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CORRECTION

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Correction: Progress of CCL20-CCR6 in the airways: a promising new therapeutic target

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Following the publication of the Original Article, the authors reported that the order of references in Table 1 was not correctly modified during the typesetting.

The online version of the original article can be found at https://doi.org/10.1186/s12950-024-00427-5.

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The incorrect table is as follows:

Table 1 Summary table of CCL20-CCR6 in the respiratory system

Name	frequency	Action mechanism	Effector cell	Phenotype	Reference
Asthma	↑	The secretion of CCL20 by airway epithelium is stimulated by MAPK and AKT pathways under the stimulation of related inflammatory factors.	DCs, Treg	Harmful	[65], [67]
NSCLC	↑	NSCLC cells promote CCL20 production and induce lung cancer cell migration through MAPK, Wnt, and PI3K pathways.	Th17, Treg, ILC3s	Harmful	[76]
COPD	↑	Activation and activation of TGF-β induced by integrin avβ8 enhances CCL20 transcription and exacerbates airway fibrosis.	DCs	Harmful	[92–93]
Pulmonary tuberculosis	↑	Induces T lymphocytes to migrate to the site of inflammation.	$CD4^+$ T, $CD8^+$T	Harmful	[99]
Pulmonary nodular disease	↑	Stimulate the activation of the PI3k/Akt pathway, causing Th17/Treg imbalance; Activate the TGF-β/Smad pathway.	Treg, Th1, Th17	Harmful	[107–108]
Pulmonary aspergillosis	↓	By depleting CCL20, the innate immune response of the host is suppressed.	Th1, Th2, Th17	Protective	[113]
Viral Infectious Pneumonia	↑	The imbalance of Th17/Treg cells and the decreased ability of $CD8^+$T cells to clear the virus aggravate lung inflammation.	Treg, Th17	Harmful	[116]

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The Original Article has been corrected.
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