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Metabolic Consequence of Short Term Starvation in Gastrointestinal Diseases: A Mini Review

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Abstract

The metabolic consequences of preoperative starvation and carbohydrate loading and mechanisms linking insulin resistance to impaired mitochondrial function are discussed. We just find out the short-term energy deprivation combinations with the health control subject. Followed by the nutrition and metabolism on molecular basis of per operative surgery. Majority of animal studies have shown that short-term energy deprivation decreases mitochondrial ATP synthesis capacity and complex activity, and increases oxidative injury which lead to development of insulin resistance (IR) during starvation and associated with an impaired mitochondrial function. From animal to human subject we thoroughly find out that short-term starvation (STS) causes mitochondrial dysfunction and it underlies the development of insulin resistance (IR) in patients undergoing elective surgery (ES).

Keywords: Digestive tract; Histology; Ontogenetic development; Starvation

Introduction

Effects of starvation were mainly evident on the degeneration of cells in digestive organs as seen in the shrinkage and separation of cells [1]. The loss of intercellular substances in the liver, pancreas, intestine and stomach changes became more severe with increased duration of starvation. In addition, the histological structure of the digestive tracts of starved larvae and juveniles partly recovered after re-feeding, and the effects of starvation on miiuy croaker were age dependent [2].

Material and Methods

We just find out the short-term energy deprivation combinations with the health control subject. Followed by the nutrition and metabolism on molecular basis of per operative surgery. In a mean while we justify that these statement as per our twenty years of the research experience.

Results and Discussion

The combined treatment profoundly impairs cancer glucose metabolism and virtually abolishes lesion growth in experimental models of breast and colon carcinoma suggest that energy metabolism (EM) is more promising target to reduce cancer progression [3]. However, short term starvation is the medical condition of congenital hypertrophic pyloric stenosis. Starvation resulting in reduction of more than 15% of the expected body weight in infancy was associated with poorer learning abilities, especially those involving short-term memory and attention [4]. As a total parenteral nutrition prevents patients with short bowel syndrome from dying of starvation, having short bowel remains a severely debilitating condition, this therapy is associated with significant morbidity and patients suffer from consequences of long-term immunosuppression. Now new frontier in medicine in the field of tissue engineering used an autologous tissue as a patch to study intestinal regeneration by consists of seeding a resorbable scaffold and implanting. Researcher construct the regeneration of neointestine is able to creation of esophagus, stomach, small bowel and colon has been demonstrated. We suggest that tissue-engineered intestine become a real therapeutic option in the not too distant future for patients having inadequate intestinal tissue [4].

An Ulcerative colitis be classified as a nutritional colitis in that colonic epithelial cells are unable to utilize SCFAs reflecting epithelial starvation despite abundant SCFAs. Additionally, increased succinate/Complex II-dependent O [2] consumption and elevated oxidative stress (OS) and apoptosis indicated that the glucose and amino acid deficiency conditions imposed through the STS promote an anti-Warburg effect. It increased oxygen consumption but failure to generate ATP and resulting

in oxidative damage and apoptosis [5]. The model of animal to human subject we thoroughly find out that short-term starvation (STS) causes mitochondrial dysfunction and it underlies the development of insulin resistance (IR) in patients who undergoing elective surgery (ES) As well.

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