The relationship between leptin and inflammation in adipose tissue determines critical points in excess nutrition

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The model: a system of differential equations

We model the rates of leptin production and inflammation by the following:

\[
\frac{dL}{dt} = \alpha SAT + \beta C - \delta_1 L \\
\frac{dC}{dt} = \gamma L^2 - \delta_2 C
\]

L: adipocyte-derived leptin; C: non-adipocyte-derived cytokines (inflammatory marker) 
SAT: amount of subcutaneous adipose tissue; \( \alpha, \beta, \gamma, \delta_1, \delta_2 \): parameters

Results and applications

- Cytokines produced directly and indirectly by leptin provide an appropriate biomarker for inflammation.
- Inflammation due to leptin secreted by enlarged adipocytes increases volume of adipose tissue and contributes to, as well as exacerbates, obesity. The model, providing points of stability and instability, helps determine when medical intervention is necessary in weight loss.
- Women have higher leptin levels because estrogen favors fat deposition in subcutaneous adipose tissue, which secretes more leptin than other types of fat. We propose that this is the reason for the sexual dimorphism seen in autoimmunity.
- Using this model, we can better determine risk for autoimmune diseases, type 2 diabetes, and metabolic syndrome.

References & Acknowledgements

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