

Better Asthma and COPD Drugs with Fewer Side Effects Are Within Reach

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constrictor effects of acetylcholine are enhanced. It has been long known that beta-adrenoceptor agonists could reverse the constricting effects of acetylcholine on airway smooth muscle, but it has not been fully clear how this occurs. It is important to understand how these medications work to assist with future development of more effective drugs with fewer side effects.

More information: Tuleen Alkawadri et al, M2 muscarinic receptor-dependent contractions of airway smooth muscle are inhibited by activation of β -adrenoceptors, *Function* (2022). DOI: [10.1093/function/zqac050](https://doi.org/10.1093/function/zqac050)

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Bronchodilators, the most common type of asthma-fighting drug, inhibits contractions of airway smooth muscle that are induced by stimulating receptors on the muscle's surface. New research highlights a novel mechanism for the drugs and will aid in the development of better medications for the treatment of asthma and chronic obstructive pulmonary disease (COPD). The findings are detailed in a new article by researchers from Dundalk Institute of Technology in Ireland and Queen's University in Northern Ireland.

Asthma and COPD are common lung disorders—caused by excessive constriction of the airways—that make breathing difficult. Bronchodilators, also known as beta-adrenoceptor agonists, relax airway [smooth muscle](#) and open the airways to make breathing easier. These drugs bind to beta-adrenoceptors located on airway muscle cells and cause them to relax.

Constriction of the airways is regulated in the nerves, which release a chemical called acetylcholine. This chemical causes the airways to constrict and narrow. For patients with COPD, the

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