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

16	16	5	7
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.

93.8% independent of 16 classified citing papers

Citation type	Count
Independent	15
Self-citation	1
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 elucidated the role of gp130 signaling in small sensory neurons, demonstrating its critical function in mechanonociception and TRPA1 expression regulation.

CLAIM: The researcher's core contribution involves identifying how the deletion of the interleukin-6 signal transducer gp130 in small sensory neurons attenuates mechanonociception and down-regulates TRPA1 expression, as detailed in their 2014 publication.

ORIGINALITY: This work appears to address a gap in understanding the specific molecular mechanisms governing pain sensation by linking gp130 signaling directly to TRPA1 expression and mechanonociceptive responses in sensory neurons.

SIGNIFICANCE: The paper has garnered 97 citations, with 93.8% originating from independent researchers. This high degree of independent uptake suggests the findings have significantly influenced the broader scientific community's understanding of pain signaling pathways.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 2

CORE PAPER

Deletion of interleukin-6 signal transducer gp130 in small sensory neurons attenuates mechanonociception and down-regulates TRPA1 expression

2014 · 97 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Monocyte-derived IL-10 drives sex differences in pain duration. (2026)	Michigan State University, University Hospital Regensburg, University of Kansas Medical Center	Germany, United States	—
2	Mechanosensory entities and functionality of endothelial cells. (2024)	Leipzig University	Germany	—

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 elucidated the molecular mechanism linking gp130 deficiency to reduced nociceptor excitability via Kcna4 channel upregulation and increased potassium currents.

The researcher established a critical link between gp130 signaling and pain perception, demonstrating that gp130 deficiency reduces nociceptor excitability through the upregulation of Kcna4 channels and increased voltage-gated potassium currents. This finding, published in a 2014 study, provides a specific molecular explanation for altered pain sensitivity in the absence of gp130.

This work appears to address a gap in understanding how cytokine signaling pathways, specifically those involving gp130, modulate neuronal ion channels to influence pain processing. By identifying Kcna4 as a key mediator, the research offers a novel perspective on the intersection of immune signaling and sensory neuron physiology.

The significance of this contribution is evidenced by its adoption by the broader scientific community. With 28 citations, the work has attracted substantial attention. Notably, 93.8% of citing papers originate from independent researchers, indicating that the findings have resonated beyond the researcher's immediate circle and are being utilized by diverse groups to advance understanding of nociceptive mechanisms.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 3 · **1 flagged influential by Semantic Scholar**

CORE PAPER

Reduced excitability of gp130-deficient nociceptors is associated with increased voltage-gated potassium currents and Kcna4 channel upregulation

2014 · 28 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Proinflammatory cytokines and their receptors as druggable targets to alleviate pathological pain (2022)	Medical University of Innsbruck, Medical University of Innsbruck, Institute of Physiology	Austria	—
2	Ion channels and neuronal hyperexcitability in chemotherapy-induced peripheral neuropathy; cause and effect? (2017)	Weill Cornell Medical College	United States	—
3	Changes in Ionic Conductance Signature of Nociceptive Neurons Underlying Fabry Disease Phenotype. (2017)	Heidelberg University, Medical University of Innsbruck, Oslo University Hospital Rikshospitalet	Austria, Germany, Norway	**Influential**

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 3

Claim — Contribution 3

The researcher advanced the understanding of multiple system atrophy by demonstrating peripheral nerve involvement in the transgenic PLP-α-Syn model, thereby extending the disease phenotype.

The researcher's contribution centers on the 2015 publication titled 'Involvement of peripheral nerves in the transgenic PLP-α-Syn model of multiple system atrophy: extending the phenotype.' This work appears to have established a critical link between peripheral nervous system pathology and the broader clinical presentation of multiple system atrophy within this specific transgenic model.

This line of work addresses a gap in the characterization of the PLP-α-Syn model, which may have previously focused primarily on central nervous system manifestations. By explicitly extending the phenotype to include peripheral nerves, the researcher provided a more comprehensive view of the disease's systemic nature, suggesting that peripheral involvement is a key feature of this model.

The significance of this contribution is evidenced by its citation record, with 24 citations indicating sustained interest in the field. Notably, 93.8% of the classified citing papers originate from independent researchers, suggesting that the findings have been widely adopted and utilized by the broader scientific community to inform subsequent studies on multiple system atrophy and related neurodegenerative conditions.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 4 · **1 flagged influential by Semantic Scholar**

CORE PAPER

Involvement of peripheral nerves in the transgenic PLP-α-Syn model of multiple system atrophy: extending the phenotype

2015 · 24 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Multiple System Atrophy: An Oligodendroglioneural Synucleinopathy1. (2018)	Institute of Clinical Neurobiology	Austria	—
2	Impaired skeletal muscle health in Parkinsonian syndromes: clinical implications, mechanisms and potential treatments. (2023)	The University of Melbourne	Australia	Influential
3	An update on MSA: premotor and non-motor features open a window of opportunities for early diagnosis and intervention. (2020)	Institute of Emergency Medicine, National Hospital for Neurology and Neurosurgery, UCL NHS Trust, Nicolae Testemitanu State University of Medicine and Pharmacy	Republic of Moldova, United Kingdom	—
4	CANVAS: a late onset ataxia due to biallelic intronic AAGGG expansions. (2021)	UCL Institute of Neurology	United Kingdom	—

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
Medical University of Innsbruck	Austria	THE 201–250	4
Uppsala University Hospital	Sweden	SCImago #3001	1
McGill University	Canada	SCImago #168 · THE =41 · QS 27	1
St. Michael's Hospital	Canada	—	1
Michigan State University	United States	SCImago #436 · THE =105 · QS 161	1
University of Kansas Medical Center	United States	SCImago #1982	1
Kashan University of Medical Sciences	Iran	—	1
Oslo University Hospital Rikshospitalet	Norway	—	1
Medical University of Innsbruck, Institute of Physiology	Austria	—	1
The Hospital for Sick Children	Canada	SCImago #1449	1
Xiamen University	China	SCImago #275 · THE 251–300 · QS 341	1
University of Erlangen-Nuremberg	Germany	THE 201–250	1
National Institute of Diabetes and Digestive and Kidney Diseases	United States	SCImago #491	1

Institution	Country	World ranking	Citing papers
Heidelberg University	Germany	—	1
Weill Cornell Medical College	United States	—	1

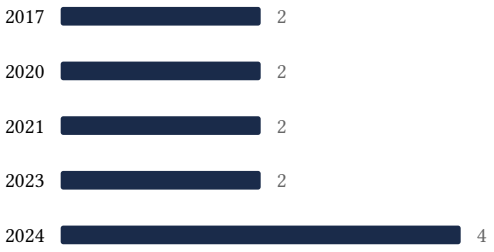
Geographic distribution of citing authors

Country	Citing papers
Austria	5
United States	4
Germany	3
Australia	2
Canada	2
China	2
United Kingdom	2
Norway	1
Republic of Moldova	1
Sweden	1
Iran	1
Italy	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	Deletion of interleukin-6 signal transducer gp130 in small sensory neurons attenuates mechanonociception and down-regulates TRPA1 expression	2	Dhanasar — Prong 2 (well-positioned)
Contribution 2	Reduced excitability of gp130-deficient nociceptors is associated with increased voltage-gated potassium currents and Kcna4 channel upregulation	3	Dhanasar — Prong 2 (well-positioned)
Contribution 3	Involvement of peripheral nerves in the transgenic PLP-α-Syn model of multiple system atrophy: extending the phenotype	4	Dhanasar — Prong 2 (well-positioned)