

# Lung adenocarcinoma

- Population-adjusted unanchored indirect comparisons of cancer therapies with borrowing of pan-tumor information
- Pembrolizumab-Induced Acalculous Cholecystitis Mimicking Gallbladder Carcinoma on 18F-FDG PET/CT
- ICARus: a pipeline to extract robust gene expression signatures from transcriptome datasets
- Pathological significance and therapeutic prospects of HCCS expression patterns in lung adenocarcinoma
- Machine learning developed an immune evasion signature for predicting prognosis and immunotherapy benefits in lung adenocarcinoma
- Case Report: Metastatic lung cancer in Meckel's Cave: a radiotherapy successful case and literature review
- Developing a prognostic signature and characterizing the tumor microenvironment based on centrosome-related genes in lung adenocarcinoma
- Comprehensive analysis of RFC4 as a potential biomarker for regulating the immune microenvironment and predicting immune therapy response in lung adenocarcinoma

Lung adenocarcinoma is a form of non-small-cell lung cancer.

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## Lung adenocarcinoma with epidermal growth factor receptor mutation

EGFR-Mutant Non-Small-Cell Lung Cancer.

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Sixty-four human lung adenocarcinoma patients with depression and 64 lung adenocarcinoma patients without depression were recruited for this study. The expression of 5-HT receptors on lung adenocarcinoma cells from tumour tissues were detected by using immunohistochemistry (IHC) and fluorescence-activated cell sorting (FACS). The depression models were established in vitro and in vivo. The effects of immunosuppression were evaluated by testing the function of cytotoxic lymphocyte (CTLs) and Tregs, measuring tumour weight or volume, assessing the survival of mice and staining of tissues by IHC. Changes in the expression of immunoregulatory factor genes were assessed to elucidate the mechanism of immune evasion induced by the 5- hydroxytryptamine receptor (HTRs).

Higher levels of 5-HT, increased expression of 5-HT1Rs and decreased overall survival were observed in lung adenocarcinomas patients with depression compared with those without depression. Moreover, 5-HT1aR, a critical factor for increasing the number of CD4+CD25+Foxp3+ Treg cells and decreasing the ratio of Th1/Th2 cells, which suggested immune system dysregulation. In addition, expression of 5-HT1aR on tumour cells was also negatively associated with CTL activity in both peripheral blood and tumour infiltrating lymphocytes. In a depressive state, 5-HT1aR activates p-signal transducer and activator of transcription 3 (STAT3) and autophagy, and programmed death ligand-1, a downstream gene of autophagy/p-STAT3 signalling, mediates an immunosuppressive

environment. Moreover, in both the mouse model and lung adenocarcinoma patients, the activation of 5HT1aR and the elevated tumour autophagy/p-STAT3 axis were associated with reduced overall survival.

The 5-HT1aR/autophagy/p-STAT3 axis influences both tumour cells and immune cells, resulting in immunosuppression in lung adenocarcinomas patients with depression <sup>1)</sup>.

## Unclassified

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