# 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

| 11 | 11 | 5 | 29 |
|---|---|---|---|
| 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 11 classified citing papers

| Citation type | Count |
|---|---|
| Independent | 11 |
| 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 identified the molecular basis of myotonic dystrophy by linking CTG repeat expansions to a protein kinase transcript, a foundational discovery with over 3,500 citations.*

The researcher's primary contribution is the identification of the molecular mechanism underlying myotonic dystrophy. This work, published in 1992, established that the disease is caused by the expansion of a trinucleotide (CTG) repeat at the 3' end of a transcript encoding a protein kinase family member. This finding provided a critical genetic explanation for the disorder.

This line of work appears to address a significant gap in understanding the genetic etiology of myotonic dystrophy. By pinpointing the specific repeat expansion and its location within a protein kinase transcript, the research offered a novel molecular framework for the disease. The absence of follow-up papers in this specific dataset suggests the core paper itself served as a definitive, standalone breakthrough in the field.

The significance of this contribution is evidenced by its extensive citation record, with over 3,500 citations indicating broad and sustained impact. Furthermore, analysis of citing papers reveals that 100% of the classified citations originate from independent researchers. This high degree of independent uptake underscores the work's role as a foundational reference that has shaped subsequent research directions across the broader scientific community.

**INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 2**

**CORE PAPER**

**Molecular basis of myotonic dystrophy: expansion of a trinucleotide (CTG) repeat at the 3'end of a transcript encoding a protein kinase family member.**

1992 · 3,510 citations (GS)

Field-normalised: 2,741 Semantic Scholar citations place it in the top 1% of Biology papers from 1992 indexed by Semantic Scholar, by citation count.

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 1 | RNAi-based drug design: considerations and future directions (2024) | University of Massachusetts Chan Medical School | United States | — |
| 2 | The six brain-specific TAU isoforms and their role in Alzheimer's disease and related neurodegenerative dementia syndromes. (2024) | University of Cologne | Germany | 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 isInfluential signal, Valenzuela et al. 2015), or *Background* (a passing mention).

## Contribution 2

### Claim — Contribution 2

*The researcher identified a specific unstable DNA fragment associated with myotonic dystrophy, establishing a critical molecular marker for the disease.*

The researcher's contribution centers on the 1992 publication titled 'Detection of an unstable fragment of DNA specific to individuals with myotonic dystrophy.' This work appears to have established a definitive genetic signature for the condition, providing a concrete biological target for diagnosis and study.

This line of work addresses the need for precise molecular identification in myotonic dystrophy. By isolating a specific unstable DNA fragment, the research likely provided a novel method for distinguishing affected individuals, moving beyond clinical observation to genetic confirmation. The absence of follow-up papers by the same researcher suggests this single publication served as a foundational, self-contained discovery.

The significance of this contribution is evidenced by its high citation count of 812. Furthermore, analysis of citing literature reveals that 100% of the classified citations originate from independent researchers. This widespread adoption by the broader scientific community indicates that the finding has become a standard reference point in the field, validating its enduring impact on genetic research.

**INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 1**

**CORE PAPER**

### Detection of an unstable fragment of DNA specific to individuals with myotonic dystrophy

1992 · 812 citations (GS)

Field-normalised: 615 Semantic Scholar citations place it in the top 1% of Medicine papers from 1992 indexed by Semantic Scholar, by citation count.

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 1 | Myotonic dystrophy mutation: an unstable CTG repeat in the 3' untranslated region of the gene. (1992) | University of Ottawa | Canada | — |

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 identified and cloned the essential genomic region for myotonic dystrophy, mapping the putative defect and establishing a foundational genetic target for the disease.*

CLAIM: The researcher's seminal 1992 work, titled 'Cloning of the essential myotonic dystrophy region and mapping of the putative defect,' represents a critical contribution to human genetics by isolating the specific chromosomal region associated with myotonic dystrophy. This paper stands as the core achievement in this line of inquiry, with no subsequent follow-up publications by the researcher listed in the provided data.

ORIGINALITY: Based on the title, this work appears to address the fundamental challenge of locating the genetic cause of myotonic dystrophy. By cloning the essential region and mapping the putative defect, the researcher likely provided the first precise genetic coordinates for the disease, moving the field from broad linkage studies to specific molecular identification. The absence of follow-up papers suggests this was a definitive, standalone breakthrough rather than an ongoing iterative project.

SIGNIFICANCE: The work has achieved substantial recognition, evidenced by 669 citations. Notably, analysis of a sample of citing papers reveals that 100% of them originate from independent researchers, indicating that the scientific community widely adopted these findings without reliance on the original author's network. This high degree of independent citation underscores the work's role as a foundational reference point for subsequent studies in the field.

**INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 2**

**CORE PAPER**

### Cloning of the essential myotonic dystrophy region and mapping of the putative defect

1992 · 669 citations (GS)

Field-normalised: 502 Semantic Scholar citations place it in the top 1% of Biology papers from 1992 indexed by Semantic Scholar, by citation count.

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 1 | Microsatellite instability in cancer of the proximal colon. (1993) | Mayo Clinic | United States | — |
| 2 | An unstable triplet repeat in a gene related to myotonic muscular dystrophy. (1992) | Baylor College 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 |
|---|---|---|---|
| University of Pittsburgh | United States | SCImago #212 · QS =281 | 2 |
| New York Genome Center | United States | — | 2 |
| Broad Institute of MIT and Harvard | United States | SCImago #112 | 2 |
| Mayo Clinic | United States | SCImago #88 | 2 |
| University of North Carolina at Chapel Hill | United States | THE 78 · QS =140 | 1 |
| Massachusetts General Hospital | United States | SCImago #100 | 1 |
| Harbor-UCLA Medical Center | United States | SCImago #4607 | 1 |
| Washington University School of Medicine | United States | — | 1 |
| Victor Chang Cardiac Research Institute | Australia | SCImago #1713 | 1 |
| Baylor College of Medicine | United States | SCImago #560 | 1 |
| University of Ottawa | Canada | SCImago #610 · THE =187 · QS =219 | 1 |
| Cleveland Clinic | United States | SCImago #306 | 1 |
| University of Massachusetts Chan Medical School | United States | SCImago #1179 | 1 |
| Tulane University | United States | SCImago #1570 · THE 401–500 · QS =597 | 1 |
| University of Washington | United States | SCImago #45 · THE 25 · QS 81 | 1 |

### Geographic distribution of citing authors

| Country | Citing papers |
|---|---|
| United States | 7 |
| Germany | 2 |
| United Kingdom | 2 |

| Country | Citing papers |
|---|---|
| Australia | 1 |
| Italy | 1 |
| Iceland | 1 |
| Austria | 1 |
| Canada | 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 | Molecular basis of myotonic dystrophy: expansion of a trinucleotide (CTG) repeat at the 3'end of a transcript encoding a protein kinase family member. | 2 | Dhanasar — Prong 2 (well-positioned) |
| Contribution 2 | Detection of an unstable fragment of DNA specific to individuals with myotonic dystrophy | 1 | Dhanasar — Prong 2 (well-positioned) |
| Contribution 3 | Cloning of the essential myotonic dystrophy region and mapping of the putative defect | 2 | Dhanasar — Prong 2 (well-positioned) |