# Article information:

Amygdala microglia modify neuronal plasticity via complement C1q/C3-CR3 signaling and contribute to visceral pain in a rat model | American Journal of Physiology-Gastrointestinal and Liver Physiology
[https://journals.physiology.org/doi/full/10.1152/ajpgi.00123.2021?rfr\_dat=cr\_pub++0pubmed=Z39.88-2003=ori%3Arid%3Acrossref.org](https://journals.physiology.org/doi/full/10.1152/ajpgi.00123.2021?rfr_dat=cr_pub++0pubmed&url_ver=Z39.88-2003&rfr_id=ori%3Arid%3Acrossref.org)

# Article summary:

1. Irritable bowel syndrome (IBS) is a chronic gastrointestinal disorder that is worsened by chronic psychological stress, indicating a link between abnormal GI function and activation of the hypothalamic-pituitary-adrenal (HPA) stress axis.

2. Microglia in the amygdala are activated by environmental stressors and undergo morphological and functional alterations, contributing to the development of visceral hypersensitivity.

3. The complement protein C1q/C3-CR3 signaling is necessary for microglia-modulated synaptic remodeling in the central nucleus of amygdala (CeA) following chronic stress and contributes to stress-induced visceral hypersensitivity.

# Article rating:

Appears moderately imbalanced: The article provides some useful information, but is missing several important points or pieces of evidence that would be required to present the discussed topics in a balanced and reliable way. You are encouraged to seek a more balanced perspective on the presented issues by exploring the provided research topics and looking at different information sources.

# Article analysis:

该文章提出了关于慢性肠道疾病和心理压力之间的联系以及小胶质细胞在这一过程中的作用的假设。然而，该文章存在以下问题：

1. 偏见来源：该文章没有提到其他可能导致慢性肠道疾病的因素，例如饮食、生活方式等。此外，该文章也没有探讨其他可能影响小胶质细胞功能的因素。

2. 片面报道：该文章只关注了小胶质细胞在慢性肠道疾病中的作用，而忽略了其他可能参与其中的细胞类型和分子机制。

3. 无根据主张：该文章提出了C1q/C3-CR3信号通路是小胶质细胞介导突触修剪的重要机制，但并未提供足够证据支持这一假设。

4. 缺失考虑点：该文章没有考虑到不同种类和程度的心理压力对患者产生不同影响的可能性。此外，该文章也没有探讨治疗方法对小胶质细胞功能和患者症状改善之间关系的可能性。

5. 主张缺失证据：尽管该文章提出了小胶质细胞介导突触修剪是慢性肠道疾病发展过程中一个重要机制，但并未提供足够证据支持这一假设。

6. 未探索反驳：该文章没有探讨其他学者对于其假设和结论所提出反驳意见，并且也没有进行充分讨论来解决这些争议点。

7. 宣传内容：尽管该文章声称是科学期刊上发表的原始研究成果，但其内容似乎更像是某种药物或治疗方法宣传材料。

8. 偏袒：该文章似乎更倾向于支持作者自己所提出的假设和结论，并且忽略了其他可能解释现象的因素。此外，作者也没有公开披露任何与本文相关的利益冲突或竞争利益。

# Topics for further research:

* Other factors contributing to chronic intestinal diseases
* Other cell types and molecular mechanisms involved in chronic intestinal diseases
* Lack of evidence for the C1q/C3-CR3 signaling pathway as a mechanism for synaptic pruning by microglia
* Variations in the effects of different types and degrees of psychological stress on patients
* Insufficient evidence for microglia-mediated synaptic pruning as a key mechanism in chronic intestinal diseases
* Lack of discussion of counterarguments to the author's hypothesis and conclusions

# Report location:

<https://www.fullpicture.app/item/bef38dfdc32bfc7323efcb80da5d259d>