Insulin

- Pancreas releases insulin from beta cells based on glucose levels
  - alpha cells release glucagon
  - The rising glucose levels signal the pancreas to secrete insulin to clear glucose from the bloodstream.
  - To do this, insulin binds with insulin receptors on the surface of cells, acting like a key that opens the cells to receive glucose. There are insulin receptors on almost all tissues in the body, including muscle cells and fat cells.
  - **Insulin receptors** have two main components—the exterior and interior portions. The exterior portion extends outside the cell and binds with insulin. When this happens, the interior part of the receptor sends out a signal inside the cell for glucose transporters to mobilize to the surface and receive the glucose. As blood sugar and insulin levels decrease, the receptors empty and the glucose transporters go back into the cell.

Insulin's main job is to store energy in the cell

fat burning stops when insulin is high

What is Insulin Resistance?

High insulin is the cause of insulin resistance

it is the failure of insulin excreted in normal amounts to lower glucose. When over-excretion fails to lower glucose, we have a runaway train.

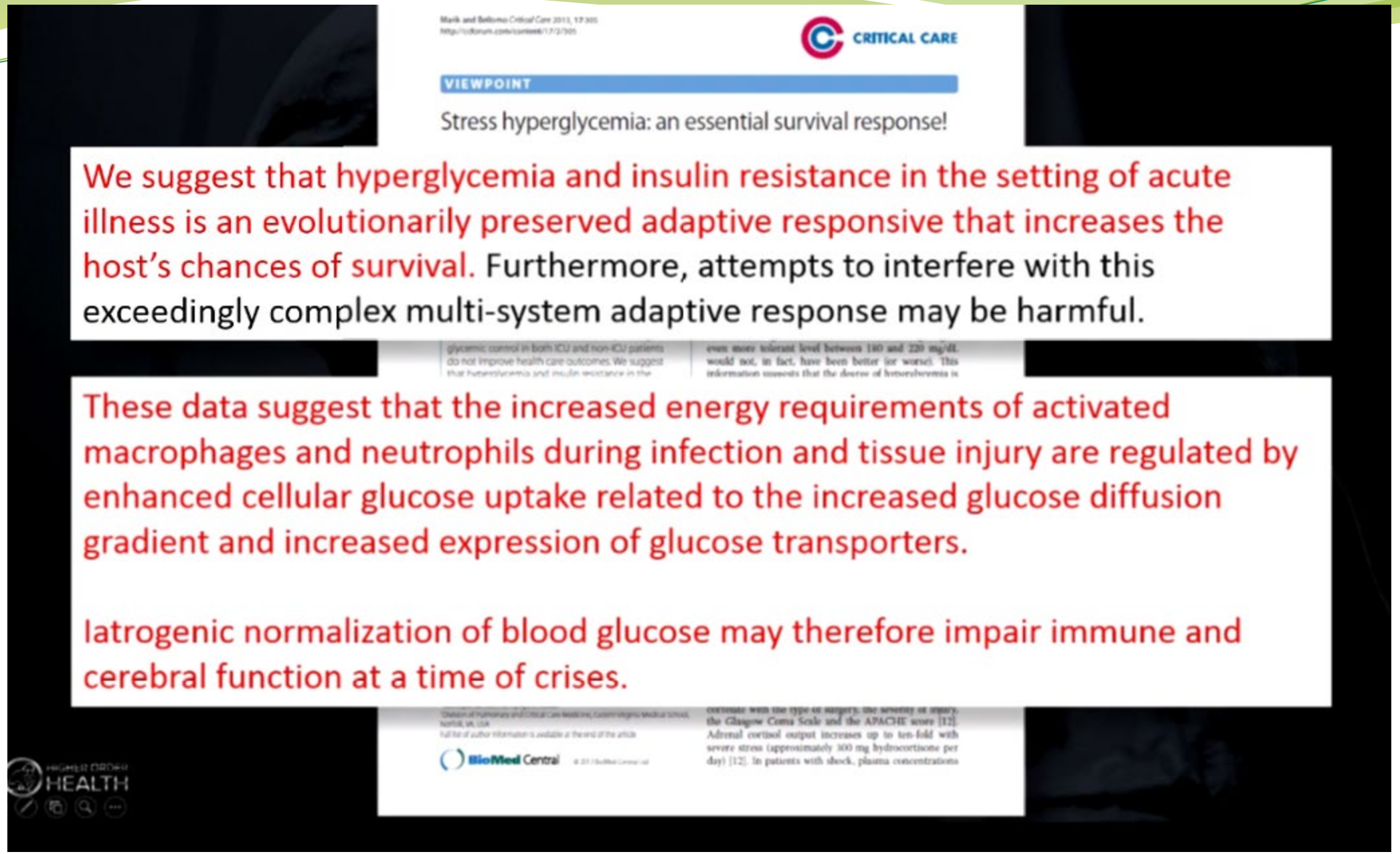
High glucose is ONLY a SYMPTOM.

What causes Insulin Resistance

- Inflammation
- **High insulin**
- Cortisol
- Excess fatty acids

Walsh:

Is insulin acting in a protective measure?


CRITICAL CARE
VIEWPOINT
Stress hyperglycemia: an essential survival response!
We suggest that hyperglycemia and insulin resistance in the setting of acute illness is an evolutionarily preserved adaptive responsive that increases the host's chances of survival. Furthermore, attempts to interfere with this exceedingly complex multi-system adaptive response may be harmful.
These data suggest that the increased energy requirements of activated macrophages and neutrophils during infection and tissue injury are regulated by enhanced cellular glucose uptake related to the increased glucose diffusion gradient and increased expression of glucose transporters.
Iatrogenic normalization of blood glucose may therefore impair immune and cerebral function at a time of crises.
BioMed Central
HEALTH

Walsh:

So, mitochondrial oxidative stress may be upstream of IR and may be common denominator and IR may be a protective mechanism, in which case we should perhaps reconsider using therapeutic strategies to overcome UNLESS they also eliminate the primary defect. If ox stress upstream? maybe the cell does not want glucose to come in as there is already too much metabolic stress within the cell.

Walsh:

In cases where we find high insulin, and where we see oxidative stress and inflammation, OS precedes models of IR and where it makes the cell intentionally become IR.

Glucose and A1c can be normal but when insulin alone is elevated, this is generally considered the early stages of insulin resistance.

Studies have shown that working on **mitochondrial function can lower IR with basic balancing of nutrients** so that the body can perform all its functions and adequately remove oxidative stress from the cell, hence removing the need to use IR as a protective measure.

Resources for Study

new paradigm
https://www.dietdoctor.com/a-new-paradigm-of-insulin-resistance

"See paper "insulin Resistance as a phys defense against metabolic stress: implications for the mgmt of subsets of T2D""
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4338588/

Insulin resistance is a cellular antioxidant defense mechanism
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2764908/

Why are we trying to use supplements to stimulate more action from the pancreas?

They produce insulin just fine.

They produce insulin just fine.