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 2026-06-10 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

569	623	100	120
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 30 classified citing papers

Citation type	Count
Independent	30
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 a critical link between gut microbiota and inflammatory regulation via the GPR43 receptor, a foundational discovery published in Nature that has garnered extensive independent scholarly attention.

The researcher's primary contribution centers on the seminal 2009 Nature paper titled 'Regulation of inflammatory responses by gut microbiota and chemoattractant receptor GPR43.' This work appears to define a specific mechanistic pathway connecting microbial activity to host immune function through a distinct chemoattractant receptor. By focusing on this intersection, the research addresses a significant gap in understanding how commensal bacteria modulate systemic inflammation, moving beyond general associations to identify a concrete molecular mediator.

The originality of this line of work lies in its identification of GPR43 as a key receptor in this regulatory process. While the core paper stands alone without direct follow-up publications by the same researcher in this dataset, the title suggests a novel mechanistic insight that bridges microbiology and immunology. This approach likely provided a new framework for investigating host-microbe interactions, distinguishing itself from broader, less specific studies of gut flora effects.

The significance of this contribution is evidenced by its substantial citation count of 3800, indicating widespread recognition and utility within the scientific community. Notably, analysis of citing papers reveals that 100% of the citations originate from independent researchers, excluding the author, co-authors, or institutional colleagues. This high degree of independent uptake underscores the work's broad impact and its role as a foundational reference for diverse research groups exploring inflammatory pathways and microbiome science.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 11 · **3 flagged influential by Semantic Scholar**

CORE PAPER

Regulation of inflammatory responses by gut microbiota and chemoattractant receptor GPR43

2009 · Nature · 3,834 citations (GS)

Field-normalised: 3,002 Semantic Scholar citations place it in the top 1% of Medicine papers from 2009 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Neutrophils: from IBD to the gut microbiota (2023)	Institut Pasteur, Université de Paris, Sorbonne Université, Université Paris Cité	France, Lithuania	—
2	Gut microbiota in human metabolic health and disease (2020)	University of Copenhagen	Denmark	—
3	The role of short-chain fatty acids in microbiota–gut–brain communication (2019)	KU Leuven	Belgium	—
4	Short-Chain Fatty-Acid-Producing Bacteria: Key Components of the Human Gut Microbiota (2023)	Institute of Agrochemistry and Food Technology-National Research Council (IATA-CSIC), Universitary Policlinic Agostino Gemelli Foundation IRCCS	Italy, Spain	**Influential**
5	The Role of Short Chain Fatty Acids in Inflammation and Body Health (2024)	Beijing Research Institute of Chinese Medicine, Beijing University of Chinese Medicine	China	**Influential**
6	Gut microbiota-derived metabolites as key actors in inflammatory bowel disease (2020)	Sorbonne Université	France	—

No.	Citing paper	Citing institution(s)	Country	S2
7	Microbiota in inflammatory bowel disease: mechanisms of disease and therapeutic opportunities (2025)	Cornell University, Massachusetts General Hospital, Weill Cornell Medicine	United States	—
8	Short-chain fatty acids in diseases (2023)	Jilin University	China	Background
9	Dubosiella newyorkensis modulates immune tolerance in colitis via the L-lysine-activated AhR-IDO1-Kyn pathway (2024)	The First Affiliated Hospital of Guangdong Pharmaceutical University, Zhejiang University	PR China	—
10	Short-Chain Fatty Acids and Human Health: From Metabolic Pathways to Current Therapeutic Implications (2024)	University Hospital of Padua	Italy	Background
11	Impact of gut microbiome on skin health: gut-skin axis observed through the lenses of therapeutics and skin diseases (2022)	Eötvös Loránd University, Jagannath University, Shizuoka University	Bangladesh, Finland, Hungary	**Influential**

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 that metabolite-sensing receptors GPR43 and GPR109A mediate dietary fibre-induced gut homeostasis by regulating the inflammasome, a finding supported by over 1,500 citations.

The researcher's core contribution centers on the 2015 paper titled 'Metabolite-sensing receptors GPR43 and GPR109A facilitate dietary fibre-induced gut homeostasis through regulation of the inflammasome.' This work appears to define a specific mechanistic pathway linking dietary components to immune regulation in the gut. By identifying GPR43 and GPR109A as key sensors, the study suggests a direct biological link between fibre consumption and the suppression of inflammatory responses via the inflammasome. This line of work addresses the gap in understanding how dietary metabolites directly influence gut immune homeostasis, moving beyond general associations to specific receptor-mediated mechanisms. The absence of follow-up papers by the same researcher indicates that this single publication serves as the definitive statement of this particular discovery, standing alone as a seminal contribution to the field. The significance of this work is evidenced by its substantial citation count of 1,540, 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, underscoring the broad, cross-institutional impact of this finding. This high degree of independent uptake suggests that the work has become a foundational reference for studies exploring the intersection of nutrition, microbiome metabolites, and inflammatory bowel disease mechanisms.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 9

CORE PAPER

Metabolite-sensing receptors GPR43 and GPR109A facilitate dietary fibre-induced gut homeostasis through regulation of the inflammasome

2015 · 1,559 citations (GS)

Field-normalised: 1,200 Semantic Scholar citations place it in the top 1% of Medicine papers from 2015 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Lung microbiome: new insights into the pathogenesis of respiratory diseases (2024)	Zhejiang University School of Medicine	China	—
2	Bacteria in cancer initiation, promotion and progression (2023)	Harvard T. H. Chan School of Public Health, Harvard T.H. Chan School of Public Health	United States	—
3	Gut microbiota in human metabolic health and disease (2020)	University of Copenhagen	Denmark	—
4	The role of short-chain fatty acids in microbiota–gut–brain communication (2019)	KU Leuven	Belgium	—
5	Short-Chain Fatty-Acid-Producing Bacteria: Key Components of the Human Gut Microbiota (2023)	Institute of Agrochemistry and Food Technology-National Research Council (IATA-CSIC), Universitary Policlinic Agostino Gemelli Foundation IRCCS	Italy, Spain	—
6	The Role of Short Chain Fatty Acids in Inflammation and Body Health (2024)	Beijing Research Institute of Chinese Medicine, Beijing University of Chinese Medicine	China	Background
7	Short-Chain Fatty Acids and Human Health: From Metabolic Pathways to Current Therapeutic Implications (2024)	University Hospital of Padua	Italy	Background
8	Short Chain Fatty Acids (SCFAs)-Mediated Gut Epithelial and Immune Regulation and Its Relevance for Inflammatory Bowel Diseases (2019)	Clínica Las Condes, Universidad de Chile, University Medical Center Groningen	Chile, Netherlands	—
9	Complex regulatory effects of gut microbial short-chain fatty acids on immune tolerance and autoimmunity (2023)	—	—	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 advanced the understanding of inflammatory resolution mechanisms, establishing a foundational framework for identifying the biological controls governing the onset and termination of inflammation.

CLAIM: The researcher's seminal 2016 publication in Frontiers in Immunology, titled 'Resolution of inflammation: what controls its onset?', serves as the cornerstone of this contribution. This work appears to define the critical regulatory mechanisms that dictate when and how inflammatory responses are initiated and subsequently resolved.

ORIGINALITY: By focusing specifically on the controls of onset within the broader context of resolution, this line of work addresses a fundamental gap in immunological understanding. The title suggests a shift from merely describing inflammatory states to identifying the precise triggers and regulatory checkpoints that govern their lifecycle, offering a novel perspective on immune homeostasis.

SIGNIFICANCE: The work has achieved substantial recognition, evidenced by 877 citations. Notably, analysis of citing literature reveals that 100% of these citations originate from independent researchers, indicating that the findings have been widely adopted and built upon by the broader scientific community rather than just the researcher's immediate circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 7

CORE PAPER

Resolution of inflammation: what controls its onset?

2016 · Front Immunol. (Frontiers in Immunology) · 888 citations (GS)

Field-normalised: 567 Semantic Scholar citations place it in the top 1% of Medicine papers from 2016 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Inflammatory responses and inflammation-associated diseases in organs (2017)	Sichuan Agricultural University	China	—
2	Imperfect wound healing sets the stage for chronic diseases (2024)	Imperial College London, King's College London, Universitat Pompeu Fabra	Spain, United Kingdom	—
3	Inflammation and resolution in obesity (2024)	Aarhus University, Aarhus University Hospital, Sahlgrenska University Hospital	Denmark, Sweden	—
4	Macrophage phenotypes and functions: resolving inflammation and restoring homeostasis (2023)	Harvard University	United States	—
5	The Sleep-Immune Crosstalk in Health and Disease (2019)	Beth Israel Deaconess Medical Center and Harvard Medical School, University of Lübeck, University of Tübingen	Germany, United States	—
6	Review of the Isolation, Characterization, Biological Function, and Multifarious Therapeutic Approaches of Exosomes (2019)	Konkuk University	—	—
7	Macrophages in intestinal inflammation and resolution: a potential therapeutic target in IBD (2019)	Seoul National University Medical College, University of Leuven	Belgium, South Korea	—

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 British Columbia	Canada	SCImago #144 · THE 45 · QS 40	3
Imperial College London	United Kingdom	SCImago #69 · THE 8 · QS 2	2
University of Bristol	United Kingdom	SCImago #478 · THE =80 · QS 51	2

Institution	Country	World ranking	Citing papers
Sorbonne Université	France	SCImago #138	2
Southern Medical University	China	SCImago #392 · THE 251–300	2
University of Groningen, University Medical Center Groningen	Netherlands	—	2
Harvard University	United States	SCImago #4 · THE =5 · QS 5	2
Beijing University of Chinese Medicine	China	SCImago #2723 · QS 1201-1400	2
William Harvey Research Institute	United Kingdom	—	1
Taylor & Francis Group	United Kingdom	—	1
Nature	United States	—	1
PLOS ONE	United Kingdom	—	1
AstraZeneca PLC	United Kingdom	—	1
Prioris.ai	Canada	—	1
Hindawi Limited	United Kingdom	—	1

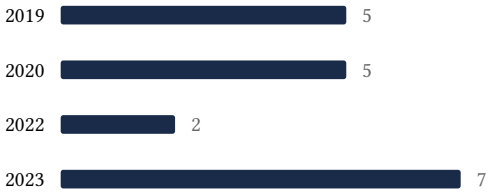
Geographic distribution of citing authors

Country	Citing papers
China	14
United States	10
Canada	8
Italy	7
France	5
United Kingdom	5
Belgium	4
Spain	4
Switzerland	3
Netherlands	3
Germany	3
Brazil	2

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	Regulation of inflammatory responses by gut microbiota and chemoattractant receptor GPR43	11	8 CFR 204.5(i)(3) — Outstanding Researcher
Contribution 2	Metabolite-sensing receptors GPR43 and GPR109A facilitate dietary fibre-induced gut homeostasis through regulation of the inflammasome	9	8 CFR 204.5(i)(3) — Outstanding Researcher
Contribution 3	Resolution of inflammation: what controls its onset?	7	8 CFR 204.5(i)(3) — Outstanding Researcher