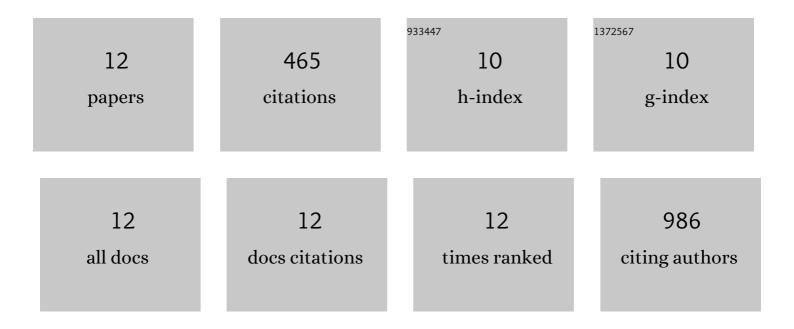
## Liye Chen

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/7900746/publications.pdf Version: 2024-02-01



LIVE CHEN

#	Article	IF	CITATIONS
1	A genetics-led approach defines the drug target landscape of 30 immune-related traits. Nature Genetics, 2019, 51, 1082-1091.	21.4	157
2	Critical Role of Endoplasmic Reticulum Aminopeptidase 1 in Determining the Length and Sequence of Peptides Bound and Presented by HLA–B27. Arthritis and Rheumatology, 2014, 66, 284-294.	5.6	71
3	Silencing or inhibition of endoplasmic reticulum aminopeptidase 1 (ERAP1) suppresses free heavy chain expression and Th17 responses in ankylosing spondylitis. Annals of the Rheumatic Diseases, 2016, 75, 916-923.	0.9	66
4	An ankylosing spondylitis-associated genetic variant in the <i>IL23R-IL12RB2</i> intergenic region modulates enhancer activity and is associated with increased Th1-cell differentiation. Annals of the Rheumatic Diseases, 2016, 75, 2150-2156.	0.9	45
5	Genetic Architecture of Adaptive Immune System Identifies Key Immune Regulators. Cell Reports, 2018, 25, 798-810.e6.	6.4	36
6	Inhibiting ex-vivo Th17 responses in Ankylosing Spondylitis by targeting Janus kinases. Scientific Reports, 2018, 8, 15645.	3.3	27
7	Position 97 of HLA-B, a residue implicated in pathogenesis of ankylosing spondylitis, plays a key role in cell surface free heavy chain expression. Annals of the Rheumatic Diseases, 2017, 76, 593-601.	0.9	17
8	From genome-wide association studies to rational drug target prioritisation in inflammatory arthritis. Lancet Rheumatology, The, 2020, 2, e50-e62.	3.9	17
9	GM-CSF Primes Proinflammatory Monocyte Responses in Ankylosing Spondylitis. Frontiers in Immunology, 2020, 11, 1520.	4.8	16
10	Identification of an Unconventional Subpeptidome Bound to the Behçet's Disease-associated HLA-B*51:01 that is Regulated by Endoplasmic Reticulum Aminopeptidase 1 (ERAP1). Molecular and Cellular Proteomics, 2020, 19, 871-883.	3.8	13
11	02.35â€Time of flight mass cytometry (cytof) and rna sequencing interrogation of â€~pathogenic' gm-csf lymphocytes in human spondyloarthritis. , 2017, , .		0
12	O38 G-Protein-coupled receptor-65 expression marks out a pathogenic GM-CSF-producing subset of CD4 T cells which are expanded in spondyloarthritis. Rheumatology, 2018, 57, .	1.9	0