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	43
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 intravascular danger signals as key guides for neutrophil recruitment to sites of sterile inflammation, establishing a foundational mechanism in immunology.

The researcher's primary contribution centers on the seminal 2010 paper titled 'Intravascular danger signals guide neutrophils to sites of sterile inflammation.' This work appears to define a specific biological mechanism wherein danger signals within blood vessels direct immune cells to areas of non-infectious tissue damage. The titles indicate a focus on the precise navigation of neutrophils, suggesting a novel understanding of how the immune system responds to sterile injury rather than pathogen invasion.

This line of work addresses a critical gap in understanding sterile inflammation, a process distinct from infectious responses. By isolating intravascular signals as the guiding force, the research offers a new perspective on neutrophil trafficking. The absence of follow-up papers by the same researcher in this dataset suggests this single publication stands as a definitive, self-contained contribution to the field, rather than part of an extended series of incremental studies.

The significance of this contribution is underscored by its substantial citation count of 1,487, indicating widespread recognition and utility within the scientific community. Furthermore, analysis of citing literature reveals that 100% of the classified citations originate from independent researchers. This high degree of independent uptake demonstrates that the work has been broadly adopted and built upon by the wider scientific community, validating its impact beyond the researcher's immediate circle.

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

CORE PAPER

Intravascular danger signals guide neutrophils to sites of sterile inflammation

2010 · 1,487 citations (GS)

Field-normalised: 1,189 Semantic Scholar citations place it in the top 1% of Medicine papers from 2010 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Molecular mechanisms of cell death: recommendations of the Nomenclature Committee on Cell Death 2018 (2018)	Albert Einstein College of Medicine, Albert-Ludwigs-University of Freiburg, Cancer Research UK Beatson Institute	Australia, Austria, Belgium	—
2	Trained immunity—basic concepts and contributions to immunopathology (2022)	Eindhoven University of Technology, Icahn School of Medicine at Mount Sinai, Massachusetts General Hospital	Netherlands, United States	—
3	DAMPs, PAMPs, and LAMPs in Immunity and Sterile Inflammation. (2020)	University of Calgary	Canada	**Influential**
4	The Neutrophil's Role During Health and Disease. (2019)	—	—	—

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 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 seminal 2012 paper identifies the activation of neutrophils by chemokines and mitochondrial products as a driver of organ injury in mouse models of acute liver failure. This work stands as the core contribution in this specific line of inquiry.

ORIGINALITY: The titles suggest this research addresses the mechanistic gap in understanding how specific molecular signals coordinate neutrophil activity to exacerbate tissue damage. By linking mitochondrial products and chemokines to neutrophil activation, the work appears to offer a novel perspective on the inflammatory cascade in liver failure.

SIGNIFICANCE: With 400 citations, the paper is highly influential in the field. Notably, 100% of the classified citing papers originate from independent researchers, indicating broad adoption and validation of these findings by the wider scientific community beyond the researcher's immediate circle.

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 3

Claim — Contribution 3

The researcher identified hepatic DNA deposition as a key driver of drug-induced liver injury and inflammation in mice, establishing a novel mechanistic link supported by substantial independent scholarly uptake.

CLAIM: The researcher's contribution centers on the 2015 paper titled 'Hepatic DNA deposition drives drug-induced liver injury and inflammation in mice,' which posits a direct causal role for DNA deposition in liver pathology. This work stands as the primary evidence for this specific line of inquiry, with no follow-up publications by the researcher listed in the provided data.

ORIGINALITY: The title suggests the work addresses a gap in understanding the molecular mechanisms underlying drug-induced liver injury. By highlighting 'hepatic DNA deposition' as a driver, the research appears to introduce a distinct pathological pathway or mechanism that was not previously established as a primary cause of inflammation in this context.

SIGNIFICANCE: The core paper has accumulated 200 citations, indicating significant engagement within the scientific community. Notably, 100% of the classified citing papers originate from independent researchers, demonstrating that the findings have been widely adopted and validated by peers outside the researcher's immediate institution or collaboration network.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 2

CORE PAPER

Hepatic DNA deposition drives drug-induced liver injury and inflammation in mice

2015 · 200 citations (GS)

Field-normalised: 162 Semantic Scholar citations place it in the top 5% of Medicine papers from 2015 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Phagocytosis of Necrotic Debris at Sites of Injury and Inflammation. (2019)	Hospital for Sick Children, KU Leuven	Belgium, Canada	—
2	Chemokine (C-C motif) receptor 2-positive monocytes aggravate the early phase of acetaminophen-induced acute liver injury. (2016)	NOXXON Pharma AG, Tobira Therapeutics, Inc., University Hospital Aachen	Belgium, Germany, 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 Calgary	Canada	SCImago #399 · THE 200 · QS 211	2
KU Leuven	Belgium	SCImago #180 · THE 46 · QS 60	2
Icahn School of Medicine at Mount Sinai	United States	SCImago #295	2
Zhejiang University	China	SCImago #6 · THE 39 · QS 49	2
Southern Medical University	China	SCImago #392 · THE 251–300	2
National University of Singapore	Singapore	SCImago #59 · THE 17 · QS 8	2
Guilin Medical University	China	SCImago #3920	2
First Affiliated Hospital of Harbin Medical University	China	—	2
University of Kansas Medical Center	United States	SCImago #1982	1
Weizmann Institute of Science	Israel	SCImago #739	1
Massachusetts General Hospital	United States	SCImago #100	1
Yale University School of Medicine	United States	—	1
National Institute of Neurological Disorders and Stroke	United States	SCImago #447	1
University of Ferrara	Italy	SCImago #2059 · THE 501–600 · QS 951-1000	1
University of South Australia	Australia	SCImago #2033	1

Geographic distribution of citing authors

Country	Citing papers
United States	5
China	5
Canada	4
Belgium	3
Singapore	2
Spain	2
France	2
Germany	2
Austria	2
Italy	1
Czech Republic	1
Israel	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	Intravascular danger signals guide neutrophils to sites of sterile inflammation	4	8 CFR 204.5(i)(3) — Outstanding Researcher
Contribution 2	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 3	Hepatic DNA deposition drives drug-induced liver injury and inflammation in mice	2	8 CFR 204.5(i)(3) — Outstanding Researcher