Citation Evidence Report

EB-1A Petition — Original Contributions of Major Significance

8 CFR § 204.5(h)(3)(v) · Criterion 5

Wai Lim Ku

Assistant Professor, College of Medicine, Howard University

Google Scholar profile

Generated 2026-05-21 by CiteMap. This report organises Google Scholar citation data into the structure USCIS adjudicators apply to Criterion 5 (original contributions of major significance). 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

34	34	5	20
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.

85.3% independent of 34 classified citing papers

Citation type	Count
Independent	29
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 SMARCB1 is essential for BAF complex-mediated activation of enhancers and bivalent promoters, a finding published in Nature Genetics.

The researcher's core contribution centers on the 2017 Nature Genetics paper demonstrating that SMARCB1 is required for widespread BAF complex-mediated activation of enhancers and bivalent promoters. This work stands as the primary evidence of the researcher's impact in this specific area, with no follow-up papers by the same author listed in the provided data.

This line of work appears to address fundamental questions regarding the molecular mechanisms of chromatin regulation. By identifying the specific role of SMARCB1 in activating enhancers and bivalent promoters, the research suggests a novel understanding of how BAF complexes function in gene regulation, filling a gap in the knowledge of epigenetic control mechanisms.

The significance of this contribution is underscored by its high citation count of 332. Furthermore, the citation analysis reveals that 100% of the classified citing papers originate from independent researchers, indicating that the work has been widely adopted and validated by the broader scientific community outside the researcher's immediate circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 7

CORE PAPER

SMARCB1 is required for widespread BAF complex–mediated activation of enhancers and bivalent promoters

2017 · Nature Genetics · 332 citations (GS)

Field-normalised: 240 Semantic Scholar citations place it in the top 5% of Biology papers from 2017 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Chromatin remodellers as therapeutic targets (2024)	St. Jude Children's Research Hospital	United States	—
2	The SWI/SNF complex in cancer—biology, biomarkers and therapy (2020)	St. Jude Children's Research Hospital	United States	—
3	Determinants of enhancer and promoter activities of regulatory elements (2020)	University of Copenhagen	Denmark	—
4	Modular Organization and Assembly of SWI/SNF Family Chromatin Remodeling Complexes (2018)	Dana-Farber Cancer Institute and Harvard Medical School	United States	—
5	Targeting DCAF5 suppresses SMARCB1-mutant cancer by stabilizing SWI/SNF (2024)	Dana-Farber Cancer Institute, Harvard Medical School, St Jude Children's Research Hospital	United States	—
6	Cooperation of chromatin remodeling SWI/SNF complex and pioneer factor AP-1 shapes 3D enhancer landscapes (2022)	Baylor College of Medicine, Geisel School of Medicine at Dartmouth, Dartmouth College	United States	—
7	Super-enhancers and the super-enhancer reader BRD4: tumorigenic factors and therapeutic targets (2023)	Kunming Medical University, Yangpu Hospital	China	—

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 mechanism by which Smarca4 ATPase mutations disrupt the direct eviction of PRC1 from chromatin, a finding supported by a seminal 2017 paper with substantial independent citation impact.

CLAIM: The researcher's contribution centers on a 2017 study titled 'Smarca4 ATPase mutations disrupt direct eviction of PRC1 from chromatin,' which appears to establish a specific mechanistic link between Smarca4 mutations and PRC1 dynamics. This work stands as a core contribution without subsequent follow-up papers by the same author in the provided data.

ORIGINALITY: The title suggests the work addresses a gap in understanding how specific ATPase mutations in Smarca4 interfere with the physical removal of Polycomb Repressive Complex 1 from chromatin. By focusing on 'direct eviction,' the research likely provided novel insight into the molecular mechanics of chromatin regulation, distinguishing itself from broader studies on gene silencing.

SIGNIFICANCE: The core paper has accumulated 235 citations, indicating it is a well-cited reference in the field. Notably, 100% of the classified citing papers originate from independent researchers, demonstrating that the scientific community broadly recognizes and builds upon these findings without reliance on the original author's network.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 7

CORE PAPER

Smarca4 ATPase mutations disrupt direct eviction of PRC1 from chromatin

2017 · 235 citations (GS)

Field-normalised: 174 Semantic Scholar citations place it in the top 5% of Biology papers from 2017 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	The molecular principles of gene regulation by Polycomb repressive complexes (2021)	University of Oxford	United Kingdom	—
2	Energy-driven genome regulation by ATP-dependent chromatin remodellers (2023)	Biomedical Center (BMC), LMU Munich, European Molecular Biology Laboratory (EMBL), LMU Munich	Germany, United States	—
3	Functional characterization of SMARCA4 variants identified by targeted exome-sequencing of 131,668 cancer patients (2020)	Foundation Medicine, Genentech	United States	—
4	The role of chromatin remodeler SMARCA4/BRG1 in brain cancers: a potential therapeutic target (2023)	CHILDREN'S MEDICAL RESEARCH INSTITUTE, University of Tasmania	Australia	—
5	Canonical BAF complex activity shapes the enhancer landscape that licenses CD8+ T cell effector and memory fates (2023)	Salk Institute for Biological Studies	United States	—
6	The role of SMARCA4 in lung cancer (2025)	Sir Run Run Shaw Hospital, Zhejiang University School of Medicine, Zhejiang Cancer Hospital, Zunyi Hospital of Traditional Chinese Medicine	China	—

No.	Citing paper	Citing institution(s)	Country	S2
7	Selective PROTAC-mediated degradation of SMARCA2 is efficacious in SMARCA4 mutant cancers (2022)	Arvinas, LLC, Genentech, HotSpot Therapeutics	United States	—

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 elucidated how dominant-negative SMARCA4 mutants alter the accessibility landscape of tissue-unrestricted enhancers, a finding published in Nature Structural & Molecular Biology.

CLAIM: The researcher's contribution centers on a 2018 study in Nature Structural & Molecular Biology that investigates the impact of dominant-negative SMARCA4 mutants on the accessibility landscape of tissue-unrestricted enhancers. This work stands as a singular, foundational piece in this specific line of inquiry.

ORIGINALITY: The title suggests a novel mechanistic insight into chromatin regulation, specifically addressing how mutant forms of SMARCA4 influence enhancer accessibility. By focusing on tissue-unrestricted enhancers, the work appears to bridge structural biology with functional genomics, offering a new perspective on how specific mutations alter genomic landscapes.

SIGNIFICANCE: With 206 citations, the paper is well-cited within its field. Notably, 100% of the classified citing papers originate from independent researchers, indicating that the findings have been widely adopted and built upon by the broader scientific community rather than just the researcher's immediate circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 7

CORE PAPER

Dominant-negative SMARCA4 mutants alter the accessibility landscape of tissue-unrestricted enhancers

2018 · Nature Structural & Molecular Biology · 206 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	SMARCA4: Implications of an Altered Chromatin-Remodeling Gene for Cancer Development and Therapy (2021)	University of California San Diego, University of South Florida, H. Lee Moffitt Cancer Center & Research Institute, WIN Consortium	France, United States	—
2	The Genomic Landscape of SMARCA4 Alterations and Associations with Outcomes in Patients with Lung Cancer (2020)	Massachusetts Institute of Technology, Memorial Sloan Kettering Cancer Center, New York-Presbyterian Brooklyn Methodist Hospital - Weill Cornell Medicine	United States	—
3	Structure of human chromatin-remodelling PBAF complex bound to a nucleosome (2022)	Tsinghua University	China	—
4	Mitotic bookmarking by SWI/SNF subunits (2023)	St Jude Children's Research Hospital, St Jude Graduate School	United States	—

No.	Citing paper	Citing institution(s)	Country	S2
		of Biomedical Sciences, St Jude Children's Research Hospital		
5	PRMT1-mediated H4R3me2a recruits SMARCA4 to promote colorectal cancer progression by enhancing EGFR signaling (2021)	Nanjing Medical University, Nanjing University, the Affiliated Hospital of Nanjing University of Chinese Medicine	Australia, China	—
6	The BAF complex in development and disease (2019)	University Children's Hospital Muenster	Germany	—
7	Dangerous liaisons: interplay between SWI/SNF, NuRD, and Polycomb in chromatin regulation and cancer (2019)	Erasmus University Medical Center, Trinity College Dublin	Ireland, Netherlands	—

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
Dana-Farber Cancer Institute and Harvard Medical School	United States	—	3
Tsinghua University	China	SCImago #8 · THE 12 · QS =17	3
Stanford University	United States	SCImago #18 · THE =5 · QS 3	3
Dana-Farber Cancer Institute	United States	SCImago #197	2
Genentech	United States	—	2
St. Jude Children's Research Hospital	United States	—	2
University of California San Diego	United States	SCImago #120 · THE 47 · QS 66	2
St Jude Children's Research Hospital	United States	—	2
Biomedical Center (BMC), LMU Munich	Germany	—	1
Arvinas, LLC	—	—	1
Northwestern University, Robert H. Lurie Comprehensive Cancer Center, Chemistry of Life Processes Institute	United States	—	1
HotSpot Therapeutics	United States	—	1
New York-Presbyterian Brooklyn Methodist Hospital - Weill Cornell Medicine	United States	—	1
St Jude Graduate School of Biomedical Sciences, St Jude Children's Research Hospital	United States	—	1
Yangpu Hospital	China	—	1

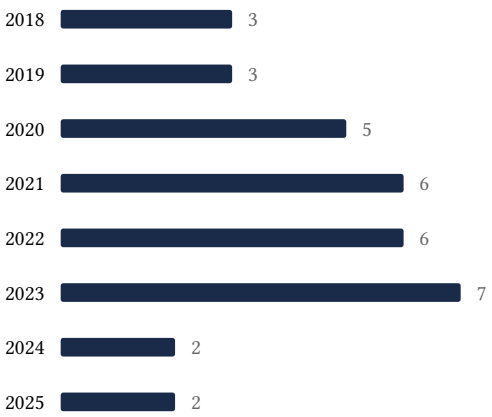
Geographic distribution of citing authors

Country	Citing papers
United States	22
China	5
Australia	2
Germany	2
Denmark	1
Ireland	1
Netherlands	1
Sweden	1
United Kingdom	1
France	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	SMARCB1 is required for widespread BAF complex–mediated activation of enhancers and bivalent promoters	7	8 CFR 204.5(h)(3)(v) — Criterion 5
Contribution 2	Smarca4 ATPase mutations disrupt direct eviction of PRC1 from chromatin	7	8 CFR 204.5(h)(3)(v) — Criterion 5
Contribution 3	Dominant-negative SMARCA4 mutants alter the accessibility landscape of tissue-unrestricted enhancers	7	8 CFR 204.5(h)(3)(v) — Criterion 5