

# Prior heat stress induces moderation of diabetic alterations in glycogen metabolism of rats

## Research Article

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Received 03 June 2013; Accepted 07 September 2013

**Abstract:** Adaptation to one environmental stressor sometimes provides protection against additional, more intensive type of stress, a phenomenon called cross-tolerance. We aimed to estimate the protection provided by acute heat stress (AHS) over carbohydrate disturbances in streptozotocin-diabetic rats. We investigated changes in activity of some hepatic glycolytic and gluconeogenic enzymes, and concentration of some substrates in control and diabetic animals exposed to AHS ( $41 \pm 0.5^\circ\text{C}$  / 1 h), with 1 h and 24 h recovery at room temperature before sacrifice or induction of streptozotocin (STZ)-diabetes, respectively. AHS with 1 h-recovery before sacrifice resulted in intensive glycogenolysis, directed to endogenous glucose production and further utilization of glucose by peripheral tissues, while 24 h recovery resulted in a slight tendency towards normalization of metabolic disturbances caused by AHS. Experimental diabetes caused a significant decrease of substrates and glycolytic enzymes, but an increase of gluconeogenic enzymes. In diabetic animals previously exposed to AHS we measured a less intensive decrease of liver glycogen and glucose-6-phosphate concentration and hexokinase activity, as well as less intensive increase of liver glucose concentration, glucose-6-phosphatase and fructose-1,6-bisphosphatase activity compared to control diabetic animals that had been maintained at room temperature. Prior AHS provided some protection over diabetes-induced alterations in carbohydrate-related parameters (see graphical abstract), indicating a possible development of cross-tolerance phenomenon between the two stressors, AHS and STZ-diabetes.

**Keywords:** Acute heat stress • Experimental diabetes • Cross-tolerance • Carbohydrate metabolism • Liver • Rats

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## Abbreviations:

STZ	– streptozotocin
G6P	– glucose-6-phosphate
G6P-ase	– glucose-6-phosphatase
F1,6BP-ase	– fructose-1,6-bisphosphatase
HK	– hexokinase
PFK	– phosphofructokinase
NAD	– nicotinamide dinucleotide phosphate
PARP	– poly(ADP) ribose polymerase
AHS	– acute heat stress
HSP	– heat shock proteins
PEPCK	– phosphoenolpyruvate carboxykinase

## 1. Introduction

An important beneficial effect of heat exposure is that adjusting to such an environmental stress can, in addition to evolving primary thermal preconditioning, add to the amount of adjustment to additional stress, which otherwise will be lethal, the so called “cross-tolerance” phenomenon [1-4]. Heat acclimation has been shown to have cross-tolerance effect through providing protection to organisms with a variety of conditions with impaired oxygen supply or oxygen demand ratios [2-4].

In our previous work [5,6] we have found existence of cross-tolerance effects between heat acclimation (30 days at  $35 \pm 1^\circ\text{C}$ ) as a moderate physiological stress

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