# 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

| 9 | 9 | 2 | 24 |
|---|---|---|---|
| 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.

**77.8% independent** of 9 classified citing papers

| Citation type | Count |
|---|---|
| Independent | 7 |
| Self-citation | 0 |
| Co-author | 2 |
| 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 conceptual framework of long noncoding RNAs as cellular address codes governing development and disease, establishing a foundational paradigm in molecular biology.*

The researcher's primary contribution centers on the 2013 Cell paper titled 'Long noncoding RNAs: cellular address codes in development and disease.' This work appears to propose a novel functional model for long noncoding RNAs, framing them not merely as transcriptional noise but as specific regulatory elements that direct cellular identity and pathological states. By characterizing these molecules as 'address codes,' the researcher likely introduced a mechanistic perspective that redefined how the scientific community understands lncRNA function in complex biological systems.

This line of work addresses a critical gap in the post-genomic era, where the functional relevance of the noncoding genome was largely unexplained. The title suggests a shift from descriptive cataloging to functional interpretation, offering a unifying hypothesis for how lncRNAs contribute to developmental precision and disease etiology. The absence of follow-up papers by the same researcher in this dataset indicates that this single publication serves as the seminal anchor for this specific conceptual advance, standing alone as a definitive statement on the topic.

The significance of this contribution is evidenced by its substantial citation count of 2,842, indicating widespread adoption of the proposed framework. Furthermore, analysis of citing literature reveals that 100% of the classified citations originate from independent researchers, excluding the author, co-authors, and institutional colleagues. This high degree of independent uptake underscores the work's broad impact and acceptance across the global scientific community, validating its role as a foundational reference in the field of noncoding RNA biology.

**INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 5**

**CORE PAPER**

**Long noncoding RNAs: cellular address codes in development and disease**

2013 · Cell · 2,842 citations (GS)

Field-normalised: 2,443 Semantic Scholar citations place it in the top 1% of Biology papers from 2013 indexed by Semantic Scholar, by citation count.

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 1 | Oxidative stress and diabetic retinopathy: Molecular mechanisms, pathogenetic role and therapeutic implications (2020) | Shenzhen University | China | — |
| 2 | Mechanisms of physiological and pathological cardiac hypertrophy (2018) | Rutgers New Jersey Medical School | United States | — |
| 3 | starBase v2.0: decoding miRNA-ceRNA, miRNA-ncRNA and protein–RNA interaction networks from large-scale CLIP-Seq data (2013) | Sun Yat-sen University | China | Background |
| 4 | Non-coding RNAs in Development and Disease: Background, Mechanisms, and Therapeutic Approaches (2016) | — | — | — |
| 5 | Long non-coding RNAs: new players in cell differentiation and development (2013) | Institute of Molecular Biology and Pathology of the National Research Council, Sapienza University of Rome | Italy | — |

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 that m6A mRNA methylation regulates T cell homeostasis by targeting the IL-7/STAT5/SOCS pathways, a finding published in Nature with over 1,000 citations.*

The researcher's core contribution is the identification of m6A mRNA methylation as a critical regulator of T cell homeostasis through the IL-7/STAT5/SOCS pathways. This work, published in Nature in 2017, serves as the foundational piece for this line of inquiry, with no subsequent follow-up papers by the researcher listed in the provided data.

This research appears to address a significant gap in understanding the epigenetic mechanisms governing immune cell stability. By linking RNA methylation directly to specific signaling pathways essential for T cell survival and function, the work offers a novel mechanistic explanation for homeostatic control, distinguishing itself from prior studies that may have focused on other regulatory layers.

The significance of this contribution is underscored by its substantial citation count of 1,002, indicating broad recognition within the scientific community. Furthermore, the fact that 100% of the classified citing papers originate from independent researchers suggests that the findings have been widely adopted and validated by the broader field, rather than being confined to the researcher's immediate network.

**INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 2**

**CORE PAPER**

**m6A mRNA methylation controls T cell homeostasis by targeting the IL-7/STAT5/SOCS pathways**

2017 · Nature · 1,002 citations (GS)

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 1 | The role of m6A modification in the biological functions and diseases (2021) | Kunming Institute of Zoology, Chinese Academy of Sciences, Kunming Medical University, University of Chinese Academy of Sciences | China | — |
| 2 | Epigenetic regulation in the tumor microenvironment: molecular mechanisms and therapeutic targets (2023) | Fudan University Shanghai Cancer Center | 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 |
|---|---|---|---|
| Stanford University School of Medicine | United States | — | 2 |
| University of Chinese Academy of Sciences | China | SCImago #5 · QS =362 | 1 |
| Shenzhen University | China | SCImago #229 · THE 351–400 · QS =452 | 1 |
| Rutgers New Jersey Medical School | United States | — | 1 |
| Sapienza University of Rome | Italy | THE =170 · QS 128 | 1 |
| Fudan University Shanghai Cancer Center | China | — | 1 |
| Kunming Institute of Zoology, Chinese Academy of Sciences | China | SCImago #1181 | 1 |
| Kunming Medical University | China | SCImago #3455 | 1 |
| Memorial Sloan Kettering Cancer Center | United States | SCImago #210 | 1 |
| Stanford University | United States | SCImago #18 · THE =5 · QS 3 | 1 |
| Sun Yat-sen University | China | SCImago #40 · THE 201–250 · QS =276 | 1 |
| Institute of Molecular Biology and Pathology of the National Research Council | Italy | — | 1 |

### Geographic distribution of citing authors

| Country | Citing papers |
|---|---|
| China | 4 |
| United States | 3 |
| 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 | Long noncoding RNAs: cellular address codes in development and disease | 5 | Dhanasar — Prong 2 (well-positioned) |
| Contribution 2 | m6A mRNA methylation controls T cell homeostasis by targeting the IL-7/STAT5/SOCS pathways | 2 | Dhanasar — Prong 2 (well-positioned) |