

# Glycobiology World Congress

August 10-12, 2015 Philadelphia, USA

## Tracking cancer progression through monitoring the dynamic serum phospho-proteome

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Serum proteome has been shown to reflect pathological changes in tissues. Changes in serum proteomic pattern during carcinogenesis have been exploited successfully to serve as diagnostic and prognostic markers. Protein Kinase A catalytic subunit  $\alpha$  has been shown to be secreted into the serum as ecto-kinase and the ecto-PKA level is significantly higher in the serum of prostate cancer patients. Here, I describe the utility of the Reverse In-gel Kinase Assay coupled with LC-MS/MS to profile potential serum substrates of PKA and in following their phosphorylation status during disease progression. The sensitivity and linearity of this assay will help identify potential biomarkers which can be used to track the progression of prostate cancer. This strategy can be adapted and extended to track the progression of other cancers and pathological conditions.

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## Starvation of cancer via induced ketogenesis and severe hypoglycemia

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Neoplasms are highly dependent on glucose as their substrate for energy production and are generally not able to catabolize other fuel sources such as ketones and fatty acids. Thus, removing access to glucose has the potential to starve cancer cells and induce apoptosis. Unfortunately, other body tissues are also dependent on glucose for energy under normal conditions. However, in human starvation (or in the setting of diet-induced ketogenesis) the body “keto-adapts” and glucose requirements of most tissues drop to almost nil. Exceptions include the central nervous system (CNS) and various other tissues which have a small but obligatory requirement of glucose. Our hypothesized treatment takes keto-adaptation as a prerequisite. We then proposes the induction of severe hypoglycemia by depressing gluconeogenesis while administering glucose to the brain. Although severe hypoglycemia normally produces adverse effects such as seizure and coma, it is relatively safe following keto-adaptation. We hypothesize that our therapeutic hypoglycemia treatment has potential to rapidly induce tumor cell necrosis.

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