# Article information:

Frontiers | Antifungal immunity mediated by C-type lectin receptors may be a novel target in immunotherapy for urothelial bladder cancer
<https://www.frontiersin.org/articles/10.3389/fimmu.2022.911325/full>

# Article summary:

1. Bladder cancer is a common urological malignancy with high incidence and mortality rates, and current therapeutic approaches have limitations in advanced stages.

2. The commensal fungi in the gut microbiota play crucial roles in a range of cancers, and anti-fungal immunity mediated by CLRs may be a novel target for immunotherapy.

3. Dectin-3 has been found to serve as an immune barrier against potential fungal invasion and participate in the tumorigenesis of colon cancer, while Dectin-1 activation on tumor-infiltrating macrophages can induce immune escape and accelerate tumor progression.

# 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:

该文章主要介绍了膀胱癌的治疗和免疫治疗的局限性，并探讨了共生菌群在免疫系统中的作用，特别是真菌共生菌群对C型凝集素受体介导的抗真菌免疫反应的影响。然而，该文章存在以下问题：

1. 偏见来源：该文章没有提及可能存在的偏见来源，例如作者是否有与药物公司或其他相关机构的利益冲突。

2. 片面报道：该文章只关注了真菌共生菌群对C型凝集素受体介导的抗真菌免疫反应的作用，而忽略了其他共生菌群对免疫系统的影响。

3. 无根据主张：该文章声称共生真菌可以成为膀胱癌免疫治疗的新靶点，但没有提供足够证据支持这一主张。

4. 缺失考虑点：该文章没有考虑到可能存在的副作用和风险，例如使用抗真菌药物可能会导致耐药性和其他不良反应。

5. 主张缺失证据：该文章没有提供足够证据支持其主张，例如共生真菌是否能够增强免疫治疗的效果。

6. 未探索反驳：该文章没有探讨可能存在的反驳观点，例如其他学者对共生真菌作为膀胱癌免疫治疗靶点的质疑。

7. 宣传内容：该文章似乎在宣传共生真菌作为膀胱癌免疫治疗靶点的概念，而没有提供足够客观的信息和证据。

综上所述，该文章存在一些问题，需要更加客观地呈现双方观点，并提供足够的证据和考虑到可能存在的风险和副作用。

# Topics for further research:

* Conflict of interest
* Other symbiotic microbiota
* Lack of evidence for the proposed target
* Potential risks and side effects
* Insufficient evidence for the claim
* Lack of exploration of opposing views

# Report location:

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