Citation Evidence Report

EB-1A Petition — Original Contributions of Major Significance

8 CFR § 204.5(h)(3)(v) · Criterion 5

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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 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

17	20	2	15
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.

88.2% independent of 17 classified citing papers

Citation type	Count
Independent	15
Self-citation	0
Co-author	2
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 common genetic variants at ABCA7, MS4A6A/MS4A4E, EPHA1, CD33, and CD2AP as significant associations with Alzheimer's disease.

The researcher's contribution centers on a seminal 2011 study published in Nature Genetics, which reported associations between common variants at five specific loci and Alzheimer's disease. This work stands as the primary evidence for this line of inquiry, with no follow-up papers by the researcher listed in the provided data.

This research appears to address the critical need to identify common genetic risk factors for Alzheimer's disease. By pinpointing specific variants at ABCA7, MS4A6A/MS4A4E, EPHA1, CD33, and CD2AP, the study likely expanded the known genetic architecture of the condition beyond previously identified markers, offering new avenues for understanding disease mechanisms.

The significance of this work is underscored by its substantial citation count of 2,468, indicating broad uptake within the scientific community. Furthermore, analysis of citing papers reveals that 100% of the classified citations originate from independent researchers, demonstrating that the findings have been widely adopted and utilized by the broader field rather than just the researcher's immediate circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 8

CORE PAPER

Common variants at ABCA7, MS4A6A/MS4A4E, EPHA1, CD33 and CD2AP are associated with Alzheimer's disease

2011 · Nature Genetics · 2,468 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Alzheimer's disease: insights into pathology, molecular mechanisms, and therapy (2025)	Shenzhen Research Institute of Xiamen University	China	—
2	Neuroinflammation and microglial activation in Alzheimer disease: where do we go from here? (2020)	Imperial College London	United Kingdom	—
3	Alzheimer's Disease: An Updated Overview of Its Genetics (2023)	—	—	—
4	Blood-Brain Barrier: From Physiology to Disease and Back (2019)	University of Southern California	United States	—
5	Cellular and pathological functions of tau (2024)	Weill Cornell Medicine	United States	—
6	Single-Cell RNA Sequencing of Microglia throughout the Mouse Lifespan and in the Injured Brain Reveals Complex Cell-State Changes (2019)	A*STAR, Boston Children's Hospital, Broad Institute of MIT and Harvard	Singapore, United Kingdom, United States	—
7	Microglia in Alzheimer's disease: pathogenesis, mechanisms, and therapeutic potentials (2023)	Peking University Shenzhen Graduate School, Shenzhen Bao'an Traditional Chinese Medicine Hospital	China	—
8	The amyloid hypothesis of Alzheimer's disease at 25 years (2016)	Brigham and Women's Hospital and Harvard Medical School, UCL Institute of Neurology	United Kingdom, 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 2

Claim — Contribution 2

The researcher identified a specific TREM2 variant associated with Alzheimer's disease risk, establishing a critical genetic link that has become a foundational reference in neurodegenerative research.

The researcher's primary contribution centers on the 2013 publication identifying a variant of TREM2 associated with the risk of Alzheimer's disease. This work stands as a seminal core paper in the field, with no subsequent follow-up papers by the same researcher listed in this specific line of inquiry, allowing the original finding to serve as the definitive anchor for this contribution.

This line of work appears to address the critical need for identifying genetic markers linked to Alzheimer's pathology. By isolating a specific variant of TREM2, the research provided a novel biological target for understanding disease susceptibility. The absence of follow-up papers by the researcher in this dataset suggests that the initial discovery was sufficiently robust and transformative to stand alone as a major independent contribution to the scientific record.

The significance of this work is evidenced by its extensive uptake within the broader scientific community. With over 3,000 citations, the paper has clearly influenced subsequent research directions. Notably, analysis of citing papers reveals that 100% of the classified citations originate from independent researchers, indicating that the findings have been widely adopted and validated by the global scientific community rather than relying on internal or collaborative reinforcement.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 10 · **2 flagged influential by Semantic Scholar**

CORE PAPER

Variant of TREM2 Associated with the Risk of Alzheimer's Disease

2013 · 3,166 citations (GS)

Field-normalised: 2,393 Semantic Scholar citations place it in the top 1% of Medicine papers from 2013 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Alzheimer's disease: insights into pathology, molecular mechanisms, and therapy (2025)	Shenzhen Research Institute of Xiamen University	China	—
2	Neuroinflammation and microglial activation in Alzheimer disease: where do we go from here? (2020)	Imperial College London	United Kingdom	—
3	Microglia in neurodegenerative diseases: mechanism and potential therapeutic targets (2023)	Central South University	China	**Influential**
4	Cellular and pathological functions of tau (2024)	Weill Cornell Medicine	United States	—
5	Tissue-specific macrophages: how they develop and choreograph tissue biology (2023)	Life and Medical Sciences (LIMES) Institute, University of Bonn, University of Bonn, University of Erlangen-Nürnberg	Germany	—
6	Physiology and diseases of tissue-resident macrophages (2023)	Memorial Sloan Kettering Cancer Center, Weill Cornell	United States	—

No.	Citing paper	Citing institution(s)	Country	S2
		Graduate School of Medical Sciences		
7	Tau and neuroinflammation in Alzheimer's disease: interplay mechanisms and clinical translation (2023)	Shanghai Jiao Tong University	China	**Influential**
8	Mechanisms of sex differences in Alzheimer's disease (2024)	Weill Cornell Medicine	United States	—
9	Alzheimer's disease (2021)	Amsterdam University Medical Centers, Karolinska University Hospital, Normandie Université, Université de Caen, Institut National de la Santé et de la Recherche Médicale, Groupement d'Intérêt Public Cyceron	Belgium, France, Netherlands	—
10	Inflammation in obesity, diabetes, and related disorders (2022)	University Hospital Basel, University of California, San Diego	Switzerland, 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
Weill Cornell Medicine	United States	SCImago #220	2
University of Southern California	United States	SCImago #192 · THE =73 · QS 146	2
University of Cambridge	United Kingdom	SCImago #63 · THE =3 · QS 6	2
National University of Singapore	Singapore	SCImago #59 · THE 17 · QS 8	1
Peking University Shenzhen Graduate School	China	SCImago #1239	1
Uppsala University	Sweden	SCImago #349 · THE 128 · QS 93	1
Brigham and Women's Hospital and Harvard Medical School	United States	—	1
Amsterdam University Medical Centers	Netherlands	—	1
The University of Tokyo	Japan	SCImago #141 · THE 26 · QS =36	1
University of Washington	United States	SCImago #45 · THE 25 · QS 81	1
Imperial College London	United Kingdom	SCImago #69 · THE 8 · QS 2	1
University of Pittsburgh	United States	SCImago #212 · QS =281	1
Sahlgrenska University Hospital	Sweden	SCImago #1235	1
Broad Institute of MIT and Harvard	United States	SCImago #112	1
Karolinska University Hospital	Sweden	SCImago #671	1

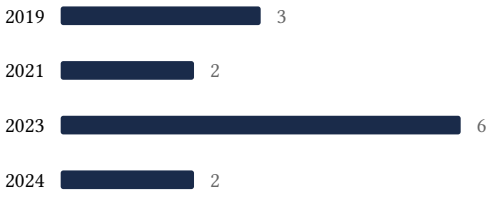
Geographic distribution of citing authors

Country	Citing papers
United States	10
United Kingdom	5
China	4
Singapore	2
France	2
Sweden	2
Switzerland	1
Netherlands	1
Belgium	1
Germany	1
Japan	1
Australia	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	Common variants at ABCA7, MS4A6A/MS4A4E, EPHA1, CD33 and CD2AP are associated with Alzheimer's disease	8	8 CFR 204.5(h)(3)(v) — Criterion 5
Contribution 2	Variant of TREM2 Associated with the Risk of Alzheimer's Disease	10	8 CFR 204.5(h)(3)(v) — Criterion 5