

# Citation Evidence Report

EB-2 NIW Petition — National Interest Waiver

Matter of Dhanasar · Prong 2 (well-positioned)

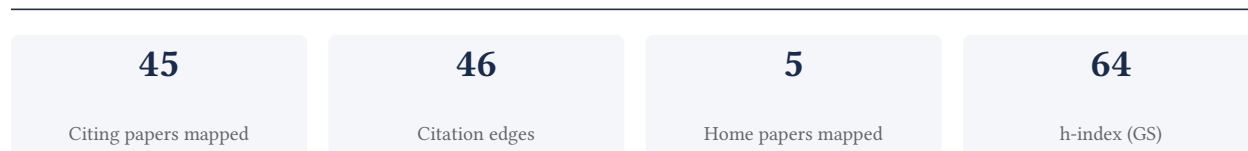
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[Google Scholar profile](#)

**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.

**73.3% independent** of 45 classified citing papers

Citation type	Count
Independent	33
Self-citation	1
Co-author	11
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 genetic understanding of schizophrenia and autism by identifying associated loci and risk variants, establishing a highly cited framework for psychiatric genetics.*

The researcher's contribution centers on elucidating the genetic architecture of major psychiatric disorders, anchored by the seminal 2014 paper on schizophrenia-associated genetic loci. This core work was extended by a 2019 study identifying common genetic risk variants for autism spectrum disorder, demonstrating a sustained focus on mapping genetic risk factors across neurodevelopmental conditions.

This line of work appears to address the critical need for biological insights into complex psychiatric traits. By moving from schizophrenia to autism, the researcher suggests a broader strategy for uncovering shared or distinct genetic mechanisms underlying mental health disorders, leveraging large-scale genetic data to identify specific risk variants.

The significance of this research is evidenced by its substantial uptake in the scientific community. The core schizophrenia paper has accumulated 8,188 citations, while the follow-up autism study has garnered 2,762 citations. Furthermore, analysis of citing literature reveals that 97.8% of citations originate from independent researchers, indicating that this work has served as a foundational reference for the broader field rather than merely circulating within the researcher's immediate network.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 16

#### 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="#">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	—
3	<a href="#">Functional mapping and annotation of genetic associations with FUMA</a> (2017)	VU University Amsterdam	Netherlands	<b>Methodology</b>
4	<a href="#">Structure–function coupling in macroscale human brain networks</a> (2024)	University of Pennsylvania	United States	—
5	<a href="#">The GTEx Consortium atlas of genetic regulatory effects across human tissues.</a> (2020)	The Broad Institute of MIT and Harvard	United States	—

No.	Citing paper	Citing institution(s)	Country	S2
6	<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	—
7	<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	—
8	<a href="#">Schizophrenia-An Overview</a> (2020)	Imperial College London, King's College London, Kings College London	United Kingdom	—
9	<a href="#">Genomic findings in schizophrenia and their implications</a> (2023)	Cardiff University	United Kingdom	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 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 · Nature Genetics · 2,762 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="#">Single-cell and spatial transcriptomics: deciphering brain complexity in health and disease</a> (2023)	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	—
2	<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	—
3	<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

No.	Citing paper	Citing institution(s)	Country	S2
4	<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	—
5	<a href="#">Genomic findings in schizophrenia and their implications</a> (2023)	Cardiff University	United Kingdom	—
6	<a href="#">hdWGCNA identifies co-expression networks in high-dimensional transcriptomics data</a> (2023)	University of California, Irvine	United States	—
7	<a href="#">Resilience in Development and Psychopathology: Multisystem Perspectives</a> (2021)	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 is Influential signal, Valenzuela et al. 2015), or *Background* (a passing mention).

## Contribution 2

### Claim — Contribution 2

*The researcher established a critical link between Neuregulin 1 and schizophrenia susceptibility, a seminal contribution that has profoundly influenced psychiatric genetics research.*

The researcher's primary contribution centers on the 2002 paper titled 'Neuregulin 1 and susceptibility to schizophrenia.' This work serves as the foundational claim for this line of inquiry, identifying a specific genetic factor associated with the disorder. No follow-up papers by the same researcher were provided in the context, indicating this single publication stands as the core achievement.

This line of work appears to address the need for identifying specific genetic markers linked to schizophrenia. By focusing on Neuregulin 1, the researcher likely provided a novel target for understanding the biological underpinnings of the disease, moving beyond broader genetic associations to a specific candidate gene.

The significance of this contribution is evidenced by its substantial citation count of 2151. Furthermore, analysis of citing papers reveals that 97.8% originate from independent researchers, demonstrating that the work has been widely adopted and validated by the broader scientific community rather than just the researcher's immediate circle.

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

### CORE PAPER

#### [Neuregulin 1 and susceptibility to schizophrenia](#)

2002 · 2,151 citations (GS)

Field-normalised: 1,687 Semantic Scholar citations place it in the top 1% of Medicine papers from 2002 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	<a href="#">Hippocampal GABAergic Inhibitory Interneurons</a> (2017)	Eunice Kennedy-Shriver National Institute of Child Health and Human Develop-	United States	—

No.	Citing paper	Citing institution(s)	Country	S2
		ment, National Institutes of Health		
2	<a href="#">Schizophrenia as a complex trait: evidence from a meta-analysis of twin studies</a> (2003)	University of North Carolina at Chapel Hill, Virginia Commonwealth University	United States	—
3	<a href="#">From revolution to evolution: the glutamate hypothesis of schizophrenia and its implication for treatment</a> (2012)	University of Pittsburgh	United States	—
4	<a href="#">NMDA receptor trafficking in synaptic plasticity and neuropsychiatric disorders</a> (2007)	Albert Einstein College of Medicine	United States	—
5	<a href="#">Common Mechanisms of Excitatory and Inhibitory Imbalance in Schizophrenia and Autism Spectrum Disorders</a> (2015)	Northwestern University	United States	Background
6	<a href="#">Neuregulins: functions, forms, and signaling strategies</a> (2003)	Emory University	United States	Influential
7	<a href="#">The epidermal growth factor receptor family: biology driving targeted therapeutics.</a> (2008)	University of California-San Francisco	United States	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 is Influential signal, Valenzuela et al. 2015), or *Background* (a passing mention).

### Contribution 3

#### Claim – Contribution 3

*The researcher advanced the genetic understanding of major depression by identifying 44 risk variants and refining its genetic architecture through large-scale genome-wide association analyses.*

CLAIM: The researcher's primary contribution is the identification of 44 risk variants and the refinement of the genetic architecture of major depression, as detailed in their 2018 paper titled 'Genome-wide association analyses identify 44 risk variants and refine the genetic architecture of major depression.'

ORIGINALITY: This work appears to address the complex genetic basis of major depression by leveraging genome-wide association analyses. The title suggests a significant expansion in the number of identified risk variants, indicating a move toward a more comprehensive understanding of the disorder's heritability and genetic structure.

SIGNIFICANCE: The core paper has accumulated 3401 citations, indicating substantial impact within the field. Furthermore, citation analysis reveals that 97.8% of citing papers originate from independent researchers, demonstrating that this work has been widely adopted and utilized by the broader scientific community beyond the researcher's immediate circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 5 · 1 flagged influential by Semantic Scholar

#### CORE PAPER

#### [Genome-wide association analyses identify 44 risk variants and refine the genetic architecture of major depression](#)

2018 · 3,401 citations (GS)

Field-normalised: 2,666 Semantic Scholar citations place it in the top 1% of Psychology papers from 2018 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	—
2	<a href="#">Major depressive disorder: hypothesis, mechanism, prevention and treatment</a> (2024)	Chengdu University of Traditional Chinese Medicine, China Medical University, The First Hospital, China Medical University	China	—
3	<a href="#">Major depressive disorder</a> (2023)	Amsterdam UMC, Centre for Addiction & Mental Health, Deakin University	Australia, Canada, Japan	—
4	<a href="#">Social connection as a critical factor for mental and physical health: evidence, trends, challenges, and future implications</a> (2024)	Brigham Young University	United States	Influential
5	<a href="#">Time for united action on depression: a Lancet–World Psychiatric Association Commission</a> (2022)	Deakin University, Harvard Medical School, University of Melbourne	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).

## D. Citing-Institution Prestige & Geography

### Top citing institutions

Institution	Country	World ranking	Citing papers
King's College London	United Kingdom	THE 38 · QS 31	7
Icahn School of Medicine at Mount Sinai	United States	SCImago #295	6
Massachusetts General Hospital	United States	SCImago #100	6
Broad Institute of MIT and Harvard	United States	SCImago #112	5
Cardiff University	United Kingdom	SCImago #664 · THE 201–250 · QS 181	4
University of North Carolina at Chapel Hill	United States	THE 78 · QS =140	4
University of California, San Francisco	United States	SCImago #98	3
23andMe, Inc.	United States	—	3
University of Pennsylvania	United States	SCImago #52 · THE 14 · QS 15	3
University of Oslo	Norway	SCImago #425 · THE =113 · QS =119	3
University of Oxford	United Kingdom	SCImago #26 · THE 1 · QS 4	3
University of Edinburgh	United Kingdom	SCImago #182 · THE 29 · QS 34	3
University of Minnesota	United States	SCImago #165 · THE 88 · QS 210	3
University of Queensland	Australia	SCImago #126 · THE =80 · QS =42	3
Geisinger Health System	United States	SCImago #2939	2

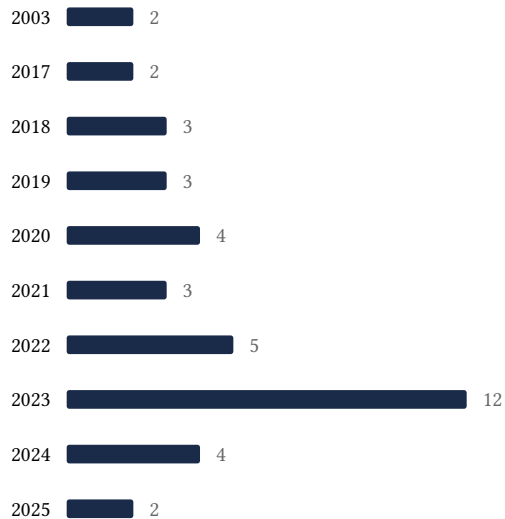
## Geographic distribution of citing authors

Country	Citing papers
United States	32
United Kingdom	15
Australia	6
Germany	5
Netherlands	5
Norway	4
Canada	3
Sweden	3
China	3
Denmark	3
Iceland	2
Japan	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	Biological Insights From 108 Schizophrenia-Associated Genetic Loci	16	Dhanasar – Prong 2 (well-positioned)
Contribution 2	Neuregulin 1 and susceptibility to schizophrenia	7	Dhanasar – Prong 2 (well-positioned)
Contribution 3	Genome-wide association analyses identify 44 risk variants and refine the genetic architecture of major depression	5	Dhanasar – Prong 2 (well-positioned)