ANS -INFLAMMATION LINK: A NEW INDEPENDENT MECHANISM FOR HOMEOSTASIS

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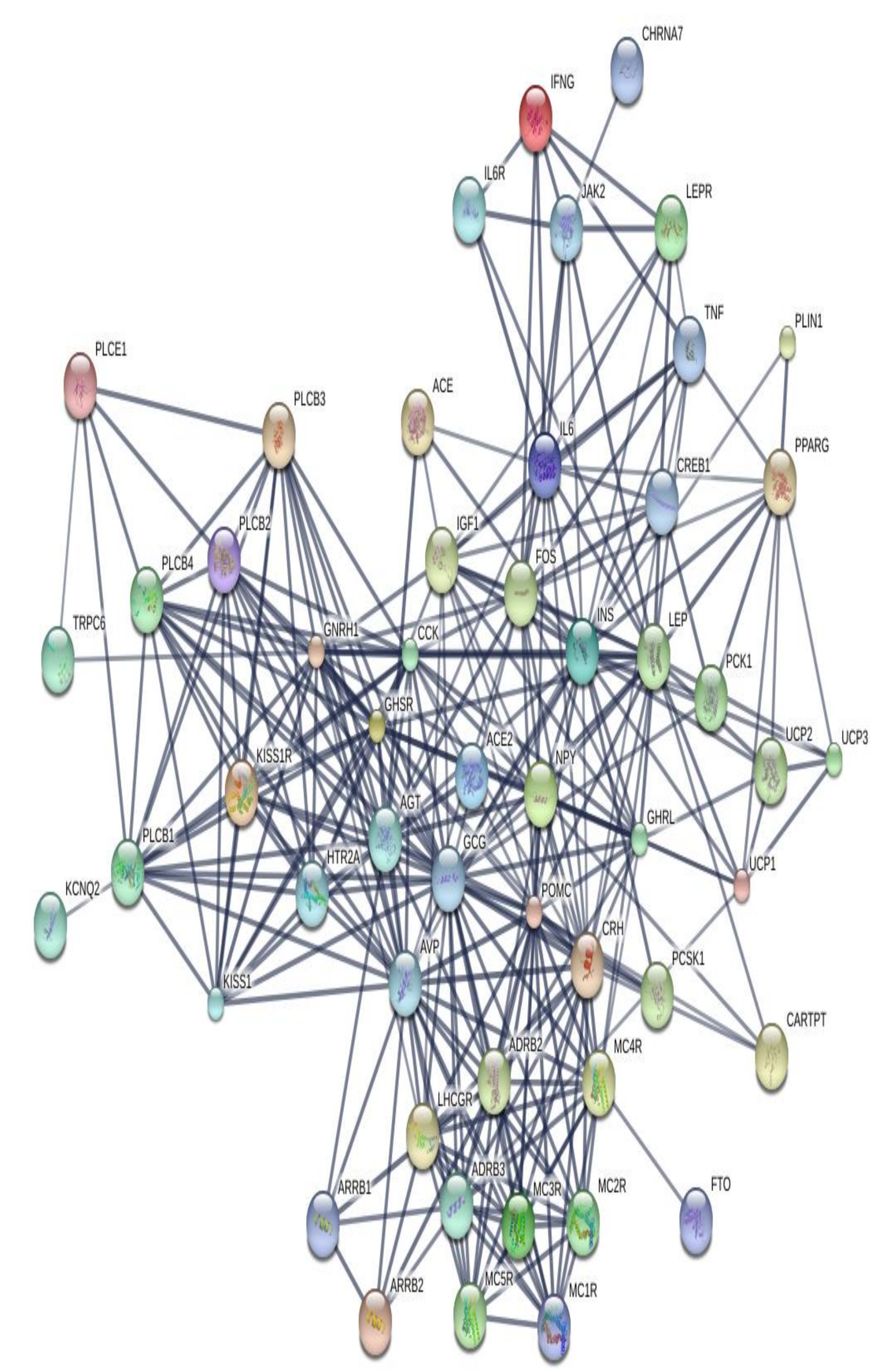
BACKGROUND

Translational research showed evidence on autonomic nervous function correlation with cytokines and gut hormones. It has been recently shown that in the gut, the cross talk of the stimulated vagus nerve with immune cells increases the cholinergic tone. In obesity hyperinsulinemia and hyperleptinemia induce ANS activation



OBJECTIVES & HYPOTHESES

To summarise literature exploring links among gut hormones, immune factors, pancreatic β-cell function and the autonomic nervous system.

METHODS

Literature search in PUBMED, UniProt, GeneCards.. Confidence >0.97

RESULTS

In human bariatrics after surgery with and without diabetes type 2, an implication of inflammation factors to autonomic nervous system response, independently of BMI or fat loss, has been suggested. More importantly a meta-analysis in bariatrics evaluated insulin resistance and ANS after surgery, suggesting a link between gut, β-cells and ANS. A separate interactions network within the so termed "Obesidome" -been created to explore this interplay. It elucidated an intricate communication network between the nervous and immune systems; this interplay could advocate in the regulation of the immune response. TGF- (and thymic stromal lymphopoietin) produced by the enterocytes and/or immune cells, contribute to the maintenance of immune homeostasis. The interactions between the inflammatory and/or autonomic nervous system biomarkers and their encoding genes revealed that JAK2 serves as a key hub for leptin and insulin activity, thus, providing the foundation to further investigation.

CONCLUSIONS

Hormones (with leptin hallmark node) implicate with the autonomic nervous system, immune system, homeostasis system, genetic factors, to a mechanism that seems to be independent and future research should elucidate its function.

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There is no conflict of interest

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