytic cycle was measured in mouse brain at short intervals after decapitation.

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