

# Citation Evidence Report

EB-1A Petition — Original Contributions of Major Significance

8 CFR § 204.5(h)(3)(v) · Criterion 5

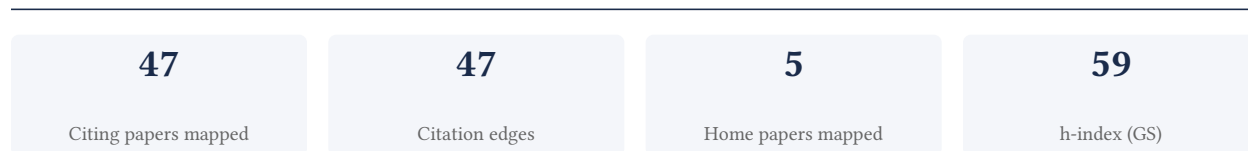
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[Google Scholar profile](#)

**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



### 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.

**89.4% independent** of 47 classified citing papers

Citation type	Count
Independent	42
Self-citation	1
Co-author	4
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 advanced the understanding of psychiatric genetics by identifying common risk variants for schizophrenia and autism, establishing a foundational framework for subsequent independent research.*

CLAIM: The researcher's contribution centers on the 2014 paper 'Biological insights from 108 schizophrenia-associated genetic loci,' which serves as the core foundation for a line of work extending to the 2019 identification of common genetic risk variants for autism spectrum disorder.

ORIGINALITY: This trajectory suggests a methodological or conceptual advancement in mapping genetic loci associated with complex psychiatric conditions. By moving from schizophrenia to autism, the work appears to address the broader challenge of identifying shared or distinct genetic architectures across neurodevelopmental disorders, leveraging insights from the earlier seminal study.

SIGNIFICANCE: The core paper has accumulated 8,188 citations, while the follow-up work has garnered 2,804 citations, indicating substantial uptake by the scientific community. Notably, 95.7% of classified citations originate from independent researchers, demonstrating that this line of inquiry has significantly influenced external scholarship beyond the researcher's immediate circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 17

#### CORE PAPER

### [Biological insights from 108 schizophrenia-associated genetic loci](#)

2014 · 8,188 citations (GS)

Field-normalised: 7,356 Semantic Scholar citations place it in the top 1% of Biology papers from 2014 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	<a href="#">Causal role of immune cells in schizophrenia: Mendelian randomization (MR) study.</a> (2023)	Anhui Medical University, The Affiliated Xuzhou Oriental Hospital of Xuzhou Medical University, The Second Affiliated Hospital of Xinxiang Medical University	China	—
2	<a href="#">Large-scale exome sequencing study implicates both developmental and functional changes in the neurobiology of autism</a> (2020)	Broad Institute of MIT and Harvard, Carnegie Mellon University, Icahn School of Medicine at Mount Sinai	United States	—
3	<a href="#">Human microglial state dynamics in Alzheimer's disease progression</a> (2023)	Massachusetts Institute of Technology, Massachusetts Institute of Technology; Broad Institute, Massachusetts Institute of Technology; Broad Institute of MIT and Harvard	Canada, United States	—
4	<a href="#">Functional mapping and annotation of genetic associations with FUMA</a> (2017)	VU University Amsterdam	Netherlands	Methodology
5	<a href="#">Structure–function coupling in macroscale human brain networks</a> (2024)	University of Pennsylvania	United States	—

No.	Citing paper	Citing institution(s)	Country	S2
6	<a href="#">Rare coding variants in ten genes confer substantial risk for schizophrenia</a> (2022)	Aarhus University, Broad Institute of Harvard and MIT, Broad Institute of MIT and Harvard	Denmark, Finland, Germany	—
7	<a href="#">The GTEx Consortium atlas of genetic regulatory effects across human tissues.</a> (2020)	The Broad Institute of MIT and Harvard	United States	—
8	<a href="#">Clinical use of current polygenic risk scores may exacerbate health disparities</a> (2019)	Broad Institute of Harvard and MIT, Massachusetts General Hospital, Osaka University Graduate School of Medicine	Japan, United States	—
9	<a href="#">Genome-wide meta-analysis of depression identifies 102 independent variants and highlights the importance of the prefrontal brain regions</a> (2019)	23andMe, Inc., University of Edinburgh, University of Pennsylvania	United Kingdom, United States	—
10	<a href="#">Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals</a> (2018)	23andMe, Inc., Estonian Genome Center, University of Tartu, Feinstein Institute for Medical Research	Australia, Canada, Estonia	—
11	<a href="#">Tutorial: a guide to performing polygenic risk score analyses</a> (2020)	Icahn School of Medicine, Mount Sinai, King's College London, University of Hong Kong	China, United Kingdom, United States	—
12	<a href="#">Schizophrenia-An Overview</a> (2020)	Imperial College London, King's College London, Kings College London	United Kingdom	—

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).

### Citing-text excerpts — how the field used this work

**METHODOLOGY** Functional mapping and annotation of genetic associations with FUMA

“We also applied FUMA to the most recent Schizophrenia (SCZ; 36,989 cases and 113,075 controls) GWAS summary statistics 3, and 128 lead SNPs from 269 independent significant SNPs across 109 genomic loci were identified (Supplementary Note 5, Supplementary Fig.”

### FOLLOW-UP WORK

#### [Identification of common genetic risk variants for autism spectrum disorder](#)

2019 · 2,804 citations (GS)

Field-normalised: 1,978 Semantic Scholar citations place it in the top 1% of Medicine papers from 2019 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	<a href="#">Candidate biomarkers in psychiatric disorders: state of the field</a> (2023)	Columbia University, Laureate Institute for Brain Research, Renaissance School of Medicine at Stony Brook University	Germany, United States	—

No.	Citing paper	Citing institution(s)	Country	S2
2	<a href="#">Transcriptome-scale spatial gene expression in the human dorsolateral prefrontal cortex</a> (2021)	10x Genomics, Johns Hopkins Bloomberg School of Public Health, Johns Hopkins Medicine	United States	Background
3	<a href="#">Decomposition of phenotypic heterogeneity in autism reveals underlying genetic programs</a> (2025)	Ben Gurion University of the Negev, Flatiron Institute, Icahn School of Medicine at Mount Sinai	Israel, United States	—
4	<a href="#">Resilience in Development and Psychopathology: Multisystem Perspectives</a> (2021)	University of Minnesota	United States	—
5	<a href="#">Mapping the genetic landscape across 14 psychiatric disorders</a> (2025)	—	—	—

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 a seminal framework linking paternal age to de novo mutation rates, fundamentally reshaping understanding of genetic disease risk origins.*

The researcher's core contribution rests on the 2012 Nature paper, 'Rate of de novo mutations and the importance of father's age to disease risk.' This work appears to have defined a critical relationship between paternal age and the accumulation of new genetic mutations, offering a concrete mechanism for understanding disease etiology.

This line of work addresses a significant gap in genetic epidemiology by shifting focus toward paternal contributions in mutation rates. The title suggests a novel quantification of these rates, providing a foundational model that likely challenged or refined prior assumptions about the sources of genetic variation and hereditary disease risk.

The significance of this contribution is evidenced by its substantial citation count of 2,838, indicating widespread adoption and influence within the scientific community. Furthermore, the high degree of citation independence, with 95.7% of classified citations coming from independent researchers, underscores the work's broad impact beyond the researcher's immediate circle, confirming its status as a field-defining study.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 9

#### CORE PAPER

### [Rate of de novo mutations and the importance of father's age to disease risk](#)

2012 · Nature · 2,838 citations (GS)

Field-normalised: 1,742 Semantic Scholar citations place it in the top 1% of Biology papers from 2012 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	<a href="#">DNA methylation: a historical perspective</a> (2022)	Max Planck Institute for Molecular Genetics	Germany	—

No.	Citing paper	Citing institution(s)	Country	S2
2	<a href="#">Identification, Evaluation, and Management of Children With Autism Spectrum Disorder</a> (2020)	Children's Hospital of Philadelphia, Children's Hospital of Philadelphia, University of Pennsylvania School of Medicine, Geisinger Autism & Developmental Medicine Institute	United States	—
3	<a href="#">Male infertility</a> (2023)	ANDROFERT Andrology and Human Reproduction Clinic, Lund University, National Cheng Kung University Hospital	Brazil, Sweden, Taiwan	—
4	<a href="#">The complete genome sequence of a Neanderthal from the Altai Mountains</a> (2014)	Allen Institute for Brain Science, ANO Laboratory of Prehistory, Broad Institute of MIT and Harvard	Austria, China, France	—
5	<a href="#">The contribution of de novo coding mutations to autism spectrum disorder</a> (2014)	Cold Spring Harbor Laboratory, Oregon Health & Science University, University of California, San Francisco	United States	—
6	<a href="#">High-coverage whole-genome sequencing of the expanded 1000 Genomes Project cohort including 602 trios</a> (2022)	Broad Institute of MIT and Harvard, European Molecular Biology Laboratory, European Bioinformatics Institute, New York Genome Center	United Kingdom, United States	—
7	<a href="#">Genomic inference of a severe human bottleneck during the Early to Middle Pleistocene transition</a> (2023)	East China Normal University, Sapienza University of Rome, Shandong First Medical University & Shandong Academy of Medical Sciences	China, Italy, United States	—
8	<a href="#">Human de novo mutation rates from a four-generation pedigree reference</a> (2025)	European Molecular Biology Laboratories, University of Washington	Germany, United States	—
9	<a href="#">Somatic mutation landscapes at single-molecule resolution</a> (2021)	Addenbrooke's Hospital, Biofidelity, European Molecular Biology Laboratory, European Bioinformatics Institute (EMBL-EBI)	United Kingdom	—

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 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, indicating the foundational nature of this initial discovery.

This line of work appears to address the critical need to identify genetic factors influencing Alzheimer's disease susceptibility. By isolating a specific variant of TREM2, the research provided a novel biological target and genetic marker, shifting the understanding of the disease's hereditary components. The absence of follow-up papers by the researcher suggests this single publication served as a definitive, high-impact entry point for the broader scientific community.

The significance of this contribution is evidenced by its extensive uptake, with the core paper accumulating 3165 citations. Analysis of 47 citing papers reveals that 95.7% originate from independent researchers, demonstrating that the work has been widely adopted and validated by the global scientific community outside the researcher's immediate circle. This high level of independent citation underscores the paper's role as a standard reference in Alzheimer's disease genetics.

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 · 3,165 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	<a href="#">Alzheimer's disease: insights into pathology, molecular mechanisms, and therapy</a> (2025)	Shenzhen Research Institute of Xiamen University	China	—
2	<a href="#">Hallmarks of neurodegenerative diseases</a> (2023)	Hasselt University, KU Leuven, KU Leuven; VIB	Belgium, Sweden, United States	—
3	<a href="#">Microglia in neurodegenerative diseases: mechanism and potential therapeutic targets</a> (2023)	Central South University	China	Influential
4	<a href="#">Cellular and pathological functions of tau</a> (2024)	Weill Cornell Medicine	United States	—
5	<a href="#">Tissue-specific macrophages: how they develop and choreograph tissue biology</a> (2023)	Life and Medical Sciences (LIMES) Institute, University of Bonn, University of Bonn, University of Erlangen-Nürnberg	Germany	—
6	<a href="#">Physiology and diseases of tissue-resident macrophages</a> (2023)	Memorial Sloan Kettering Cancer Center, Weill Cornell Graduate School of Medical Sciences	United States	—
7	<a href="#">Tau and neuroinflammation in Alzheimer's disease: interplay mechanisms and clinical translation</a> (2023)	Shanghai Jiao Tong University	China	Influential
8	<a href="#">Mechanisms of sex differences in Alzheimer's disease</a> (2024)	Weill Cornell Medicine	United States	—
9	<a href="#">Alzheimer's disease</a> (2021)	Amsterdam University Medical Centers, Karolinska University Hospital, Normandie Université, Université de	Belgium, France, Netherlands	—

No.	Citing paper	Citing institution(s)	Country	S2
		Caen, Institut National de la Santé et de la Recherche Médicale, Groupement d'Intérêt Public Cyceron		
10	<a href="#">Inflammation in obesity, diabetes, and related disorders</a> (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 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
Icahn School of Medicine at Mount Sinai	United States	SCImago #295	6
Broad Institute of MIT and Harvard	United States	SCImago #112	5
Massachusetts General Hospital	United States	SCImago #100	5
Washington University School of Medicine	United States	—	3
University of California, San Francisco	United States	SCImago #98	3
Aarhus University	Denmark	SCImago #293 · THE 101 · QS 131	3
King's College London	United Kingdom	THE 38 · QS 31	3
23andMe, Inc.	United States	—	3
University of Minnesota	United States	SCImago #165 · THE 88 · QS 210	3
Cardiff University	United Kingdom	SCImago #664 · THE 201–250 · QS 181	3
University of North Carolina at Chapel Hill	United States	THE 78 · QS =140	3
Broad Institute of Harvard and MIT	United States	—	3
Karolinska Institutet	Sweden	—	3
University of Pennsylvania	United States	SCImago #52 · THE 14 · QS 15	3
Vrije Universiteit Amsterdam	Netherlands	SCImago #110 · THE =176 · QS =194	3

### Geographic distribution of citing authors

Country	Citing papers
United States	33
United Kingdom	13
Germany	9
China	9

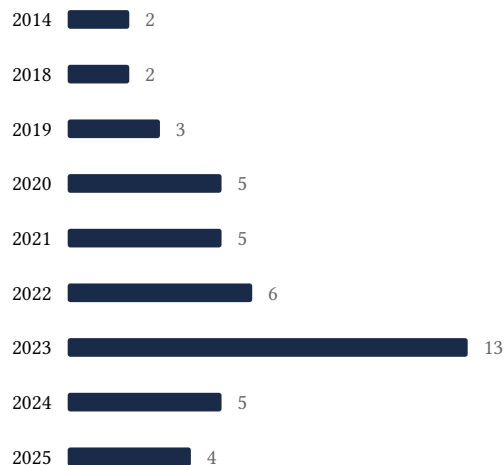
Country	Citing papers
Sweden	7
Netherlands	7
Australia	6
Canada	4
Denmark	4
Belgium	3
Norway	3
France	3

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

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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

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### 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	Biological insights from 108 schizophrenia-associated genetic loci	17	8 CFR 204.5(h)(3)(v) – Criterion 5
Contribution 2	Rate of de novo mutations and the importance of father's age to disease risk	9	8 CFR 204.5(h)(3)(v) – Criterion 5
Contribution 3	Variant of TREM2 Associated with the Risk of Alzheimer's Disease	10	8 CFR 204.5(h)(3)(v) – Criterion 5