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

8	8	1	19
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 8 classified citing papers

Citation type	Count
Independent	8
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 that mTOR inhibition induces autophagy to reduce polyglutamine toxicity in Huntington disease models, a seminal finding published in Nature Genetics.

The researcher's primary contribution is the demonstration that inhibiting mTOR induces autophagy, thereby reducing the toxicity of polyglutamine expansions in fly and mouse models of Huntington disease. This work, published in Nature Genetics in 2004, serves as the foundational core of this research line, with no subsequent follow-up papers by the same researcher provided in this context.

This line of work appears to address the critical need for therapeutic strategies targeting protein aggregation in neurodegenerative disorders. By linking mTOR inhibition to autophagy and reduced toxicity in Huntington disease models, the research suggests a novel mechanistic pathway for intervention, distinguishing itself through its focus on cellular clearance mechanisms in genetic disease models.

The significance of this contribution is underscored by its substantial citation count of 2955, indicating widespread recognition and utility within the scientific community. Furthermore, analysis of citing papers reveals that 100% of the classified citations originate from independent researchers, highlighting the work's broad impact beyond the researcher's immediate institutional or collaborative network.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 8 · **1 flagged influential by Semantic Scholar**

CORE PAPER

Inhibition of mTOR induces autophagy and reduces toxicity of polyglutamine expansions in fly and mouse models of Huntington disease

2004 · Nature Genetics · 2,955 citations (GS)

Field-normalised: 2,450 Semantic Scholar citations place it in the top 1% of Medicine papers from 2004 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	The mechanisms and roles of selective autophagy in mammals (2022)	Nagasaki University, National Institutes of Health, Osaka University	Japan, United Kingdom, United States	—
2	Autophagy in Human Health and Disease (2013)	Brigham and Women's Hospital, University of Texas Southwestern Medical Center	United States	—
3	Autophagy and Neurodegeneration: Pathogenic Mechanisms and Therapeutic Opportunities (2017)	Cambridge Institute for Medical Research, University Medical Center Göttingen	Germany, United Kingdom	Influential
4	Molecular mechanisms of cell death in neurological diseases (2021)	The University of Melbourne, The Walter and Eliza Hall Institute	Australia	Background
5	Neuropathogenesis-on-chips for neurodegenerative diseases (2024)	Brigham and Women's Hospital, Chungnam National University Hospital, Eulji University	South Korea, United States	Background
6	The lysosome as a cellular centre for signalling, metabolism and quality control (2019)	University of California at Berkeley	United States	—

No.	Citing paper	Citing institution(s)	Country	S2
7	Autophagy fights disease through cellular self-digestion (2008)	Baylor College of Medicine and Texas Children Hospital, New York University Grossman School of Medicine, Tokyo Medical and Dental University	Japan, United States	—
8	Huntington's Disease: Complex Pathogenesis and Therapeutic Strategies (2024)	Jinan University	China	—

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
Brigham and Women's Hospital	United States	SCImago #130	2
Institute for Basic Science	South Korea	SCImago #1451	1
Tokyo Medical and Dental University	Japan	QS =697	1
University Medical Center Göttingen	Germany	—	1
Osaka University	Japan	SCImago #546 · QS 91	1
Sungkyunkwan University	South Korea	SCImago #527 · THE 87 · QS =126	1
University of California at Berkeley	United States	—	1
University of Texas Southwestern Medical Center	United States	SCImago #562	1
New York University Grossman School of Medicine	United States	—	1
National Institutes of Health	United States	SCImago #44	1
University of Michigan	United States	SCImago #43 · THE 23 · QS 45	1
University College London	United Kingdom	SCImago #30	1
Nagasaki University	Japan	SCImago #3522 · THE 1201–1500 · QS 901-950	1
The University of Melbourne	Australia	SCImago #72 · THE 37 · QS 19	1
Cambridge Institute for Medical Research	United Kingdom	—	1

Geographic distribution of citing authors

Country	Citing papers
United States	5
Japan	2
United Kingdom	2
Australia	1

Country	Citing papers
South Korea	1
China	1
Germany	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	Inhibition of mTOR induces autophagy and reduces toxicity of polyglutamine expansions in fly and mouse models of Huntington disease	8	Dhanasar — Prong 2 (well-positioned)