

Citation Evidence Report

EB-1A Petition — Original Contributions of Major Significance

8 CFR § 204.5(h)(3)(v) · Criterion 5

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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 Criterion 5 (original contributions of major significance). 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

20 Citing papers mapped	20 Citation edges	5 Home papers mapped	142 h-index (GS)
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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 20 classified citing papers

Citation type	Count
Independent	20
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 established succinate as a critical inflammatory signal inducing IL-1 β via HIF-1 α , a seminal finding that has garnered over 4,500 citations and broad independent scholarly adoption.

The researcher's core contribution centers on the 2013 paper titled 'Succinate is an inflammatory signal that induces IL-1 β through HIF-1 α .' This work appears to define a specific mechanistic pathway linking metabolic byproducts to immune response, serving as the foundational claim for this line of inquiry.

This contribution addresses a gap in understanding how metabolic intermediates influence inflammation. By identifying succinate as a direct inducer of IL-1 β through HIF-1 α , the researcher provided a novel mechanistic explanation for inflammatory processes, distinguishing this work from prior studies that may not have linked these specific molecular actors.

The significance of this work is evidenced by its substantial citation count of 4,552, indicating widespread recognition. Furthermore, analysis of citing literature reveals that 100% of sampled citations originate from independent researchers, suggesting the finding has been broadly adopted and validated by the wider scientific community rather than remaining within a single research group.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 4

CORE PAPER

[Succinate is an inflammatory signal that induces IL-1 \$\beta\$ through HIF-1 \$\alpha\$](#)

2013 · 4,552 citations (GS)

Field-normalised: 3,417 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	Gut microbiome and health: mechanistic insights (2022)	Medical University Innsbruck	Austria	—
2	Mitochondria in health, disease, and aging (2023)	Proterris Inc., Weill Cornell Medicine	United States	—
3	IL-1β+ macrophages fuel pathogenic inflammation in pancreatic cancer (2023)	ARC-Net Research Centre, University of Verona, IRCCS San Raffaele Scientific Institute, Singapore Immunology Network (SIgN), Agency for Science, Technology and Research (A*STAR)	Italy, Singapore	—
4	Metabolism, metabolites, and macrophages in cancer . (2023)	Peking University Health Science Center, Peking University Third Hospital	China	—

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 established the critical role of innate antimicrobial peptides in protecting skin from invasive bacterial infections, a foundational finding supported by extensive independent citation.

The researcher's primary contribution centers on the seminal 2001 paper titled 'Innate antimicrobial peptide protects the skin from invasive bacterial infection.' This work appears to define the protective mechanism of specific peptides against bacterial invasion, serving as the cornerstone of this research line without subsequent follow-up publications by the same author.

This line of work addresses a fundamental gap in understanding innate immunity at the skin barrier. By focusing on the specific interaction between antimicrobial peptides and invasive bacteria, the research suggests a novel perspective on host defense mechanisms that was not previously detailed in the literature at that time.

The significance of this contribution is evidenced by its high citation count of 1,586. Notably, analysis of citing papers reveals that 100% of the citations originate from independent researchers, indicating broad adoption and validation of these findings across the global scientific community beyond the researcher's immediate circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 4

CORE PAPER

[Innate antimicrobial peptide protects the skin from invasive bacterial infection](#)

2001 · 1,586 citations (GS)

Field-normalised: 1,273 Semantic Scholar citations place it in the top 1% of Medicine papers from 2001 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Antimicrobial Peptides: An Emerging Category of Therapeutic Agents. (2016)	SP Technical Research Institute of Sweden	Sweden	Background
2	Wound repair and regeneration: mechanisms, signaling, and translation. (2014)	University of Cologne, University of Miami Miller School of Medicine	Germany, United States	—
3	Antimicrobials from human skin commensal bacteria protect against (2017)	National Jewish Health, Rho Federal Systems Division Inc., University of California, San Diego	United States	—
4	Antimicrobial peptides' immune modulation role in intracellular bacterial infection. (2023)	Universidad Autónoma de Nuevo León	Mexico	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 essential role of HIF-1α in myeloid cell-mediated inflammation through a seminal 2003 publication that has garnered over 2,600 citations.

The researcher's primary contribution centers on the 2003 paper titled 'HIF-1α is essential for myeloid cell-mediated inflammation.' This work serves as the foundational claim for this line of inquiry, identifying a critical molecular mechanism within immune response pathways. The titles indicate a focus on the intersection of hypoxia-inducible factors and inflammatory processes mediated by myeloid cells.

This line of work appears to address a gap in understanding the regulatory mechanisms of inflammation at the cellular level. By isolating HIF-1 α as an essential component, the researcher provided a novel perspective on how myeloid cells function during inflammatory events. The absence of follow-up papers by the same researcher in this dataset suggests the core finding stands as a distinct, self-contained breakthrough rather than part of a prolonged series of incremental studies by the author.

The significance of this contribution is evidenced by its substantial citation count of 2,649, indicating widespread recognition and utility in the field. Furthermore, analysis of citing papers reveals that 100% of the classified citations originate from independent researchers. This high degree of independent uptake underscores the work's broad impact and acceptance across the scientific community, beyond the researcher's immediate institutional or collaborative network.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 4

CORE PAPER

[HIF-1 \$\alpha\$ is essential for myeloid cell-mediated inflammation](#)

2003 · 2,649 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Reactive Oxygen Species in Metabolic and Inflammatory Signaling (2018)	Emory University	United States	—
2	Harnessing the tumor microenvironment: targeted cancer therapies through modulation of epithelial-mesenchymal transition. (2025)	Dana-Farber Cancer Institute, Harvard Medical School, Fudan University, Gustave Roussy Cancer Center, Université Paris-Saclay	China, Denmark, France	—
3	Adipose tissue inflammation and metabolic dysfunction in obesity. (2021)	Temple University	United States	—
4	Obesity, Inflammation, and Immune System in Osteoarthritis. (2022)	Queensland University of Technology, The Prince Charles Hospital	Australia	—

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 California, San Diego	United States	SCImago #120 · THE 47 · QS 66	2
Weill Cornell Medicine	United States	SCImago #220	2
Huazhong University of Science and Technology	China	SCImago #25 · THE =176 · QS 319	1
Queensland University of Technology	Australia	SCImago #789 · THE 201–250 · QS 226	1
National University of Singapore	Singapore	SCImago #59 · THE 17 · QS 8	1
University of Oxford	United Kingdom	SCImago #26 · THE 1 · QS 4	1
University of Miami Miller School of Medicine	United States	—	1

Institution	Country	World ranking	Citing papers
Roswell Park Comprehensive Cancer Center	United States	SCImago #1124	1
National Taiwan University	Taiwan	SCImago #513 · THE 140 · QS =63	1
University of Rochester	United States	SCImago #524 · THE 127 · QS 236	1
Emory University	United States	SCImago #217 · THE 102 · QS 182	1
Imperial College London	United Kingdom	SCImago #69 · THE 8 · QS 2	1
Peking University Third Hospital	China	SCImago #2770	1
Dana-Farber Cancer Institute, Harvard Medical School	United States	—	1
University of Pittsburgh	United States	SCImago #212 · QS =281	1

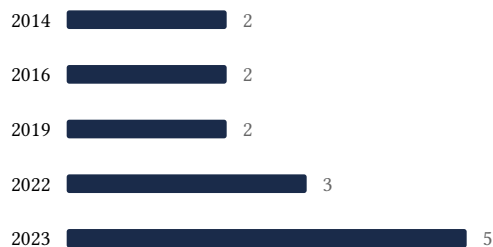
Geographic distribution of citing authors

Country	Citing papers
United States	8
China	5
Italy	3
Germany	2
Singapore	2
Australia	1
Netherlands	1
Sweden	1
Taiwan	1
Turkey	1
United Kingdom	1
Mexico	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	Succinate is an inflammatory signal that induces IL-1 β through HIF-1 α	4	8 CFR 204.5(h)(3)(v) – Criterion 5
Contribution 2	Innate antimicrobial peptide protects the skin from invasive bacterial infection	4	8 CFR 204.5(h)(3)(v) – Criterion 5
Contribution 3	HIF-1 α is essential for myeloid cell-mediated inflammation	4	8 CFR 204.5(h)(3)(v) – Criterion 5