# Article information:

Epithelial cells activate fibroblasts to promote esophageal cancer development - ScienceDirect
<https://www.sciencedirect.com/science/article/pii/S1535610823000491>

# Article summary:

1. Esophageal squamous-cell carcinoma (ESCC) develops through multistage epithelial cancer formation, from normal epithelium to intraepithelial neoplasia and finally to invasive carcinoma.

2. Loss of ANXA1 expression in epithelial cells due to its transcription factor KLF4 suppression along the lesion progression promotes ESCC via activating cancer-associated fibroblast formation.

3. Cancer-associated fibroblasts (CAFs) can promote ESCC progression through multiple mechanisms, and the occurrence of a certain subtype of CAFs has prognostic value for ESCC.

# Article rating:

Appears strongly imbalanced: The article is written in a biased or one-sided way, and the information it provides is not trustworthy enough to be considered a reliable source. You should consult other sources to find reliable information on the presented issues.

# Article analysis:

该文章是一篇关于食管鳞状细胞癌（ESCC）发展机制的研究，通过单细胞RNA测序和空间转录组学分析，揭示了上皮细胞中ANXA1表达逐渐降低，并通过激活癌相关成纤维细胞（CAFs）促进ESCC发展的机制。然而，该文章存在以下问题：

1. 偏见来源：该文章没有提及其他可能的因素对ESCC发展的影响，如遗传、环境等因素。此外，该研究只涉及29名患者的样本，样本量较小。

2. 片面报道：该文章只关注了ANXA1/FPR2信号通路在ESCC发展中的作用，而忽略了其他可能的信号通路和因素。

3. 无根据主张：该文章声称ANXA1缺失会导致NFs向CAFs转化，并且可以被恶性上皮细胞分泌的TGF-β增强。然而，这些主张并没有得到充分证明。

4. 缺失考虑点：该文章没有考虑到不同类型的CFA对ESCC发展的影响可能不同，并且没有探讨如何针对不同类型的CFA进行治疗。

5. 主张缺失证据：该文章没有提供足够的实验证据来支持其主张，如ANXA1/FPR2信号通路在ESCC发展中的作用。

6. 未探索反驳：该文章没有探讨其他可能的解释或反驳对其主张的质疑。

7. 宣传内容：该文章可能存在宣传内容，如强调CAFs在癌症发展中的重要性，并且没有平等地呈现双方。

综上所述，该文章存在一些问题，需要更多的研究来证实其主张，并考虑到其他可能的因素和解释。

# Topics for further research:

* Other factors affecting ESCC development
* Other signaling pathways and factors in ESCC development
* Lack of evidence for ANXA1's role in CAF transformation and TGF-β enhancement
* Different types of CAFs and their impact on ESCC development
* Insufficient experimental evidence to support ANXA1/FPR2 signaling pathway's role in ESCC development
* Lack of exploration of alternative explanations or counterarguments

# Report location:

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