

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

17	17	5	32
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 elucidated how chemokines and mitochondrial products activate neutrophils to amplify organ injury during acute liver failure, establishing a key mechanism in inflammatory pathology.

CLAIM: The researcher’s core contribution is defined by the 2012 paper titled ‘Chemokines and mitochondrial products activate neutrophils to amplify organ injury during mouse acute liver failure,’ which identifies specific molecular triggers for neutrophil activation in liver injury.

ORIGINALITY: This work appears to address the mechanistic gap in understanding how innate immune cells are recruited and activated during acute liver failure. By linking mitochondrial products and chemokines to neutrophil activity, the research suggests a novel pathway for amplifying organ damage, distinct from previously known inflammatory routes.

SIGNIFICANCE: With 400 citations, the paper is highly influential in the field. Notably, 100% of the classified citing papers originate from independent researchers, indicating that the findings have been widely adopted and validated by the broader scientific community outside the researcher’s immediate network.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 2

CORE PAPER

[Chemokines and mitochondrial products activate neutrophils to amplify organ injury during mouse acute liver failure](#)

2012 · 400 citations (GS)

Field-normalised: 313 Semantic Scholar citations place it in the top 5% of Medicine papers from 2012 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Acetaminophen Hepatotoxicity . (2019)	University of Kansas Medical Center	United States	—
2	Trial Watch: Toll-like receptor agonists in cancer immunotherapy . (2018)	Hospital Universitario Morales Meseguer, Inserm, Memorial Sloan Kettering Cancer Center	Czech Republic, France, Spain	—

Independent citing papers only; self- and co-author citations excluded. The S2 column flags citations Semantic Scholar identifies as *influential* — ones that substantively build on the work (S2’s isInfluential signal, Valenzuela et al. 2015) — the “built on / relied upon” pattern the AAO credits. Counsel should quote the citing text for the strongest of these.

Contribution 2

Claim – Contribution 2

The researcher functionally characterized the CrzA homologue in Aspergillus fumigatus, establishing a foundational understanding of this key regulatory protein in fungal pathogenesis.

The researcher’s contribution centers on the functional characterization of the CrzA homologue in *Aspergillus fumigatus*, as detailed in their 2008 paper. This work serves as the core reference for this line of inquiry, with no subsequent follow-up papers by the same author listed in the provided data.

This research appears to address the need for detailed molecular insights into the regulatory mechanisms of *A. fumigatus*. By focusing on the CrzA homologue, the work likely provided novel functional data that was previously lacking, thereby clarifying the role of this specific protein in the organism’s biology.

The significance of this contribution is evidenced by its 198 citations, indicating substantial uptake by the scientific community. Notably, 100% of the classified citing papers originate from independent researchers, suggesting that the work has had a broad and unbiased impact across the field, rather than being driven by internal or collaborative networks.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 4

CORE PAPER

Functional characterization of the *Aspergillus fumigatus* CRZ1 homologue, CrzA

2008 · 198 citations (GS)

Field-normalised: 164 Semantic Scholar citations place it in the top 10% of Biology papers from 2008 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Aspergillus fumigatus and Aspergillosis in 2019. (2019)	University of Crete	Greece	—
2	Regulatory circuitry governing fungal development, drug resistance, and disease. (2011)	University of Toronto	Canada	—
3	Moulding the mould: understanding and reprogramming filamentous fungal growth and morphogenesis for next generation cell factories. (2019)	Tianjin Institute of Industrial Biotechnology, Chinese Academy of Sciences	China	—
4	Recent developments in (2025)	Geisel School of Medicine at Dartmouth	United States	—

Independent citing papers only; self- and co-author citations excluded. The S2 column flags citations Semantic Scholar identifies as *influential* — ones that substantively build on the work (S2's isInfluential signal, Valenzuela et al. 2015) — the “built on / relied upon” pattern the AAO credits. Counsel should quote the citing text for the strongest of these.

Contribution 3

Claim – Contribution 3

The researcher elucidated the mechanistic link between NLRP3 inflammasome activation and leukotriene B4-mediated neutrophil recruitment in gout, establishing a critical pathway for hypernociception.

CLAIM: The researcher’s core contribution is defined by the 2012 publication titled ‘NLRP3 inflammasome–mediated neutrophil recruitment and hypernociception depend on leukotriene B4 in a murine model of gout’ This work appears to establish a specific molecular dependency in the inflammatory response associated with gout.

ORIGINALITY: Based on the title, this line of work addresses the mechanistic gap between inflammasome activation and downstream inflammatory mediators. It suggests a novel role for leukotriene B4 as a necessary mediator for neutrophil recruitment and pain signaling in this context, distinguishing it from broader inflammasome studies.

SIGNIFICANCE: The core paper has accumulated 289 citations, indicating substantial uptake by the scientific community. Notably, 100% of the classified citing papers originate from independent researchers, suggesting that the findings have been widely adopted and built upon by external groups rather than primarily by the researcher’s own circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 4

CORE PAPER

NLRP3 inflammasome–mediated neutrophil recruitment and hypernociception depend on leukotriene B4 in a murine model of gout

2012 · 289 citations (GS)

Field-normalised: 249 Semantic Scholar citations place it in the top 5% of Medicine papers from 2012 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Dietary Fibers from Fruits and Vegetables and Their Health Benefits via Modulation of Gut Microbiota. (2019)	Chenguang Biotech Group Co., Ltd., Chinese Academy of Agricultural Sciences, University of Massachusetts	China, United States	—
2	Redox regulation of NLRP3 inflammasomes: ROS as trigger or effector? (2015)	Virginia Commonwealth University	United States	—
3	Inflammatory Response to Regulated Cell Death in Gout and Its Functional Implications. (2022)	Computation and Informatics in Biology and Medicine, University of Wisconsin-Madison, Madison, WI, United States, Fudan University, Guanghua Clinical Medical College, Shanghai University of Traditional Chinese Medicine, Shanghai, China	China, United States	—
4	Reactive oxygen species at the crossroads of inflammasome and inflammation. (2014)	University of Illinois Chicago	United States	—

Independent citing papers only; self- and co-author citations excluded. The S2 column flags citations Semantic Scholar identifies as *influential* — ones that substantively build on the work (S2's isInfluential signal, Valenzuela et al. 2015) — the “built on / relied upon” pattern the AAO credits. Counsel should quote the citing text for the strongest of these.

D. Citing-Institution Prestige & Geography

Top citing institutions

Institution	Country	World ranking	Citing papers
University of Toronto	Canada	SCImago #39 · THE 21 · QS 29	1
Ludwig-Maximilians-Universität München	Germany	SCImago #363 · QS =58	1
University of Pennsylvania	United States	SCImago #52 · THE 14 · QS 15	1
University of Crete	Greece	SCImago #2556 · THE 601–800 · QS =628	1
University of Kansas Medical Center	United States	SCImago #1982	1
Guanghua Clinical Medical College, Shanghai University of Traditional Chinese Medicine, Shanghai, China	China	—	1
Computation and Informatics in Biology and Medicine, University of Wisconsin-Madison, Madison, WI, United States	United States	—	1
Virginia Commonwealth University	United States	SCImago #938 · THE 401–500 · QS 901-950	1
Chongqing Medical University	China	SCImago #1049	1

Institution	Country	World ranking	Citing papers
Laval University	Canada	SCImago #966 · THE 401–500 · QS =469	1
Inserm	France	—	1
Yale University	United States	SCImago #76 · THE 10 · QS 21	1
Sapienza University of Rome	Italy	THE =170 · QS 128	1
Fudan University	China	SCImago #46 · THE 36 · QS 30	1
Tianjin Institute of Industrial Biotechnology, Chinese Academy of Sciences	China	—	1

Geographic distribution of citing authors

Country	Citing papers
United States	9
China	4
Canada	2
Germany	2
Spain	2
Greece	1
Italy	1
Czech Republic	1
France	1
Sweden	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.

2014  3

2019  4

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	Chemokines and mitochondrial products activate neutrophils to amplify organ injury during mouse acute liver failure	2	8 CFR 204.5(i)(3) – Outstanding Researcher
Contribution 2	Functional characterization of the <i>Aspergillus fumigatus</i> CRZ1 homologue, CrzA	4	8 CFR 204.5(i)(3) – Outstanding Researcher
Contribution 3	NLRP3 inflammasome-mediated neutrophil recruitment and hypernociception depend on leukotriene B4 in a murine model of gout	4	8 CFR 204.5(i)(3) – Outstanding Researcher