Keto Lean Call #1

Homework

Article: Insulin, glucose and beta-hydroxybutyrate responses to a medium-chain triglyceride-based sports supplement: A pilot study

Apps:

Zero

Insight Meditation Timer

Wearable(s):

CGM

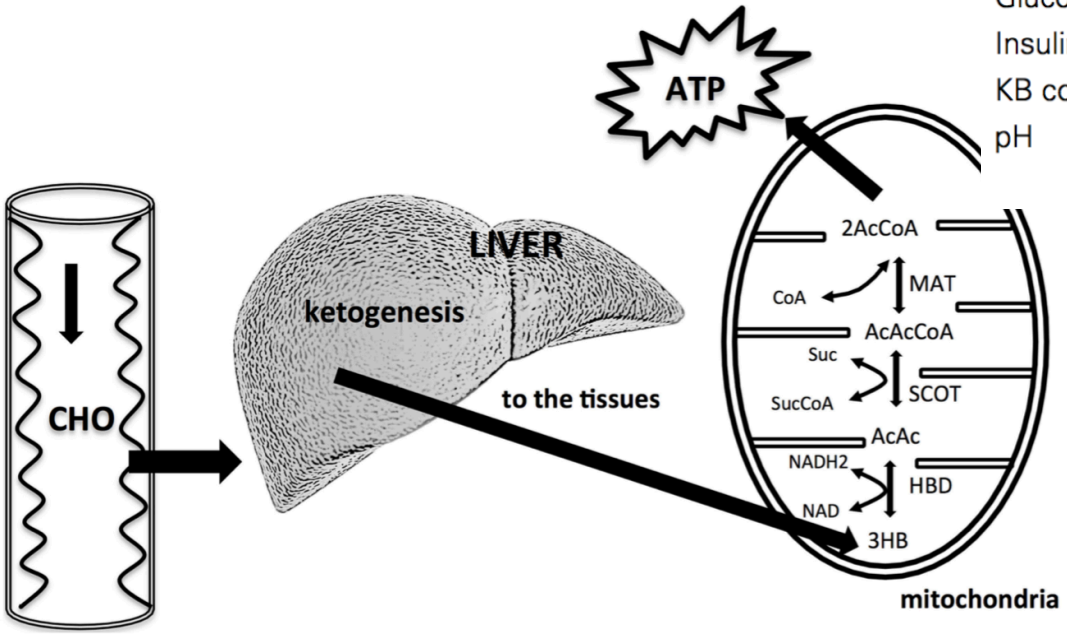
Glucometer

Review

Basics: liver produces ketone bodies: (Beta-hydroxyburutrate, acetoacetate and acetone) from fatty acids

Table 1 | Blood levels during a normal diet, ketogenic diet, and diabetic ketoacidosis (Paoli et al., 2012).

Blood levels	Normal diet	Ketogenic diet	Diabetic ketoacidosis
Glucose (mg/dL)	80–120	65–80	>300
Insulin (μU/L)	6–23	6.6–9.4	≅ 0
KB conc (mmol/L)	0.1	7–8	>25
pH	7.4	7.4	<7.3



Longevity pathways regulated by ketones and CR The crosstalk

Factors Increase/Decrease Ketosis

	Effect on ketogenesis	Mechanisms of action
Environmental factors		
Low carbohydrate diet	↑	Low insulin, leptin
Low calorie diet	↑	Low insulin, leptin, T3 High IGF-I, ghrelin
Physical activity	↑	Low insulin High AMPK gene expression High IGF-I
Moderate alcohol consumption	↑	High adiponectin, ghrelin Low insulin
Intrinsic factors		
AMPK activity	↑	Directly induces ketogenesis
ARNT/HIF1beta	↑	Stimulates AMPK activity

Ketones Increase AcetylCoA Pool

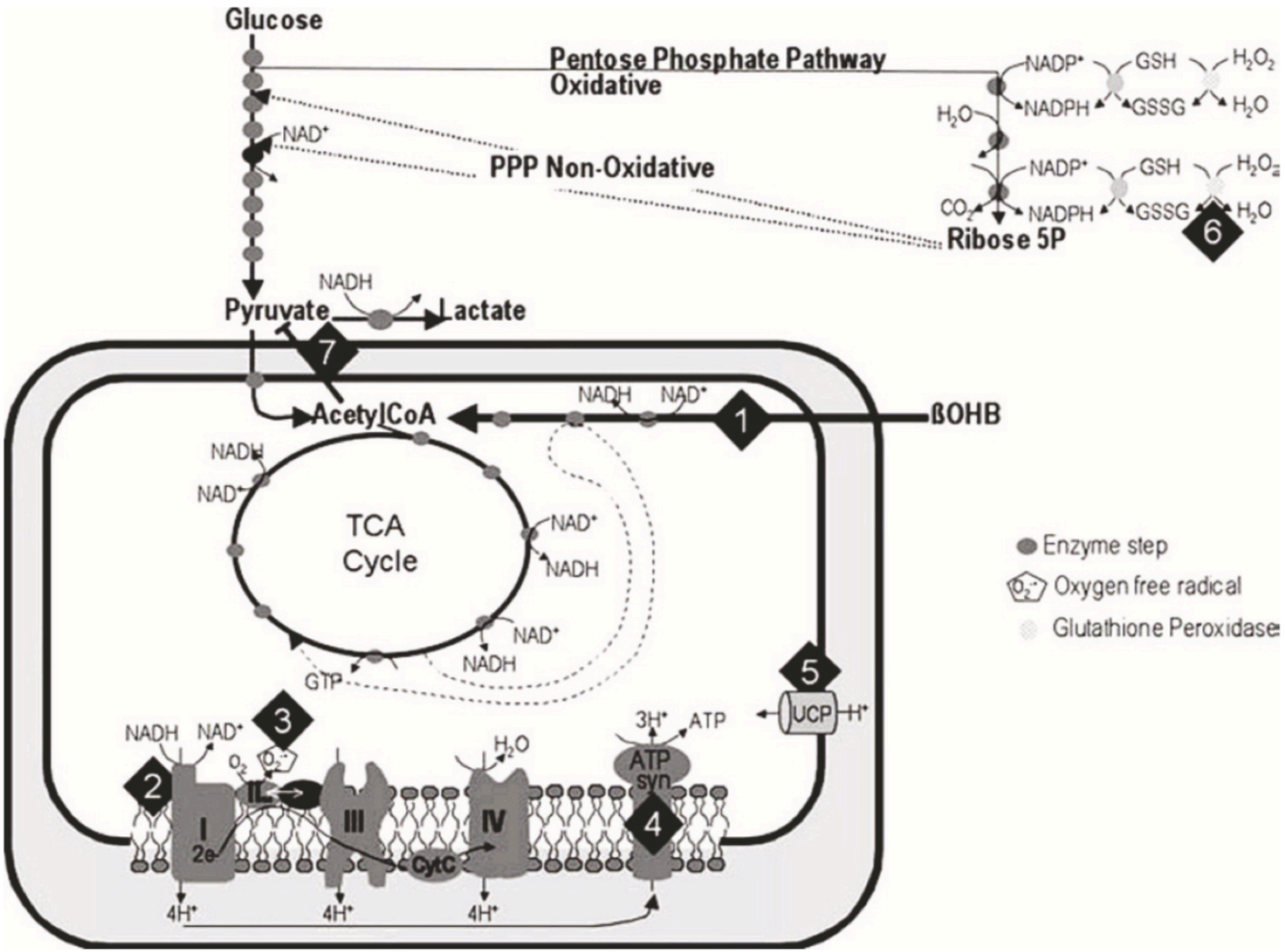


Figure 1. Potential neuroprotective mechanisms of ketones. Ketones (1) require only three enzymatic steps to enter the tricarboxylic acid (TCA) cycle, (2) reduce the NAD couple, (3) decrease free radical formation, (4) increase production of ATP, (5) increase mitochondrial uncoupling, (6) increase glutathione peroxidase activity, and (7) inhibit pyruvate entry into the TCA cycle. CoA, coenzyme A; GSH, reduced glutathione; GSSG, oxidized glutathione; NAD^+, nicotinamide adenine dinucleotide; NADH, nicotinamide adenine dinucleotide hydrogenase; NADP, nicotinamide adenine dinucleotide phosphate; NADPH, nicotinamide adenine dinucleotide phosphate hydrogenase; OHB, hydroxybutyrate; PPP, pentose phosphate pathway; UCP, uncoupling protein. Reprinted with permission from [43].

T cell metabolism drives immunity

Michael D. Buck, David O'Sullivan, and E

Department of Pathology and Immunology, Washington University Scho

Lymphocytes must adapt to a wide array of environm development, during which they undergo a dramatic search in this area has yielded surprising findings on ways and metabolites, which have been found to regu influence differentiation, function and fate. In this r in the field to provide an up-to-date resource on lym

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Abbreviations used: ACC1, acetyl-CoA carboxylase 1; AMPK, adenosine monophosphate–activated protein kinase; ASS1, argininosuccinate 1; CPT1a, carnitine palmitoyltransferase 1a; DN, double negative; DP, double positive; ERRα, estrogen-related receptor α; ETC, electron transport chain; FAS, fatty acid synthesis; HAT, histone acetyltransferase; HDAC, histone deacetylase; IDO, indoleamine-2,3-dioxygenase; PDK1, pyruvate dehydrogenase kinase; PDPK1, 3-phosphoinositide–dependent protein kinase-1; PI3K, phosphatidylinositol 3-kinase; PKA, protein kinase A; PTM, post-translational modification; ROS, reactive oxygen species; TCA, tricarboxylic acid; TPPII, tripeptidyl peptidase II; TSC1, tuberous sclerosis complex 1.

"Part of the secret of success in life is to eat what you like and let the food fight it out inside."
– Mark Twain

Simply stated, we are what we eat. Our genetics, coupled with environmental influences, dictate how we metabolize the nutrients that we consume and how this shapes our growth, function, and overall health. The same principles hold true at the cellular level. Just as a track runner quickly engages their muscles to propel themselves from rest to sprint in response to a starting gun, pathogen-derived or inflammatory signals drive T cells out of quiescence, resulting in rapid modulation of gene expression and the acquisition of new functions. These changes range from increased production of cytokines and cytolytic molecules to the ability to undergo cell division and migration. Intimately integrated into this program of activation is the regulation of cellular metabolism.

The engagement of specific metabolic pathways profoundly affects cell differentiation and function. Metabolic reprogramming is controlled by key receptor signaling events and growth factor cytokines, as well as availability of nutrients. In addition, metabolic products provide substrates that can alter the functional fate of a cell through posttranslational modifications (PTMs) or epigenetic remodeling. Several recent articles have covered these and other emerging topics in T cell metabolism (Chapman and Chi, 2014; Bird, 2015; Lochner et al., 2015; O'Sullivan and Pearce, 2015; Palmer et al., 2015; Ramsay and Cantrell, 2015; Ron-Harel et al., 2015). In this

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