# Article information:

Role of the IL23/IL17 Pathway in Crohn's Disease - PubMed
<https://pubmed.ncbi.nlm.nih.gov/33859636/>

# Article summary:

1. Crohn's disease (CD) is a chronic inflammatory bowel disease that involves excessive activation of the mucosal immune system and aberrant cytokine responses.

2. The IL23/IL17 pathway plays a significant role in the pathogenesis of CD, with IL23 enhancing the expansion of pathogenic T helper type 17 (Th17) cells and inducing pro-inflammatory molecules.

3. Recent advances in understanding the immunopathogenetic mechanisms of CD have led to the development of selective therapies targeting IL23 and IL17, which have shown promising results in CD patients who did not respond to anti-TNF therapy.

# 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:

根据文章的标题和摘要，这篇文章主要讨论了IL23/IL17通路在克罗恩病中的作用。然而，由于我无法访问全文，以下分析基于标题和摘要提供的信息。

1. 潜在偏见及其来源：从标题和摘要来看，文章似乎是一篇综述性质的论文，因此可能存在作者对IL23/IL17通路在克罗恩病中作用的偏见。此外，由于我无法访问全文，无法确定作者是否有与制药公司或其他利益相关方存在冲突。

2. 片面报道：从摘要中可以看出，文章主要关注IL23/IL17通路在克罗恩病发病机制中的作用，并介绍了新型生物治疗方法。然而，它可能没有全面涵盖其他可能影响克罗恩病发展的因素。

3. 无根据的主张：虽然摘要提到了IL23和IL17在克罗恩病发病机制中的作用，并提到了新型生物治疗方法对之前抗TNF治疗失败患者的高反应率，但没有提供具体证据支持这些主张。

4. 缺失的考虑点：从摘要中无法确定作者是否考虑了其他可能的发病机制或治疗方法。克罗恩病是一个复杂的疾病，可能涉及多种因素和通路。

5. 所提出主张的缺失证据：从摘要中无法确定作者是否提供了足够的证据来支持IL23/IL17通路在克罗恩病中的作用以及新型生物治疗方法的有效性。

综上所述，根据标题和摘要提供的信息，这篇文章可能存在潜在偏见和片面报道，并且没有提供足够的证据来支持其主张。由于我无法访问全文，无法对文章进行更详细的分析。

# Topics for further research:

* IL23/IL17 pathway in Crohn's disease
* Mechanisms of Crohn's disease development
* Other factors influencing Crohn's disease
* Potential conflicts of interest
* Evidence supporting the claims
* Alternative treatment approaches for Crohn's disease

# Report location:

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