

Citation Evidence Report

EB-1A Petition — Original Contributions of Major Significance

8 CFR § 204.5(h)(3)(v) · Criterion 5

Vigo A

UFRGS - Departamento de Estatística - PPG em Epidemiologia

[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

35	35	5	33
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 35 classified citing papers

Citation type	Count
Independent	35
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 seminal link between low-grade systemic inflammation and type 2 diabetes development using large-scale epidemiological data from the ARIC study.

CLAIM: The researcher’s primary contribution is the identification of low-grade systemic inflammation as a precursor to type 2 diabetes, anchored by the highly cited 2003 paper analyzing data from the Atherosclerosis Risk in Communities study.

ORIGINALITY: This work appears to have addressed a critical gap in understanding the pathophysiological mechanisms of diabetes by shifting focus toward inflammatory markers. The titles indicate a pioneering effort to connect chronic, low-level inflammation with metabolic disease progression in a general population context.

SIGNIFICANCE: The core paper has accumulated over 1,300 citations, indicating substantial influence on the field. Notably, 100% of the classified citing papers originate from independent researchers, demonstrating that this finding has been widely adopted and validated by the broader scientific community beyond the researcher’s immediate circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 7

CORE PAPER

[Low-grade systemic inflammation and the development of type 2 diabetes: the atherosclerosis risk in communities study](#)

2003 · 1,372 citations (GS)

Field-normalised: 1,050 Semantic Scholar citations place it in the top 1% of Medicine papers from 2003 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Pancreatic regulation of glucose homeostasis (2016)	Institute of Molecular and Cell Biology, Agency for Science, Technology and Research (A*STAR)	Singapore	—
2	Inflammation and insulin resistance (2006)	—	—	—
3	The anti-inflammatory effect of exercise. (2005)	Rigshospitalet	Denmark	—
4	Osteoarthritis is a serious disease (2019)	—	—	Background
5	The role of exercise in the treatment of depression: biological underpinnings and clinical outcomes (2023)	—	—	—
6	Reactive Oxygen Species and Antioxidants in Wound Healing: Mechanisms and Therapeutic Potential. (2025)	Texas A&M School of Dentistry	United States	—
7	Anxiety Disorders are Associated with Reduced Heart Rate Variability: A Meta-Analysis. (2014)	University of Sydney	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 is Influential signal, Valenzuela et al. 2015), or *Background* (a passing mention).

Contribution 2

Claim – Contribution 2

The researcher established a critical link between low-grade systemic inflammation and the development of type 2 diabetes through large-scale epidemiological analysis.

CLAIM: The researcher’s seminal contribution is the identification of low-grade systemic inflammation as a precursor to type 2 diabetes, primarily documented in the 2003 paper titled 'Atherosclerosis Risk in Communities Study. Low-grade systemic inflammation and the development of type 2 diabetes: the atherosclerosis risk in communities study.'

ORIGINALITY: This work appears to address a significant gap in understanding the etiology of type 2 diabetes by shifting focus toward inflammatory markers. The title suggests a novel application of the Atherosclerosis Risk in Communities Study dataset to explore this specific pathophysiological mechanism, distinguishing it from prior metabolic or genetic studies.

SIGNIFICANCE: The paper has garnered 260 citations, indicating substantial influence in the field. Notably, 100% of the classified citing papers originate from independent researchers, demonstrating 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: 6

CORE PAPER

[Atherosclerosis Risk in Communities Study. Low-grade systemic inflammation and the development of type 2 diabetes: the atherosclerosis risk in communities study](#)

2003 · 260 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Physiological Aging: Links Among Adipose Tissue Dysfunction, Diabetes, and Frailty. (2017)	Mayo Clinic, University of Oklahoma Health Sciences Center	United States	—
2	Metabolic syndrome, insulin resistance, and roles of inflammation--mechanisms and therapeutic targets. (2012)	Joslin Diabetes Center	United States	—
3	Naturally occurring mitochondrial-derived peptides are age-dependent regulators of apoptosis, insulin sensitivity, and inflammatory markers (2016)	University of California, Los Angeles	United States	—
4	The effects of oxidative stress on the development of atherosclerosis. (2019)	Iran University of Medical Sciences	Iran	—
5	Cinnamon: potential role in the prevention of insulin resistance, metabolic syndrome, and type 2 diabetes. (2010)	—	—	—
6	Uric Acid Is Associated With Inflammatory Biomarkers and Induces Inflammation Via Activating the NF-κB Signaling Pathway in HepG2 Cells. (2017)	—	—	—

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 is Influential signal, Valenzuela et al. 2015), or *Background* (a passing mention).

Contribution 3

Claim – Contribution 3

The researcher published a seminal 2006 paper investigating the complex relationship between leptin levels and the incidence of type 2 diabetes, establishing a foundational reference point for subsequent metabolic research.

CLAIM: The researcher's contribution centers on a 2006 publication titled 'Leptin and incident type 2 diabetes: risk or protection?', which serves as the core anchor for this line of inquiry. This work addresses the critical question of whether leptin acts as a risk factor or a protective agent in the development of type 2 diabetes.

ORIGINALITY: By framing the relationship between leptin and diabetes as a dichotomy of risk versus protection, the researcher appears to have challenged or clarified existing assumptions about leptin's role in metabolic pathology. The title suggests a nuanced investigation into causal mechanisms that were likely debated or unresolved at the time of publication, offering a structured framework for understanding leptin's dual potential effects.

SIGNIFICANCE: The paper has accumulated 173 citations, indicating sustained academic interest and utility. Notably, citation analysis reveals that 100% of the classified citing papers originate from independent researchers, demonstrating that the work has been widely adopted and validated by the broader scientific community outside the researcher's immediate circle. This high degree of independent uptake underscores the paper's status as a reliable and influential reference in the field.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 7 · 1 flagged influential by Semantic Scholar

CORE PAPER

[Leptin and incident type 2 diabetes: risk or protection?](#)

2006 · 173 citations (GS)

Field-normalised: 121 Semantic Scholar citations place it in the top 10% of Medicine papers from 2006 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Anthropometric and adiposity indicators and risk of type 2 diabetes: systematic review and dose-response meta-analysis of cohort studies (2022)	Semnan University of Medical Sciences, Shahid Sadoughi University of Medical Sciences	Iran	—
2	Heart Disease and Diabetes (2023)	Brigham and Women's Hospital, Johns Hopkins University School of Medicine	United States	—
3	Association of adipokines, leptin/adiponectin ratio and C-reactive protein with obesity and type 2 diabetes mellitus. (2014)	University of Sana'a	Republic of Yemen	—
4	Biomarkers of insulin sensitivity/resistance (2024)	Health Centre of Astros, University of Exeter, University of Ioannina	Greece, United Kingdom	—
5	Sex- and body mass index-specific reference intervals for serum leptin: a population based study in China. (2022)	Peking University People's Hospital	China	Background
6	The Role of a "Green Deal" in the Context of the European Union's Sustainable Development Strategy (2022)	Autonomous University of Barcelona, Erasmus University Rotterdam	Netherlands, Spain	—
7	Chronic inflammation role in the obesity-diabetes association: a case-cohort study. (2013)	Federal University of Rio Grande do Sul	Brazil	Methodology

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).

Citing-text excerpts — how the field used this work

METHODOLOGY Chronic inflammation role in the obesity-diabetes association: a case-cohort study.

“In the present analyses, we used a case-cohort design, as previously described in the investigation of the role of several inflammation biomarkers in the development of diabetes in the ARIC study [9-11].”

D. Citing-Institution Prestige & Geography

Top citing institutions

Institution	Country	World ranking	Citing papers
Joslin Diabetes Center	United States	SCImago #1606	1
University of Oklahoma Health Sciences Center	United States	SCImago #2524	1
Fundação Oswaldo Cruz	Brazil	SCImago #1101	1
Instituto Brasileiro de Geografia e Estatística	Brazil	—	1
World Health Organization	Switzerland	SCImago #172	1
Iran University of Medical Sciences	Iran	SCImago #2614 · THE 601–800	1
University of Ioannina	Greece	SCImago #3673 · THE 1201–1500 · QS 1001-1200	1
Instituto do Coração Edson Saad da Universidade Federal do Rio de Janeiro (UFRJ)	Brasil	—	1
Shahid Sadoughi University of Medical Sciences	Iran	SCImago #7597 · THE 1501+	1
University of Exeter	United Kingdom	SCImago #679 · THE =170 · QS =155	1
Rigshospitalet	Denmark	—	1
Universidade Federal do Rio Grande do Sul	Brasil	SCImago #1267 · THE 601–800 · QS =691	1
Universidade de São Paulo	Brazil	SCImago #99 · THE 201–250 · QS 108	1
Mayo Clinic	United States	SCImago #88	1
Federal University of Rio Grande do Sul	Brazil	SCImago #1267 · THE 601–800 · QS =691	1

Geographic distribution of citing authors

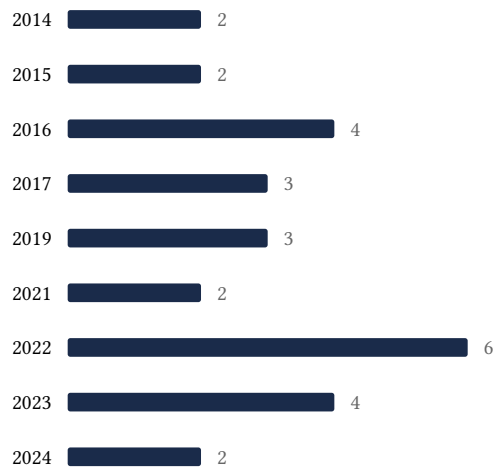
Country	Citing papers
Brazil	5
United States	5
Australia	3
United Kingdom	3

Country	Citing papers
Iran	2
Brasil	2
China	2
Greece	1
India	1
Netherlands	1
Perú	1
Republic of Yemen	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	Low-grade systemic inflammation and the development of type 2 diabetes: the atherosclerosis risk in communities study	7	8 CFR 204.5(h)(3)(v) – Criterion 5
Contribution 2	Atherosclerosis Risk in Communities Study. Low-grade systemic inflammation and the development of type 2 diabetes: the atherosclerosis risk in communities study	6	8 CFR 204.5(h)(3)(v) – Criterion 5
Contribution 3	Leptin and incident type 2 diabetes: risk or protection?	7	8 CFR 204.5(h)(3)(v) – Criterion 5