PREVIEW 1 - David Ludwig (Denver 2019)

Prof. David Ludwig: So according to what could be termed the carbohydrate insulin model of obesity the problem begins at the right side of this figure. Something has triggered fat cells to take in and hold on to too many calories.

So, there are too few calories in the bloodstream, not too many, as in the other model there are too few. The brain perceives that as an energy crisis, not enough calories to run metabolism, to feed the brain, to satisfy the needs of the other organs in the body.

And so that's why we become hungry and wind up overeating and the brain recognizing this potential metabolic problem also lowers energy expenditure by making us fatigued and less likely to move, lowers resting energy expenditure, changes muscular efficiency.

Now if this model is true then the advice to just eat less and move more will be doomed to failure and actually might make the problem worse because it's further going to restrict that already limited fuel supply circulating in the bloodstream.

So, what could be triggering fat cells into this calorie storage overdrive, this feeding frenzy? Well, of course you've heard about insulin quite a bit in this conference so far. Insulin is the dominant anabolic hormone with regard to energy metabolism it regulates the availability of all of the metabolic fuels.

States have increased insulin action not insulin levels per se, because we have to think of insulin resistance too, but states have increased insulin action with more secretion or injection of insulin for somebody with type 2 diabetes, or insulin promoting secreting tumors consistently leads to weight gain.

Whereas states have decreased insulin action consistently are associated with weight loss. Such as under treatment of a child with type 1 diabetes who can't make enough insulin.