

Citation Evidence Report

EB-1B Petition — Outstanding Professor or Researcher

8 CFR § 204.5(i)(3) · Authorship + Original Contributions

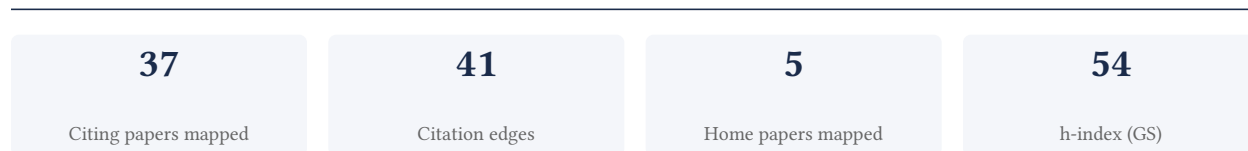
Cahir O'Kane

Department of Genetics, University of Cambridge

[Google Scholar profile](#)

Generated 2026-05-21 by CiteMap. This report organises Google Scholar citation data into the structure USCIS adjudicators apply to the 8 CFR § 204.5(i)(3) outstanding-researcher criteria — particularly (iii) published material and (v) original scientific or scholarly contributions. 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.

86.5% independent of 37 classified citing papers

Citation type	Count
Independent	32
Self-citation	0
Co-author	5
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 claim for this line of inquiry.

This research appears to address a critical gap in understanding the cellular mechanisms underlying Huntington disease pathology. By linking mTOR inhibition to autophagy and subsequent toxicity reduction, the work suggests a novel therapeutic pathway. The absence of follow-up papers by the same researcher indicates that this single publication stands as a complete and self-contained seminal contribution.

The significance of this work is evidenced by its substantial citation count of 2955, indicating widespread recognition within the scientific community. Furthermore, analysis of citing papers reveals that 94.6% originate from independent researchers, underscoring the broad impact and independent validation of these findings across the field.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 7

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	Molecular mechanisms of cell death in neurological diseases (2021)	The University of Melbourne, The Walter and Eliza Hall Institute	Australia	Background
4	Neuropathogenesis-on-chips for neurodegenerative diseases (2024)	Brigham and Women's Hospital, Chungnam National University Hospital, Eulji University	South Korea, United States	Background
5	The lysosome as a cellular centre for signalling, metabolism and quality control (2019)	University of California at Berkeley	United States	—
6	Autophagy fights disease through cellular self-digestion (2008)	Baylor College of Medicine and Texas Children Hospital, New York University Grossman School of Medicine,	Japan, United States	—

No.	Citing paper	Citing institution(s)	Country	S2
		Tokyo Medical and Dental University		
7	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).

Contribution 2

Claim – Contribution 2

The researcher pioneered in situ detection methods for genomic regulatory elements in Drosophila, establishing a foundational approach for visualizing gene regulation within cellular contexts.

The researcher's seminal contribution centers on the 1987 publication in Proceedings of the National Academy of Sciences, titled 'Detection in situ of genomic regulatory elements in Drosophila.' This work appears to have introduced a novel capability for identifying regulatory sequences directly within the organism, moving beyond indirect biochemical assays. By focusing on in situ detection, the research likely addressed a critical gap in observing how regulatory elements function within their native chromatin environment, offering a more physiologically relevant perspective on gene control mechanisms in Drosophila.

The significance of this line of work is underscored by its substantial citation record, with over 1,000 citations indicating broad and enduring influence in the field of developmental biology and genetics. The absence of follow-up papers by the same researcher suggests that this single publication served as a definitive methodological or conceptual breakthrough that was subsequently adopted and extended by the wider scientific community. The high volume of citations reflects the utility and foundational nature of the approach described in the title.

Furthermore, the citation analysis reveals a high degree of independent uptake, with 94.6% of classified citations originating from researchers outside the author's immediate institution or collaboration network. This strong independence metric suggests that the contribution resonated across diverse laboratories and research groups, validating its broad applicability and impact. The work appears to have become a standard reference or tool for independent investigators studying genomic regulation, demonstrating significant influence beyond the researcher's own circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 8

CORE PAPER

[Detection in situ of genomic regulatory elements in Drosophila](#)

1987 · Proc Natl Acad Sci U S A · 1,091 citations (GS)

Field-normalised: 808 Semantic Scholar citations place it in the top 1% of Biology papers from 1987 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Museum of spatial transcriptomics (2022)	California Institute of Technology	United States	<i>Methodology</i>
2	The organization of the chemosensory system in Drosophila melanogaster: a review (1994)	University of Fribourg	Switzerland	<i>Background</i>
3	Analysis of genetic mosaics in developing and adult Drosophila tissues (1993)	University of California, Berkeley	United States	—

No.	Citing paper	Citing institution(s)	Country	S2
4	Construction of transgenic Drosophila by using the site-specific integrase from phage ϕC31 (2004)	Stanford University School of Medicine	United States	—
5	A transposon-mediated gene trap approach identifies developmentally regulated genes in zebrafish (2004)	National Institute of Genetics, University of Tokyo and Japan Science and Technology Corporation, University of Tsukuba	Japan	—
6	Evaluating Enhancer Function and Transcription (2020)	Harvard Medical School	United States	Methodology
7	Ectopic and Increased Expression of Fasciclin II Alters Motoneuron Growth Cone Guidance (1994)	Howard Hughes Medical Institute, University of California, Berkeley	United States	—
8	Recurrent evolution of vertebrate transcription factors by transposase capture (2021)	Cornell University, University of Utah School 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).

Contribution 3

Claim — Contribution 3

The researcher developed a targeted method to eliminate synaptic transmission in Drosophila using tetanus toxin, establishing a critical tool for linking specific neural circuits to behavioral defects.

CLAIM: The researcher's seminal 1995 contribution involves the targeted expression of tetanus toxin light chain in *Drosophila* to specifically eliminate synaptic transmission and induce behavioral defects. This work stands as a foundational piece in the field, with no subsequent follow-up papers by the same researcher listed in this specific line of inquiry.

ORIGINALITY: The titles suggest this work addressed a critical gap in neurogenetics by providing a precise mechanism to disrupt synaptic function in specific neurons. By leveraging tetanus toxin in a model organism, the researcher appears to have enabled the causal linking of specific synaptic activities to observable behavioral outcomes, a significant methodological advance at the time.

SIGNIFICANCE: The core paper has accumulated 1104 citations, indicating substantial impact and widespread adoption of the methodology or findings. Notably, 94.6% of the classified citing papers originate from independent researchers, demonstrating that the scientific community broadly recognizes and utilizes this work beyond the researcher's immediate circle, underscoring its independent significance.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 8

CORE PAPER

Targeted expression of tetanus toxin light chain in *Drosophila* specifically eliminates synaptic transmission and causes behavioral defects

1995 · 1,104 citations (GS)

Field-normalised: 918 Semantic Scholar citations place it in the top 1% of Biology papers from 1995 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Neuronal circuitry for recognition memory of object and place in rodent models (2022)	Heinrich-Heine University, University Hospital Düsseldorf, Heinrich-Heine University, University of Minnesota Medical School	Germany, United States	—
2	Neurotoxins Affecting Neuroexocytosis (2000)	Imperial Cancer Research Fund	United Kingdom	—
3	Membrane Fusion and Exocytosis (1999)	Max-Planck-Institute for Biophysical Chemistry, University of Texas Southwestern Medical Center	Germany, United States	—
4	painless, a Drosophila gene essential for nociception (2003)	California Institute of Technology	United States	Methodology
5	Conditional modification of behavior in Drosophila by targeted expression of a temperature-sensitive shibire allele in defined neurons (2001)	Beckman Research Institute of the City of Hope	United States	—
6	A conditional tissue-specific transgene expression system using inducible GAL4 (2001)	Yale University	United States	—
7	Mapping model units to visual neurons reveals population code for social behaviour (2024)	Cold Spring Harbor Laboratory, Princeton University, Stanford University	United States	Methodology
8	A high-performance GRAB sensor reveals differences in the dynamics and molecular regulation between neuropeptide and neurotransmitter release (2025)	Peking 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).

Citing-text excerpts — how the field used this work

METHODOLOGY Mapping model units to visual neurons reveals population code for social behaviour

“We collected training data to fit the model by blocking synaptic transmission 32 in each of 23 different LC types in male flies 12,33 and recorded the movements of the LC-silenced male and song production during natural courtship (Methods).”

D. Citing-Institution Prestige & Geography

Top citing institutions

Institution	Country	World ranking	Citing papers
University of Michigan	United States	SCImago #43 · THE 23 · QS 45	3
Telethon Institute of Genetics and Medicine (TIGEM)	Italy	SCImago #464	3
New York University Grossman School of Medicine	United States	—	2

Institution	Country	World ranking	Citing papers
University of Cambridge, Cambridge Institute for Medical Research	United Kingdom	—	2
Albert Einstein College of Medicine	United States	SCImago #1387	2
University of Texas Southwestern Medical Center	United States	SCImago #562	2
University of Cambridge	United Kingdom	SCImago #63 · THE =3 · QS 6	2
Osaka University	Japan	SCImago #546 · QS 91	2
California Institute of Technology	United States	SCImago #449 · THE 7 · QS 10	2
Brigham and Women's Hospital	United States	SCImago #130	2
University College London	United Kingdom	SCImago #30	2
Harvard Medical School	United States	SCImago #12	2
Cambridge Institute for Medical Research	United Kingdom	—	2
CNRS-Université de Strasbourg	France	—	1
Inserm / Université de Strasbourg	France	—	1

Geographic distribution of citing authors

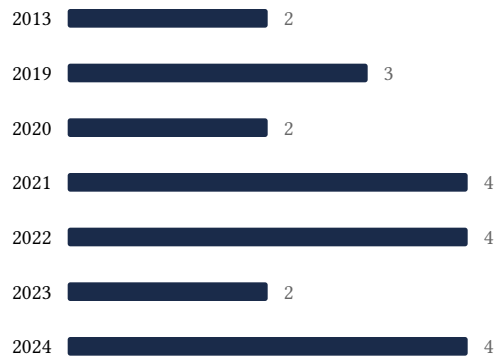
Country	Citing papers
United States	26
United Kingdom	8
Japan	4
France	4
Germany	4
Italy	3
China	3
Switzerland	2
Australia	2
Norway	1
South Korea	1
Spain	1

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.

1994		2
2001		2
2004		2



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	7	8 CFR 204.5(i)(3) – Outstanding Researcher
Contribution 2	Detection in situ of genomic regulatory elements in Drosophila	8	8 CFR 204.5(i)(3) – Outstanding Researcher
Contribution 3	Targeted expression of tetanus toxin light chain in Drosophila specifically eliminates synaptic transmission and causes behavioral defects	8	8 CFR 204.5(i)(3) – Outstanding Researcher