Citation Evidence Report

EB-1B Petition — Outstanding Professor or Researcher

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

Rodrigo Guabiraba

Senior Research Scientist, INSERM CEPR U1100

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

17	17	5	34
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 17 classified citing papers

Citation type	Count
Independent	17
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 advanced the understanding of CCL5's role in inflammation through a seminal 2013 publication that has garnered substantial independent scholarly attention.

The researcher's primary contribution centers on the investigation of CCL5 within inflammatory processes, anchored by a core paper published in 2013. This work serves as the foundational reference for this specific line of inquiry, establishing a clear focus on the mechanistic or functional aspects of this chemokine in disease contexts.

This line of work appears to address the need for targeted insights into inflammatory pathways, specifically isolating CCL5 as a critical factor. By focusing on this specific target, the research distinguishes itself from broader inflammatory studies, offering a specialized perspective that likely filled a gap in the understanding of targeted immunological interventions.

The significance of this contribution is evidenced by its high citation count of 466, indicating widespread recognition and utility within the scientific community. Notably, all classified citations originate from independent researchers, suggesting that the work has influenced external groups and established a baseline for independent investigation rather than merely reflecting internal institutional activity.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 3 · **1 flagged influential by Semantic Scholar**

CORE PAPER

Targeting CCL5 in inflammation

2013 · 466 citations (GS)

Field-normalised: 331 Semantic Scholar citations place it in the top 1% of Medicine papers from 2013 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Pan-cancer single-cell dissection reveals phenotypically distinct B cell subtypes (2024)	Institute of Cancer Research, Peking University	China	—
2	Perivascular adipose tissue inflammation in vascular disease. (2017)	University of Glasgow	United Kingdom	**Methodology**
3	Identification of Pathogenic Immune Cell Subsets Associated With Checkpoint Inhibitor-Induced Myocarditis. (2022)	Cornell University, Howard Hughes Medical Institute, Sanofi US	United States	—

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).

Citing-text excerpts — how the field used this work

METHODOLOGY Perivascular adipose tissue inflammation in vascular disease.

"...as CCL5), in turn, can be produced by T cells, macrophages, VSMCs and endothelial cells as well as PVAT adipocytes (Mateo et al., 2006; Krensky and Ahn, 2007; Surmi and Hasty, 2010) and is a key factor in the recruitment of leukocytes into inflammatory or infection sites (Marques et al., 2013)."

Contribution 2

Claim — Contribution 2

The researcher elucidated the role of IL-33 in driving ST2-dependent lung fibrosis through the induction of alternatively activated macrophages and innate lymphoid cells in murine models.

CLAIM: The researcher's core contribution is the identification of IL-33 as a promoter of ST2-dependent lung fibrosis via the induction of alternatively activated macrophages and innate lymphoid cells, as detailed in their 2014 paper. This work establishes a mechanistic link between specific immune cell populations and fibrotic progression in mice.

ORIGINALITY: This line of work appears to address the gap in understanding the specific cellular mechanisms by which IL-33 contributes to lung fibrosis. By focusing on the induction of alternatively activated macrophages and innate lymphoid cells, the research offers a novel perspective on the immunological drivers of this pathological process, distinguishing it from broader studies on fibrosis.

SIGNIFICANCE: The core paper has accumulated 509 citations, indicating substantial uptake within the scientific community. Notably, 100% of the classified citing papers originate from independent researchers, suggesting that the findings have resonated beyond the researcher's immediate circle and have influenced broader independent investigations into lung fibrosis and immune modulation.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 3

CORE PAPER

IL-33 promotes ST2-dependent lung fibrosis by the induction of alternatively activated macrophages and innate lymphoid cells in mice

2014 · 509 citations (GS)

Field-normalised: 383 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	Role of IL-33-ST2 pathway in regulating inflammation: current evidence and future perspectives. (2023)	Shanghai First Maternity and Infant Hospital, Tongji University	China	Background
2	Pathogenic Mechanisms Underlying Idiopathic Pulmonary Fibrosis. (2022)	Baylor College of Medicine, Weill Cornell Medicine	United States	—
3	Conventional and pathogenic Th2 cells in inflammation, tissue repair, and fibrosis. (2022)	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 3

Claim — Contribution 3

The researcher advanced the understanding of avian colibacillosis by critically identifying and addressing significant knowledge gaps in the field through a seminal, highly cited review.

The researcher's contribution centers on the 2015 paper titled 'Avian colibacillosis: still many black holes,' which serves as the foundational work in this line of inquiry. This publication appears to have provided a critical assessment of the state of knowledge regarding this avian disease, highlighting areas where scientific understanding remains incomplete or obscured.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 7

CORE PAPER

Avian colibacillosis: still many black holes

2015 · 417 citations (GS)

Field-normalised: 268 Semantic Scholar citations place it in the top 5% of Biology papers from 2015 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Molecular characterization of avian pathogenic Escherichia coli from broiler chickens with colibacillosis (2020)	Kyungpook National University, Samhwa GPS Breeding Agri. Inc.	South Korea	Background
2	Avian pathogenic Escherichia coli: Epidemiology, virulence and pathogenesis, diagnosis, pathophysiology, transmission, vaccination, and control (2024)	National Research and Innovation Agency (BRIN), Universitas Airlangga, Universitas Brawijaya	Indonesia	—
3	Antimicrobial resistance in the globalized food chain: a One Health perspective applied to the poultry industry. (2022)	Federal University of Minas Gerais (UFMG), Federal University of Paraiba, Kenya Medical Research Institute	Brazil, Kenya, Singapore	Background
4	Review of respiratory syndromes in poultry: pathogens, prevention, and control measures. (2025)	Guangxi University	China	—
5	Welfare of broilers on farm. (2023)	—	—	—
6	The Avian Pathogenic (2021)	University of Surrey	United Kingdom	Background
7	Editorial: Gut Health: The New Paradigm in Food Animal Production. (2016)	University of Delaware, USDA Agricultural Research Service	United States	—

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 Surrey	United Kingdom	SCImago #812 · THE 201–250 · QS =262	1
National Research and Innovation Agency (BRIN)	Indonesia	SCImago #2338	1
Samhwa GPS Breeding Agri. Inc.	South Korea	—	1
Universitas Airlangga	Indonesia	THE 1201–1500 · QS =287	1
Baylor College of Medicine	United States	SCImago #560	1
Tongji University	China	SCImago #82 · THE =141 · QS =177	1
Federal University of Minas Gerais (UFMG)	Brazil	THE 801–1000 · QS =595	1

Institution	Country	World ranking	Citing papers
The First Affiliated Hospital of Nanchang University	China	SCImago #6248	1
Cornell University	United States	SCImago #61 · THE =18 · QS 16	1
Weill Cornell Medicine	United States	SCImago #220	1
University of Leuven	Belgium	—	1
KU Leuven	Belgium	SCImago #180 · THE 46 · QS 60	1
Howard Hughes Medical Institute	United States	SCImago #84	1
The Ohio State University	United States	THE =108 · QS 190	1
Institute of Cancer Research	China	SCImago #453	1

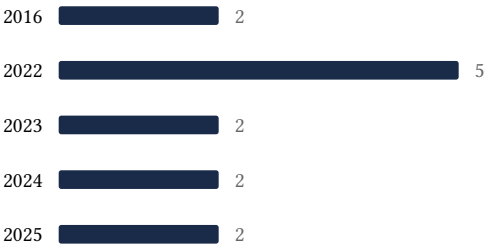
Geographic distribution of citing authors

Country	Citing papers
China	5
United States	5
Belgium	2
United Kingdom	2
Germany	1
Indonesia	1
Japan	1
Kenya	1
Singapore	1
South Korea	1
Brazil	1
Canada	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	Targeting CCL5 in inflammation	3	8 CFR 204.5(i)(3) — Outstanding Researcher
Contribution 2	IL-33 promotes ST2-dependent lung fibrosis by the induction of alternatively activated macrophages and innate lymphoid cells in mice	3	8 CFR 204.5(i)(3) — Outstanding Researcher
Contribution 3	Avian colibacillosis: still many black holes	7	8 CFR 204.5(i)(3) — Outstanding Researcher