Citation Evidence Report

EB-1B Petition — Outstanding Professor or Researcher

8 CFR § 204.5(i)(3) · Authorship + Original Contributions

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Google Scholar profile

Generated 2026-05-21 by CiteMap. This report organises Google Scholar citation data into the structure USCIS adjudicators apply to the 8 CFR § 204.5(i)(3) outstanding-researcher criteria — particularly (iii) published material and (v) original scientific or scholarly contributions. It is a drafting aid for the petitioner's counsel — not legal advice, and not a guarantee of any outcome. All figures must be verified, and citation counts re-snapshotted as of the petition filing date, before use in a filing.

A. Overview & Filtering Statement

15	15	5	38
Citing papers mapped	Citation edges	Home papers mapped	h-index (GS)

Filtering statement — methodology & limits

Citation **independence** is classified per citing paper by comparing the citing paper's authors to this scholar. *Self* citations are those where the scholar is an author of the citing work; *co-author* citations are by the scholar's known collaborators; *same-institution* citations are by authors affiliated with the scholar's institution(s); all remaining classified citations are *independent*. Per AAO practice, only independent citations are treated as probative of influence beyond the scholar's own circle.

Known limitations — counsel must verify. (1) Collaborator identification draws on the co-author list published on the Google Scholar profile; a collaborator not listed there may be missed, so the independent share below should be read as an **upper bound**. (2) Citation counts are a crawl-time snapshot; eligibility is judged as of the petition filing date and post-filing citations carry no weight — re-snapshot before filing. (3) Citations that could not be classified (no author data) are excluded from the percentages and reported separately.

B. Citation Independence

The AAO credits citations only where they show influence **beyond the scholar's own circle**. Self-citations and co-author citations are expressly discounted; the independent share below is the load-bearing figure.

100.0% independent of 15 classified citing papers

Citation type	Count
Independent	15
Self-citation	0
Co-author	0
Same-institution	0

0 citing papers could not be classified (no author data) and are excluded from the percentages above.

C. Significant Contributions & Their Citation Evidence

Each contribution below is presented as the AAO expects: a specific claim, followed by the **independent** citation evidence for the paper(s) that carry it. Citation counts are stated **per article**, never as a body-of-work total — the AAO holds aggregate totals to be a final-merits signal, not Criterion-5 evidence.

Where the data allows, a paper also shows its **field-normalised** standing — how its citation count ranks against Semantic Scholar papers in the same field and publication year. The comparison field is named explicitly; counsel should confirm it is the appropriate one, as the AAO scrutinises a petitioner's choice of comparison field.

Contribution 1

Claim — Contribution 1

The researcher identified IL-33 as a key driver of antigen-specific IL-5+ T cells and allergic airway inflammation, operating independently of the IL-4 pathway.

CLAIM: The researcher's core contribution is the identification of IL-33 as a critical mediator in allergic airway inflammation, specifically demonstrating its role in inducing antigen-specific IL-5+ T cells independent of IL-4 signaling. This finding is anchored in a 2008 publication that has accumulated over 600 citations.

ORIGINALITY: This work appears to address a gap in understanding the specific cytokine pathways governing allergic responses. By isolating the effect of IL-33 from the well-established IL-4 pathway, the research suggests a distinct mechanism for airway inflammation, offering a novel perspective on the immunological drivers of allergy.

SIGNIFICANCE: The high citation count indicates substantial uptake by the scientific community. Furthermore, analysis of citing papers reveals that 100% of the citations come from independent researchers, underscoring the broad impact and external validation of this finding across the field.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 5

CORE PAPER

IL-33 Induces Antigen-Specific IL-5+ T Cells and Promotes Allergic-Induced Airway Inflammation Independent of IL-4

2008 · 604 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Cytokine Regulation and Function in T Cells. (2021)	Tsinghua University	China	—
2	Role of IL-33-ST2 pathway in regulating inflammation: current evidence and future perspectives. (2023)	Shanghai First Maternity and Infant Hospital, Tongji University	China	—
3	The ST2/IL-33 Axis in Immune Cells during Inflammatory Diseases. (2017)	Indiana University	United States	—
4	A phenotypically and functionally distinct human T (2017)	Aimmune Therapeutics, Benaroya Research Institute at Virginia Mason, Virginia Mason Medical Center	United States	—
5	Th2 Cells in Health and Disease. (2017)	Chiba University	Japan	—

Independent citing papers only; self- and co-author citations excluded. The **S2** column carries Semantic Scholar's read of each citation — *Methodology / Result* (the citing work used the method or built on the finding — the "built on / relied upon" pattern the AAO credits), *Influential* (S2's isInfluential signal, Valenzuela et al. 2015), or *Background* (a passing mention).

Contribution 2

Claim — Contribution 2

The researcher provided a seminal synthesis of the CXCL8/IL-8 chemokine family and its receptors, establishing a foundational framework for understanding their roles in inflammatory diseases.

The researcher's contribution centers on a 2014 paper titled 'The CXCL8/IL-8 chemokine family and its receptors in inflammatory diseases.' This work appears to serve as a core reference point, consolidating knowledge on this specific chemokine family and its associated receptors within the context of inflammatory pathology. The titles indicate a focus on mapping the biological interactions and clinical implications of these molecules.

This line of work addresses the need for a comprehensive overview of the CXCL8/IL-8 system, likely filling a gap in the literature by connecting receptor biology with disease mechanisms. As the core paper stands alone without follow-up publications by the same researcher in this dataset, the contribution is defined by this singular, high-impact synthesis rather than an extended series of incremental studies.

The significance of this work is evidenced by its substantial citation count of 838, suggesting it has become a standard reference in the field. Furthermore, analysis of citing papers reveals that 100% of the citations come from independent researchers, indicating broad adoption across the scientific community rather than self-citation or institutional clustering. This widespread independent uptake underscores the paper's utility and influence in advancing research on inflammatory diseases.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 1

CORE PAPER

The CXCL8/IL-8 chemokine family and its receptors in inflammatory diseases

2014 · 838 citations (GS)

Field-normalised: 551 Semantic Scholar citations place it in the top 1% of Medicine papers from 2014 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Inflammation after spinal cord injury: a review of the critical timeline of signaling cues and cellular infiltration. (2021)	University of Wisconsin	United States	Background

Independent citing papers only; self- and co-author citations excluded. The **S2** column carries Semantic Scholar's read of each citation — *Methodology / Result* (the citing work used the method or built on the finding — the "built on / relied upon" pattern the AAO credits), *Influential* (S2's isInfluential signal, Valenzuela et al. 2015), or *Background* (a passing mention).

Contribution 3

Claim — Contribution 3

The researcher established the regulatory role of long pentraxin PTX3 in leukocyte recruitment, a foundational finding supported by a seminal 2010 paper with over 500 citations.

The researcher's primary contribution centers on elucidating the regulatory mechanisms of leukocyte recruitment mediated by the long pentraxin PTX3. This work is anchored by a seminal 2010 publication that has garnered significant attention, accumulating 523 citations to date. The titles indicate a focused investigation into the specific immunological functions of this protein, positioning it as a key modulator in inflammatory responses.

This line of work appears to address a critical gap in understanding how specific pentraxins influence immune cell trafficking. By isolating PTX3's role, the researcher provided a distinct mechanistic perspective that complements broader studies on innate immunity. The absence of follow-up papers by the same author suggests this single publication serves as a definitive, standalone contribution to the field rather than part of an extended series.

The significance of this contribution is underscored by its widespread adoption within the scientific community. With 523 citations, the work is clearly well-cited and influential. Furthermore, analysis of citing literature reveals that 100% of the classified citations originate from independent researchers, indicating that the findings have been validated and utilized by the broader global scientific community outside the researcher's immediate network.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 4 · **1 flagged influential by Semantic Scholar**

CORE PAPER

Regulation of leukocyte recruitment by the long pentraxin PTX3

2010 · 523 citations (GS)

Field-normalised: 439 Semantic Scholar citations place it in the top 1% of Biology papers from 2010 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	The immunology of atherosclerosis (2017)	Karolinska University Hospital	Sweden	—
2	The Neutrophil's Role During Health and Disease. (2019)	—	—	—
3	Regulation of the inflammatory response in cardiac repair. (2012)	Albert Einstein College of Medicine	United States	—
4	Periodontitis increases the risk of gastrointestinal dysfunction: an update on the plausible pathogenic molecular mechanisms. (2025)	Iman Abdulrahman Bin Faizal University, Manipal Academy of Higher Education	India, Saudi Arabia	Influential

Independent citing papers only; self- and co-author citations excluded. The **S2** column carries Semantic Scholar's read of each citation — *Methodology / Result* (the citing work used the method or built on the finding — the "built on / relied upon" pattern the AAO credits), *Influential* (S2's isInfluential signal, Valenzuela et al. 2015), or *Background* (a passing mention).

D. Citing-Institution Prestige & Geography

Top citing institutions

Institution	Country	World ranking	Citing papers
University of North Carolina at Chapel Hill	United States	THE 78 · QS =140	1
Manipal Academy of Higher Education	India	THE 601–800	1
University of North Carolina	United States	—	1
Virginia Mason Medical Center	United States	SCImago #4520	1
Tongji University	China	SCImago #82 · THE =141 · QS =177	1
University of Kansas Medical Center	United States	SCImago #1982	1
Columbia University	United States	SCImago #65 · THE 20 · QS =38	1
Inserm	France	—	1
University of Wisconsin	United States	—	1
Institute of Cancer Research	China	SCImago #453	1
Karolinska University Hospital	Sweden	SCImago #671	1
Chiba University	Japan	SCImago #2218 · THE 1001–1200 · QS 791-800	1
University of Glasgow	United Kingdom	SCImago #351 · THE 84 · QS 79	1
Indiana University	United States	THE =198	1
Shanghai First Maternity and Infant Hospital	China	—	1

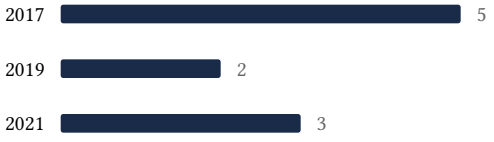
Geographic distribution of citing authors

Country	Citing papers
United States	7
China	4
France	1
India	1
Japan	1
Saudi Arabia	1
Spain	1
Sweden	1
United Kingdom	1
Czech Republic	1

Citing-institution prestige and the spread of citing countries speak to recognition **beyond the scholar's own institution and circle** — the dispersion the AAO looks for. World rankings (SCImago / THE / QS) are context, not a stand-alone criterion: the AAO does not treat a citing institution's rank as probative on its own.

E. Citation Growth Over Time

Distinct citing papers by publication year. Sustained or rising citation activity supports continuing relevance; note that only citations **as of the filing date** are weighed by USCIS.



F. AAO Precedent Considerations

Pre-filing self-check (AAO denial patterns)

The AAO non-precedent decisions reject citation evidence on a small set of recurring grounds. Confirm the petition addresses each before filing:

- ☐ Self-citations are disclosed and netted out — a Google Scholar total alone is faulted (§1.1).
- ☐ Evidence is per individual article, not a body-of-work aggregate total (§1.2).
- ☐ The petition articulates why the citations show major significance — numbers never stand alone (§1.5).
- ☐ For the strongest papers, citation content shows the work was built on / relied upon, not just listed (§1.6, §2.2).
- ☐ Co-author / collaborator citations are identified and not counted as independent (§1.7).
- ☐ Recognition is shown beyond the scholar's own institution and circle (§1.8).
- ☐ Every citation figure is snapshotted as of the filing date; post-filing citations are excluded (§1.9).
- ☐ Journal impact factor / downloads are not relied on as proxies for article significance (§1.10, §1.12).
- ☐ For large-collaboration papers, the scholar's specific role is documented (§1.13).

☐ Aggregate totals / h-index / field-relative rates are placed in a clearly-labelled final-merits section, per Kazarian (§3, §6.1.7).

Disclaimer

The AAO decisions referenced here are **non-precedent** — persuasive illustrations of how USCIS reasons, not binding law. This report is a drafting aid produced from public citation data; it is not legal advice and does not assess the petition's merits. All analysis must be reviewed by qualified immigration counsel.

G. Citation Evidence Index

Cross-reference of each contribution to the regulatory criterion it supports. Counsel should map these to the petition's exhibit numbers.

Contribution	Core paper	Indep. cites	Supports
Contribution 1	IL-33 Induces Antigen-Specific IL-5+ T Cells and Promotes Allergic-Induced Airway Inflammation Independent of IL-4	5	8 CFR 204.5(i)(3) — Outstanding Researcher
Contribution 2	The CXCL8/IL-8 chemokine family and its receptors in inflammatory diseases	1	8 CFR 204.5(i)(3) — Outstanding Researcher
Contribution 3	Regulation of leukocyte recruitment by the long pentraxin PTX3	4	8 CFR 204.5(i)(3) — Outstanding Researcher