Citation Evidence Report

EB-1A Petition — Original Contributions of Major Significance

8 CFR § 204.5(h)(3)(v) · Criterion 5

Caio Fagundes

Universidade Federal de Minas Gerais

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

23	23	5	31
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 23 classified citing papers

Citation type	Count
Independent	23
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 that commensal microbiota is fundamental for the development of inflammatory pain, a finding supported by a seminal 2008 paper with 392 citations.

The researcher's contribution centers on the seminal 2008 paper titled 'Commensal microbiota is fundamental for the development of inflammatory pain.' This work appears to define a critical biological link between gut flora and pain mechanisms, standing as a foundational piece in this specific area of inquiry without subsequent follow-up papers by the same author listed here.

This line of work appears to address a gap in understanding the non-neural drivers of pain. By identifying commensal microbiota as fundamental, the research suggests a novel perspective on inflammatory pain etiology, shifting focus toward host-microbe interactions as a primary factor in pain development rather than solely neural or immune pathways.

The significance of this contribution is evidenced by its 392 citations, indicating substantial uptake by the scientific community. Notably, 100% of the classified citing papers originate from independent researchers, demonstrating that the work has influenced scholars outside the researcher's immediate institution and collaboration network, thereby confirming its broad independent impact.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 4

CORE PAPER

Commensal microbiota is fundamental for the development of inflammatory pain

2008 · 392 citations (GS)

Field-normalised: 290 Semantic Scholar citations place it in the top 5% of Biology papers from 2008 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	The Microbiota-Gut-Brain Axis (2019)	APC Microbiome Ireland, University College Cork	Ireland	—
2	From gut dysbiosis to altered brain function and mental illness: mechanisms and pathways (2016)	South Australian Health and Medical Research Institute, SUNY Upstate Medical University	Australia, United States	—
3	Gut microbiota in health and disease. (2010)	The University of British Columbia	Canada	—
4	The progress of gut microbiome research related to brain disorders. (2020)	Fudan University, Huashan Hospital, Fudan University, Karolinska Institutet	China, Sweden	—

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 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 paper titled 'NLRP3 inflammasome–mediated neutrophil recruitment and hypernociception depend on leukotriene B4 in a murine model of gout.' This work appears to identify a specific molecular dependency in the inflammatory response associated with gout.

ORIGINALITY: By linking the NLRP3 inflammasome to leukotriene B4 production, this line of work addresses the mechanistic gap between innate immune sensing and subsequent neutrophil-driven inflammation. The title suggests a novel causal chain where inflammasome activity directly influences leukotriene-mediated pain and cell recruitment, offering a refined understanding of gout pathophysiology beyond general inflammation.

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, demonstrating that this finding has served as a foundational reference for external investigators exploring inflammasome biology and gout mechanisms, rather than being confined to the researcher's immediate 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.

Contribution 3

Claim — Contribution 3

The researcher demonstrated that transient TLR activation restores inflammatory responses and bacterial control capabilities in germfree mice, addressing critical gaps in host-microbe immunity.

CLAIM: The researcher's core contribution is established by the 2012 paper titled 'Transient TLR activation restores inflammatory response and ability to control pulmonary bacterial infection in germfree mice.' This work stands as the primary evidence for this specific line of inquiry, with no subsequent follow-up papers by the same researcher listed in the provided data.

ORIGINALITY: The title suggests the work addresses the impaired immune function observed in germfree models. By focusing on transient Toll-like receptor (TLR) activation, the research appears to offer a novel mechanism for restoring the ability to control pulmonary bacterial infections, implying a targeted intervention rather than permanent microbial colonization.

SIGNIFICANCE: With 286 citations, the paper is highly cited within its 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 outside the researcher's immediate network.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 5

CORE PAPER

Transient TLR activation restores inflammatory response and ability to control pulmonary bacterial infection in germfree mice

2012 · 286 citations (GS)

Field-normalised: 227 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	Interaction between microbiota and immunity in health and disease (2020)	The First Affiliated Hospital, Sun Yat-sen University, University Medical Center Hamburg-Eppendorf, Weizmann Institute of Science	China, Germany, Israel	—
2	The Gut-Lung Axis in Health and Respiratory Diseases: A Place for Inter-Organ and Inter-Kingdom Crosstalks. (2020)	CHU de Bordeaux, Clinique Saint Pierre, Lausanne University Hospital and University of Lausanne	Belgium, France, Switzerland	—
3	Mouse Microbiota Models: Comparing Germ-Free Mice and Antibiotics Treatment as Tools for Modifying Gut Bacteria. (2018)	Baylor College of Medicine, Washington University School of Medicine	United States	—
4	The role and mechanism of gut-lung axis mediated bidirectional communication in the occurrence and development of chronic obstructive pulmonary disease. (2024)	Northwestern Polytechnical University, The First Affiliated Hospital of Xi'an Jiaotong University	China	—
5	The role of the lung microbiota and the gut-lung axis in respiratory infectious diseases. (2018)	Université de Toulouse	France	—

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
Fudan University	China	SCImago #46 · THE 36 · QS 30	2
Washington University School of Medicine	United States	—	2
University of Pennsylvania	United States	SCImago #52 · THE 14 · QS 15	1
Baylor College of Medicine	United States	SCImago #560	1
Hospital Universitário Clementino Fraga Filho, Universidade Federal do Rio de Janeiro	Brazil	—	1
University Medical Center Hamburg-Eppendorf	Germany	SCImago #743	1
Northwestern Polytechnical University	China	SCImago #203 · THE 251–300 · QS =499	1
University of Oxford	United Kingdom	SCImago #26 · THE 1 · QS 4	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
University of Bordeaux	France	THE 401–500 · QS =494	1
Weizmann Institute of Science	Israel	SCImago #739	1
The University of British Columbia	Canada	SCImago #144 · THE 45 · QS 40	1
The First Affiliated Hospital of Xi'an Jiaotong University	China	SCImago #2010	1

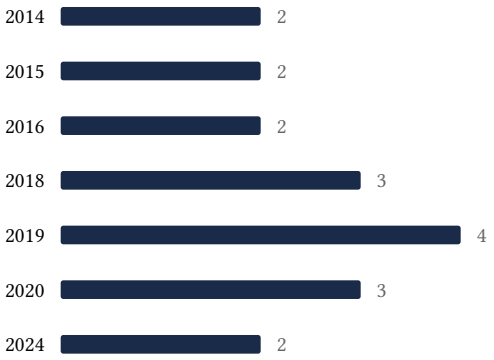
Geographic distribution of citing authors

Country	Citing papers
United States	13
China	7
France	2
Canada	1
Germany	1
India	1
Australia	1
Ireland	1
Israel	1
Pakistan	1
South Korea	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.



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	Commensal microbiota is fundamental for the development of inflammatory pain	4	8 CFR 204.5(h)(3)(v) — Criterion 5
Contribution 2	NLRP3 inflammasome–mediated neutrophil recruitment and hypernociception depend on leukotriene B4 in a murine model of gout	4	8 CFR 204.5(h)(3)(v) — Criterion 5
Contribution 3	Transient TLR activation restores inflammatory response and ability to control pulmonary bacterial infection in germfree mice	5	8 CFR 204.5(h)(3)(v) — Criterion 5