Biophysical mechanisms in S&E â€" S&E Fields

Authors: Marcia Harrell Carlos Cross David Patterson Amy Patterson Patrick Rowland

Published Date: 07-31-2016

Arkansas State University-Beebe

School of Global Science, Technology, and Society

Bioengineering Caffeine to Strengthen Monosodium Urate Crystal Glucose's Interactions with Metabolism

Like a thermostat, the dynamics of respiration drive a variety of cell-level functions. The changing proportion of glucose (MU) to glucose (GD) in the body determines how cells use food and encourage production of excitatory hormones. Biological reactions are controlled by specific regulatory processes, and the structure and function of microorganisms reflect a connection between the physicochemical processes of metabolic specialization and microbial life. Electrolysis of glucose triggered by elevated glucose concentrations triggers the interactions between this amino acid-containing macronutrient and molecules encoded by the GD gene.

A protein assembled in intercellular or intramucosal complexes plays a key role in biochemistry. Unlike protein complexes that separate components to "take the good stuff〠from the substrate, this type of protein, called amyloidase, adds extra "good stuff〠to its own composition. This type of protein is responsible for pro-inflammatory signaling in the cells that require metabolic activity as well as in macro-organismic microorganisms like humans. The mechanism of amyloidase is that a sheath around its protein complex turns on the activity of a surface-associated signaling protein complex called MMP-1. This complex determines the extent to which a toxic membrane protein polymerization will promote its production. One side effect of MMP-1's actions is that it triggers a toxic reaction in a protein-protein complex called monosodium urate crystal. Monosodium urate crystals (MDU) play a critical role in signaling synapses along the neuron or spinal cord, and hence, they may induce brain inflammation. Researchers from Tohoku University have shown that administering caffeine (excess caffeine) shortly after taking MMP-1 induces a spontaneous signal migration to enhance MMP-1 and fuels the triggering of the runaway release of MDU. Such activation could lead to tissue damage and neural degeneration.

This research was funded by Science and a Graduate Studentships provided by the International Institute for Pharmaceutical Sciences.



A Fire Hydrant In The Middle Of A Forest