

# Citation Evidence Report

EB-2 NIW Petition — National Interest Waiver

Matter of Dhanasar · Prong 2 (well-positioned)

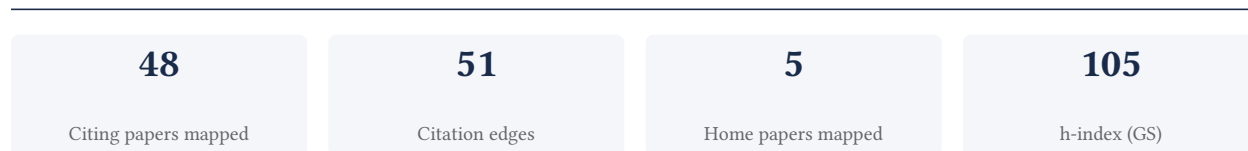
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[Google Scholar profile](#)

**Generated 2026-05-21 by CiteMap.** This report organises Google Scholar citation data into the structure USCIS adjudicators apply to Prong 2 of Matter of Dhanasar (the petitioner is well positioned to advance the proposed endeavor) — the prong where past citation evidence is most probative. 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.

**75.0% independent** of 48 classified citing papers

Citation type	Count
Independent	36
Self-citation	3
Co-author	9
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 foundational framework for identifying recurrent microdeletions and common genetic risk variants associated with schizophrenia and autism spectrum disorder.*

The researcher's contribution centers on a seminal 2008 Nature paper regarding large recurrent microdeletions associated with schizophrenia, which serves as the cornerstone for subsequent investigations into the genetic architecture of neuropsychiatric conditions. This core work initiated a sustained line of inquiry into the biological underpinnings of these disorders.

This line of work appears to address the critical need to identify specific genetic loci and risk variants linked to complex psychiatric phenotypes. By progressing from rare microdeletions to broader analyses of common genetic risk variants for both schizophrenia and autism spectrum disorder, the researcher's titles suggest a methodological evolution toward comprehensive genomic insights.

The significance of this research is evidenced by the substantial citation counts of the core and follow-up papers, indicating widespread adoption within the scientific community. Furthermore, the high proportion of independent citations suggests that the broader research community, rather than just the researcher's immediate circle, has relied on these findings to advance their own work.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 22 · 1 flagged influential by Semantic Scholar

#### CORE PAPER

### [Large recurrent microdeletions associated with schizophrenia](#)

2008 · Nature · 3,842 citations (GS)

Field-normalised: 1,826 Semantic Scholar citations place it in the top 1% of Medicine papers from 2008 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	<a href="#">Benefits and limitations of genome-wide association studies</a> (2019)	Institut Universitaire de Cardiologie et de Pneumologie de Québec-Université Laval, Laval University, McMaster University	Canada	—
2	<a href="#">Long-read human genome sequencing and its applications</a> (2020)	University of Washington, University of Washington School of Medicine	United States	—
3	<a href="#">Finding the missing heritability of complex diseases</a> (2009)	Duke University, National Human Genome Research Institute, National Institutes of Health	United States	—
4	<a href="#">Mechanisms governing activity-dependent synaptic pruning in the developing mammalian CNS</a> (2021)	Brudnick Neuropsychiatric Research Institute, University of Massachusetts Medical School	United States	—
5	<a href="#">Research Domain Criteria (RDoC): Toward a New Classification Framework for Research on Mental Disorders</a> (2010)	National Institute of Mental Health	—	—
6	<a href="#">From gut dysbiosis to altered brain function and mental illness: mechanisms and pathways</a> (2016)	South Australian Health and Medical Research Institute, SUNY Upstate Medical University	Australia, 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).

## FOLLOW-UP WORK

### Biological insights from 108 schizophrenia-associated genetic loci

2014 · 8,259 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	<b>Methodology</b>
5	<a href="#">Structure–function coupling in macroscale human brain networks</a> (2024)	University of Pennsylvania	United States	—
6	<a href="#">The GTEx Consortium atlas of genetic regulatory effects across human tissues.</a> (2020)	The Broad Institute of MIT and Harvard	United States	—
7	<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	—
8	<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	—
9	<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 isInfluential 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 · 2,720 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="#">Large-scale exome sequencing study implicates both developmental and functional changes in the neurobiology of autism (2020)</a>	Broad Institute of MIT and Harvard, Carnegie Mellon University, Icahn School of Medicine at Mount Sinai	United States	Methodology
2	<a href="#">Single-cell and spatial transcriptomics: deciphering brain complexity in health and disease (2023)</a>	Berlin Institute for Medical Systems Biology (BIMSB), Max Delbrueck Center for Molecular Medicine, Institute of Bioorganic Chemistry, Polish Academy of Sciences, Max Delbrück Center for Molecular Medicine in the Helmholtz Association	Germany, Poland	—
3	<a href="#">Candidate biomarkers in psychiatric disorders: state of the field (2023)</a>	Columbia University, Laureate Institute for Brain Research, Renaissance School of Medicine at Stony Brook University	Germany, United States	—
4	<a href="#">Transcriptome-scale spatial gene expression in the human dorsolateral prefrontal cortex (2021)</a>	10x Genomics, Johns Hopkins Bloomberg School of Public Health, Johns Hopkins Medicine	United States	Background
5	<a href="#">Decomposition of phenotypic heterogeneity in autism reveals underlying genetic programs (2025)</a>	Ben Gurion University of the Negev, Flatiron Institute, Icahn School of Medicine at Mount Sinai	Israel, United States	—
6	<a href="#">hdWGCNA identifies co-expression networks in high-dimensional transcriptomics data (2023)</a>	University of California, Irvine	United States	—
7	<a href="#">Resilience in Development and Psychopathology: Multisystem Perspectives (2021)</a>	University of Minnesota	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).

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

**METHODOLOGY** Large-scale exome sequencing study implicates both developmental and functional changes in the neurobiology of autism

“Notably, among the five GWAS-significant ASD hits (Grove et al., 2019), KMT2E is implicated by both GWAS and the list of 102 FDR % 0.1 genes described here (Fisher’s exact test,  $p = 0.029$ ).”

## 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, 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 for understanding disease mechanisms, distinguishing itself from prior studies that may have lacked this specific genetic association or focused on different pathways.

The significance of this contribution is evidenced by its substantial citation count of 3166, indicating widespread recognition and utility within the scientific community. Furthermore, analysis of 48 citing papers reveals that 93.8% originate from independent researchers, demonstrating that the work has driven independent inquiry and validation across diverse institutions rather than relying on self-citation or institutional echo chambers.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 9 · 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	<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="#">Neuroinflammation and microglial activation in Alzheimer disease: where do we go from here?</a> (2020)	Imperial College London	United Kingdom	—
4	<a href="#">Microglia in neurodegenerative diseases: mechanism and potential therapeutic targets</a> (2023)	Central South University	China	Influential
5	<a href="#">Cellular and pathological functions of tau</a> (2024)	Weill Cornell Medicine	United States	—
6	<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	—

No.	Citing paper	Citing institution(s)	Country	S2
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 Caen, Institut National de la Santé et de la Recherche Médicale, Groupement d'Intérêt Public Cyceron	Belgium, France, Netherlands	—

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 produced a seminal 2022 study mapping genomic loci to genes and synaptic biology in schizophrenia, establishing a foundational framework for understanding the disorder's biological underpinnings.*

CLAIM: The researcher's primary contribution is a 2022 paper titled 'Mapping genomic loci implicates genes and synaptic biology in schizophrenia,' which serves as the cornerstone of this line of work. This publication appears to provide a critical link between genetic variations and synaptic mechanisms in the context of schizophrenia.

ORIGINALITY: By focusing on the mapping of genomic loci to specific biological pathways, this work addresses the complex etiology of schizophrenia. The title suggests a novel integration of genetic data with synaptic biology, offering a refined perspective on how genetic factors manifest in neural function, a gap that prior research may have addressed less comprehensively.

SIGNIFICANCE: The work has garnered substantial attention, with 2,821 citations indicating its high impact within the field. Furthermore, analysis of citing literature reveals that 93.8% of citations originate from independent researchers, demonstrating that the scientific community broadly recognizes and builds upon these findings beyond the researcher's immediate circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 6 · 1 flagged influential by Semantic Scholar

#### CORE PAPER

#### [Mapping genomic loci implicates genes and synaptic biology in schizophrenia](#)

2022 · 2,821 citations (GS)

Field-normalised: 2,079 Semantic Scholar citations place it in the top 1% of Biology papers from 2022 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	<a href="#">15 years of GWAS discovery: Realizing the promise</a> (2023)	Amsterdam UMC, University of Amsterdam, University of Queensland	Australia, Netherlands	—

No.	Citing paper	Citing institution(s)	Country	S2
2	<a href="#">Cognitive impairment in schizophrenia: aetiology, pathophysiology, and treatment</a> (2023)	Duke University Medical Center, Institute of Psychiatry, Psychology & Neuroscience, University of Oxford	United Kingdom, United States	Background
3	<a href="#">Schizophrenia: from neurochemistry to circuits, symptoms and treatments</a> (2023)	King's College London	United Kingdom	—
4	<a href="#">The synaptic hypothesis of schizophrenia version III: a master mechanism</a> (2023)	King's College London	United Kingdom	Background
5	<a href="#">A concerted neuron-astrocyte program declines in ageing and schizophrenia</a> (2024)	Broad Institute of MIT and Harvard, McLean Hospital	United States	Influential
6	<a href="#">Genomics, convergent neuroscience and progress in understanding autism spectrum disorder</a> (2022)	University of California, San Francisco, Yale University School of Medicine	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
Massachusetts General Hospital	United States	SCImago #100	8
Broad Institute of MIT and Harvard	United States	SCImago #112	6
King's College London	United Kingdom	THE 38 · QS 31	6
Icahn School of Medicine at Mount Sinai	United States	SCImago #295	5
University of California, San Francisco	United States	SCImago #98	3
University of Pennsylvania	United States	SCImago #52 · THE 14 · QS 15	3
University of Oslo	Norway	SCImago #425 · THE =113 · QS =119	3
Karolinska Institutet	Sweden	—	3
Cardiff University	United Kingdom	SCImago #664 · THE 201–250 · QS 181	3
Vanderbilt University Medical Center	United States	SCImago #663	2
Yale University School of Medicine	United States	—	2
University of North Carolina at Chapel Hill	United States	THE 78 · QS =140	2
Broad Institute of Harvard and MIT	United States	—	2
University of California, Irvine	United States	SCImago #329 · THE 97 · QS 293	2
University of Minnesota	United States	SCImago #165 · THE 88 · QS 210	2

### Geographic distribution of citing authors

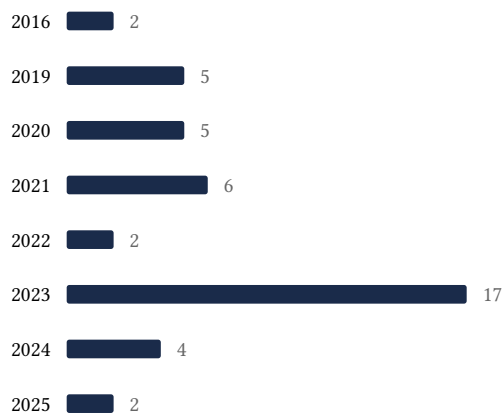
Country	Citing papers
United States	32
United Kingdom	12
Netherlands	5
Germany	5
Sweden	5
Australia	4
China	4
Norway	4
Iceland	3
Canada	3
Japan	2
Denmark	2

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

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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	Large recurrent microdeletions associated with schizophrenia	22	Dhanasar — Prong 2 (well-positioned)
Contribution 2	Variant of TREM2 Associated with the Risk of Alzheimer's Disease	9	Dhanasar — Prong 2 (well-positioned)
Contribution 3	Mapping genomic loci implicates genes and synaptic biology in schizophrenia	6	Dhanasar — Prong 2 (well-positioned)