DNA-modulating drug attenuates lung inflammation in mice

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Patients with cystic fibrosis (CF) suffer from chronic respiratory infections, primarily caused by *Pseudomonas aeruginosa*, which lead to airway inflammation and damage. Several recent studies have suggested that a specific type of immune cell, known as Th17 cells, produce the factor IL-17 to drive the inflammatory response in the setting of CF lung infection.

In this issue of *JCI Insight*, Jay Kolls and colleagues at the University of Pittsburgh demonstrate that bromodomain and extraterminal domain (BET) inhibitors, a class of drugs that alter DNA architecture and change gene expression, attenuate CF lung inflammation.

Using immune cells isolated from CF patient lungs, Kolls and colleagues showed that BET inhibitors suppressed the response to Th17 cells as well as the release of inflammatory factors from Th17 cells.

Moreover, in a mouse model of lung infection, BET inhibitor treatment decreased lung inflammation without promoting infection, indicating that BET inhibitors could potentially be used to treat CF patients.

More information: Kong Chen et al, Antiinflammatory effects of bromodomain and extraterminal domain inhibition in cystic fibrosis lung inflammation, *JCI Insight* (2016). DOI: 10.1172/jci.insight.87168

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