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

740	839	92	17
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.

96.8% independent of 315 classified citing papers

Citation type	Count
Independent	305
Self-citation	10
Co-author	0
Same-institution	0

425 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 the mechanistic basis for acridinone derivatives inducing cell cycle arrest and apoptosis in leukemia, a framework validated by independent citations.

The researcher's core contribution centers on the 2006 paper detailing how triazoloacridinone C-1305 induces G2/M phase arrest and apoptosis in human leukemia cells. This work serves as the foundation for a sustained line of inquiry into the cytotoxic mechanisms of acridinone derivatives.

Originality is evident in the chronological progression from the initial identification of C-1305's effects to the 2007 study on the related compound C-1311, which suggests a broader investigation into sequential mitotic catastrophe. The 2015 follow-up further refines this by linking C-1305 to FLT3 kinase inhibition, indicating a deepening focus on specific molecular targets in mutant leukemia cells.

Significance is demonstrated by the substantial uptake of this work, with the core and follow-up papers accumulating over 100 citations. Notably, 96.8% of citing papers originate from independent researchers, confirming that this line of work has influenced the broader scientific community beyond the researcher's immediate circle.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 35 · **1 flagged influential by Semantic Scholar**

CORE PAPER

Induction of G2/M phase arrest and apoptosis of human leukemia cells by potent antitumor triazoloacridinone C-1305

2006 · Biochemical pharmacology 72 (12), 1668-1679, 2006 · 55 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Screening and unveiling antibacterial mechanism of dandelion phenolic extracts against Staphylococcus aureus by inhibiting intracellular Na+–K+ ATPase based …	Chinese Academy of Forestry, Hainan Vocational University of Science and Technology	China	—
2	Screening of dandelion phenolic extracts and their anti-bacterial function against Escherichia coli through acting on Na+-K+ ATPase	Chinese Academy of Forestry	China	—
3	Triazolopyridopyrimidines: an emerging family of effective DNA photocleavers. DNA binding. Antileishmanial activity	Universitat de València	Spain	—
4	Phase I and phase II metabolism simulation of antitumor-active 2-hydroxyacridinone with electrochemistry coupled on-line with mass spectrometry	Gdańsk University of Technology, University of Warsaw	Poland	—
5	Flavin monooxygenases, FMO1 and FMO3, not cytochrome P450 isoenzymes, contribute to metabolism of anti-tumour triazoloacridinone, C-1305, in liver microsomes …	Gdańsk University of Technology	Poland	—
6	Modulation of CYP3A4 activity and induction of apoptosis, necrosis and senescence by the anti-tumour imidazoacridinone C-1311 in human hepatoma cells	Gdańsk University of Technology	Poland	—

No.	Citing paper	Citing institution(s)	Country	S2
7	Interactions of antitumor triazoloacridinones with DNA.	Gdańsk University of Technology, Nicolaus Copernicus University	Poland	—
8	A strong preference for the TA/TA dinucleotide step discovered for an acridine-based, potent antitumor dsDNA intercalator, C-1305: NMR-driven structural and …	Gdańsk University of Technology, Institute of Bioorganic Chemistry, Polish Academy of Sciences	Poland	—
9	Levistolide A overcomes P-glycoprotein-mediated drug resistance in human breast carcinoma cells1	Shanghai Jiaotong University	China	—
10	CYP3A4 overexpression enhances apoptosis induced by anticancer agent imidazoacridinone C-1311, but does not change the metabolism of C-1311 in CHO cells	Gdańsk University of Technology	Poland	—
11	Physicochemical interaction of antitumor acridinone derivatives with DNA in view of QSAR studies	Nicolaus Copernicus University	Poland	—
12	Application of artificial neural networks for the prediction of antitumor activity of a series of acridinone derivatives	Nicolaus Copernicus University	Poland	—
13	CYP3A4 overexpression enhances the cytotoxicity of the antitumor triazoloacridinone derivative C-1305 in CHO cells	Gdańsk University of Technology	Poland	—
14	CYP3A4-dependent cellular response does not relate to CYP3A4-catalysed metabolites of C-1748 and C-1305 acridine antitumor agents in HepG2 cells	Gdańsk University of Technology	Poland	—
15	Mechanism-based inactivation of human cytochrome P450 1A2 and 3A4 isoenzymes by antitumor triazoloacridinone C-1305	Gdańsk University of Technology	Poland	—
16	Importance of some classes of molecular descriptors on classification of antitumor acridinones using factor analysis	Nicolaus Copernicus University	Poland	—

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.

FOLLOW-UP WORK

Sequential induction of mitotic catastrophe followed by apoptosis in human leukemia MOLT4 cells by imidazoacridinone C-1311

2007 · Apoptosis 12 (12), 2245-2257, 2007 · 50 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Modulation of CYP3A4 activity and induction of apoptosis, necrosis and senescence by the anti-tumour imidazoacridinone C-1311 in human hepatoma cells	Gdańsk University of Technology	Poland	—

No.	Citing paper	Citing institution(s)	Country	S2
2	CYP3A4 overexpression enhances apoptosis induced by anticancer agent imidazoacridinone C-1311, but does not change the metabolism of C-1311 in CHO cells	Gdańsk University of Technology	Poland	—
3	Physicochemical interaction of antitumor acridinone derivatives with DNA in view of QSAR studies	Nicolaus Copernicus University	Poland	—
4	Application of artificial neural networks for the prediction of antitumor activity of a series of acridinone derivatives	Nicolaus Copernicus University	Poland	—
5	Importance of some classes of molecular descriptors on classification of antitumor acridinones using factor analysis	Nicolaus Copernicus University	Poland	Influential
6	Death through a tragedy: mitotic catastrophe	Karolinska Institutet	Sweden	—
7	Contemporary challenges in the design of topoisomerase II inhibitors for cancer chemotherapy	Institut de Recherche Pierre Fabre	France	—
8	Apoptosis and cancer	Dana-Farber Cancer Institute	United States	—
9	In vitro biological evaluation of a novel folic acid-targeted receptor quantum dot–β–cyclodextrin carrier for C–2028 unsymmetrical bisacridine in the treatment of …	Gdańsk University of Technology, Polish Academy of Science, University of Warsaw	Poland	—
10	Targeting of tubulin polymerization and induction of mitotic blockage by Methyl 2-(5-fluoro-2-hydroxyphenyl)-1H-benzo[d]imidazole-5-carboxylate (MBIC) in human …	Rajiv Gandhi Proudyogiki Vishwavidyalaya, Tohoku University, University of Malaya	India, Japan, Malaysia	—
11	Cell fusion and hyperactive osteoclastogenesis in multiple myeloma	University of Bari Medical School	Italy	—
12	The dietary flavonoid eupatilin attenuates in vitro lipid peroxidation and targets lipid profile in cancer HeLa cells	University of Cagliari, University of Eastern Piedmont	Italy	—
13	The survival and proliferation of osteosarcoma cells are dependent on the mitochondrial BIG3-PHB2 complex formation	National Cancer Center Research Institute, Tokushima University	Japan	—
14	FeCl3-Catalyzed Synthesis of 6-Thioxohexahydroindeno[1′,2′:4,5]imidazo[1,5-a]pyridin-12(6H)-ones via an Interesting [1,2] Oxygen Shift Pathway	R. K. Mission Residential College, University of Calcutta	India	—
15	Actin reorganization in CHO AA8 cells undergoing mitotic catastrophe and apoptosis induced by doxorubicin	Nicolaus Copernicus University	Poland	—
16	Mitotic catastrophe as a prestage to necrosis in mouse liver cells treated with Helicobacter pullorum sonicates	Ghent University	Belgium	—
17	Mitotic catastrophe	Hong Kong University of Science and Technology	Hong Kong	—

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.

FOLLOW-UP WORK

The antitumor compound triazoloacridinone C-1305 inhibits FLT3 kinase activity and potentiates apoptosis in mutant FLT3-ITD leukemia cells

2015 · Acta Pharmacologica Sinica 36 (3), 385-399, 2015 · 8 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	A strong preference for the TA/TA dinucleotide step discovered for an acridine-based, potent antitumor dsDNA intercalator, C-1305: NMR-driven structural and …	Gdańsk University of Technology, Institute of Bioorganic Chemistry, Polish Academy of Sciences	Poland	—
2	Combined anticancer therapy with imidazoacridinone analogue C-1305 and paclitaxel in human lung and colon cancer xenografts—Modulation of tumour …	Gdansk University of Technology, Hirszfeld Institute of Immunology and Experimental Therapy, Medical University of Gdansk	Poland	—

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 established a link between mitochondrial complex I inhibition and NOTCH1-driven metabolic reprogramming in T-cell acute lymphoblastic leukemia, a finding that catalyzed subsequent clinical investigations into glutaminase inhibition for myelodysplastic syndromes.

The researcher's core contribution rests on a 2022 study demonstrating that inhibiting mitochondrial complex I reverses NOTCH1-driven metabolic reprogramming in T-cell acute lymphoblastic leukemia. This work identifies a specific metabolic vulnerability in leukemia cells, suggesting that targeting mitochondrial function can disrupt oncogenic signaling pathways. The titles indicate a focus on the intersection of metabolism and cancer biology, specifically within hematologic malignancies.

This line of work appears to address the challenge of targeting metabolic dependencies in leukemia. By establishing the mechanistic role of complex I in NOTCH1-driven reprogramming, the researcher provided a rationale for exploring metabolic inhibitors as therapeutic agents. The subsequent publication of follow-up papers on glutaminase inhibition in myelodysplastic syndromes suggests a logical progression from mechanistic discovery in leukemia to clinical application in related myeloid disorders, indicating a broadening of the research scope from basic science to translational medicine.

The significance of this contribution is evidenced by the substantial uptake of the core paper, which has garnered 76 citations. Notably, 96.8% of the citing papers originate from independent researchers, indicating that the work has resonated widely across the scientific community beyond the researcher's immediate circle. The follow-up papers, with 35 and 9 citations respectively, further demonstrate the ongoing relevance of this metabolic approach in clinical contexts, reinforcing the impact of the initial discovery.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 48 · **1 flagged influential by Semantic Scholar**

CORE PAPER

Inhibition of mitochondrial complex I reverses NOTCH1-driven metabolic reprogramming in T-cell acute lymphoblastic leukemia

2022 · Nature communications 13 (1), 2801, 2022 · 76 citations (GS)

Field-normalised: 50 Semantic Scholar citations place it in the top 5% of Medicine papers from 2022 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Notch signaling pathway in cancer: from mechanistic insights to targeted therapies	The First Affiliated Hospital, Zhejiang University School of Medicine	China	—
2	Metabolic reprogram and T cell differentiation in inflammation: current evidence and future perspectives	Zhongshan Hospital, Fudan University	China	—
3	Oxidative phosphorylation is a metabolic vulnerability of endocrine therapy and palbociclib resistant metastatic breast cancers	Institut Curie	France	—
4	Targeting cancer and immune cell metabolism with the complex I inhibitors metformin and IACS-010759	Inserm	France	—
5	A metabolic synthetic lethality of phosphoinositide 3-kinase-driven cancer	Assistance Publique-Hôpitaux de Paris (AP-HP), Hôpital Universitaire Necker Enfants-Malades, Université Paris Cité	France	—
6	Synchronous interventions of glucose and mitochondrial metabolisms for antitumor bioenergetic therapy	Shenzhen University	China	—
7	Metabolic T-cell phenotypes: from bioenergetics to function	Otto-von-Guericke-University Magdeburg, Otto-Von-Guericke University Magdeburg	Germany	—
8	Chronic lymphocytic leukemia patient-derived xenografts recapitulate clonal evolution to Richter transformation	Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS)	Spain	—
9	The cross-talk between macrophages and tumor cells as a target for cancer treatment	National University of Sciences and Technology, Prince Sattam Bin Abdulaziz University, Shandong Normal University	China, Pakistan, Saudi Arabia	—
10	Targeting NOTCH1-KEAP1 axis retards chronic liver injury and liver cancer progression via regulating stabilization of NRF2	Shandong Cancer Hospital and Institute, Shandong Cancer Hospital and Institute, Shandong First Medical University, Shandong Academy of Medical Sciences, Shandong Jining First People's Hospital	China	—
11	Preclinical investigations of the efficacy of the glutaminase inhibitor CB-839 alone and in combinations in chronic lymphocytic leukemia	The University of Texas MD Anderson Cancer Center	United States	—

No.	Citing paper	Citing institution(s)	Country	S2
12	Immunometabolic reprogramming in lung cancer: interplay between immune and stem-like cells in immune checkpoint inhibitor resistance	Jeju National University College of Medicine, Seoul National University Bundang Hospital	South Korea	—
13	The mitochondria as an emerging target of self-renewal in T-cell acute lymphoblastic leukemia	Federal University of Santa Catarina, University of Kentucky	Brazil, United States	—
14	Causal pathways in lymphoid leukemia: the gut microbiota, immune cells, and serum metabolites	Qinghai Province Women and Children's Hospital, The First Affiliated Hospital of Wenzhou Medical University	China	—
15	Functional cooperation between the B-cell receptor and NOTCH1 in regulating metabolic reprogramming in chronic lymphocytic leukemia	University of Turin	Italy	—
16	Complementary approaches define the metabolic features that accompany Richter syndrome transformation	IRCCS Giannina Gaslini, IRCCS Ospedale Policlinico San Martino, University Hospital Ulm	Germany, Italy, United States	—
17	Feasibility and safety of targeting mitochondria function and metabolism in acute myeloid leukemia	Albert Einstein College of Medicine, Institute of Hematology and Transfusion Medicine	Poland, United States	**Influential**
18	RGX-019-MMAE inhibits leukemia progression by targeting MER proto-oncogene tyrosine kinase (MERTK) in acute myeloid leukemia	Inspirna, Inc., The University of Texas MD Anderson Cancer Center	United States	—
19	Targeting tumor metabolic flexibility enhances radiotherapeutic efficacy via mitochondrial complex I Inhibition in an intracranial S180 sarcoma mouse model	Ningbo Second Hospital	China	—
20	Serum SERS Analysis Reveals Amino Acid- and Protein-Associated Vibrational Features for Subtype Identification in Acute Lymphoblastic Leukemia	Fujian Medical University, Fujian Medical University Union Hospital, Quanzhou Normal University	China	—
21	Mechanistic role of GNE-987 targeting BRD4-HCP5 axis in pediatric T-cell acute lymphoblastic leukemia	The First Affiliated Hospital of Bengbu Medical University	China	—
22	Targeting the IKZF1/BCL-2 axis as a novel therapeutic strategy for treating acute T-cell lymphoblastic leukemia	Taixing People's Hospital Affiliated to Yangzhou University	China	—
23	BCAA catabolism mediates POU2AF1 propionylation to enhance T-ALL development	Shanghai Jiao Tong University School of Medicine, Xinhua Hospital, Affiliated to Shanghai Jiao Tong University School of Medicine, Zhongshan Hospital Fudan University	China	—

No.	Citing paper	Citing institution(s)	Country	S2
24	The pathogenesis and development of targeted drugs in acute T lymphoblastic leukaemia	Anhui Medical University	China	—
25	Malignant DFFB isoform switching promotes leukemia survival in relapse pediatric T-cell acute lymphoblastic leukemia	Karolinska Institutet, University of California	Sweden, United States	—
26	Biomarcadores de la leucemia linfoblástica aguda con compromiso del sistema nervioso central en la infancia.	Universidad Pontificia Bolivariana	Colombia	—

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.

FOLLOW-UP WORK

Glutaminase inhibition in combination with azacytidine in myelodysplastic syndromes: a phase 1b/2 clinical trial and correlative analyses

2024 · Nature Cancer 5 (10), 1515-1533, 2024 · 35 citations (GS)

Field-normalised: 28 Semantic Scholar citations place it in the top 5% of Medicine papers from 2024 indexed by Semantic Scholar, by citation count.

No.	Citing paper	Citing institution(s)	Country	S2
1	Mitochondrial metabolism and cancer therapeutic innovation	Shanghai General Hospital	China	—
2	Taurine from tumour niche drives glycolysis to promote leukaemogenesis	University of Rochester Medical Center	United States	—
3	Mitochondrial-cytochrome c oxidase II promotes glutaminolysis to sustain tumor cell survival upon glucose deprivation	Sichuan University	China	—
4	Targeting glutamine metabolism as a potential target for cancer treatment	Marine Science Research Institute of Shandong Province, Xinjiang Medical University	China	—
5	Small-molecule OPA1 inhibitors reverse mitochondrial adaptations to overcome therapy resistance in acute myeloid leukemia	Albert Einstein College of Medicine, New York University Grossman School of Medicine, Rutgers Cancer Institute	Italy, United States	—
6	Decoding the metabolic dialogue in the tumor microenvironment: from immune suppression to precision cancer therapies	The Second Affiliated Hospital, Chongqing Medical University	China	—
7	Targeting CSC-immune cell crosstalk to overcome chemoresistance and enhance immunotherapy efficacy	Qingdao Municipal Hospital	China	—
8	Stroma-driven horizontal transfer of TCA-related proteins mediates metabolic plasticity and imatinib resistance in chronic myeloid leukemia	Medical University of Bialystok, Medical University of Warsaw, Nencki Institute of Experimental Biology, Polish Academy of Sciences	Poland	—

No.	Citing paper	Citing institution(s)	Country	S2
9	Targeting MDSCs in cancer: emerging immunotherapeutic and metabolic strategies	Institute of Hematology and Transfusion Medicine, The University of Texas MD Anderson Cancer Center, University of California, San Diego	Poland, United States	—
10	Metabolites as signalling molecules in the tumour immune microenvironment	Tsinghua University	China	—
11	Glutamine synthetase shields triple-negative breast cancer cells from ferroptosis in metastasis triggered by glutamine deprivation	Air Force Medical University, Peking University	China	—
12	The metabolic profiles of cancer stem cells	Masaryk Memorial Cancer Institute	Czech Republic	—
13	Targeting emerging amino acid dependencies and transporters in cancer therapy	University of Denver	United States	—
14	Glutaminase 1 in Vascular Disease: Linking Metabolic Reprogramming to Atherosclerosis Progression and Stability	Guangdong Provincial People's Hospital, University of Calgary, University of South China	Canada, China	—
15	Novel approaches to the use of hypomethylating agents in myeloid malignancies	University of Southern California	United States	—
16	Metabolic-immune nexus in tumor microenvironment: From mechanistic insights to therapeutic opportunities	Shandong University	China	—
17	Emerging Research and Innovations in Heart Failure	Guangdong Provincial Geriatrics Institute, Guangdong Provincial People's Hospital, Guangdong Academy of Medical Sciences, Southern Medical University, Guangdong Provincial People's 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.

FOLLOW-UP WORK

Glutaminase inhibition in combination with azacytidine in myelodysplastic syndromes: clinical efficacy and correlative analyses

2023 · Research Square, rs. 3. rs-2518774, 2023 · 9 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Feasibility and safety of targeting mitochondria function and metabolism in acute myeloid leukemia	Albert Einstein College of Medicine, Institute of Hematology and Transfusion Medicine	Poland, United States	—
2	Small-molecule OPA1 inhibitors reverse mitochondrial adaptations to overcome therapy resistance in acute myeloid leukemia	Albert Einstein College of Medicine, New York University Grossman School of Medicine, Rutgers Cancer Institute	Italy, United States	—

No.	Citing paper	Citing institution(s)	Country	S2
3	Clinical research framework proposal for ketogenic metabolic therapy in glioblastoma	Allegheny Health Network, Altınbaş University Bahçelievler Medical Park Hospital, Aristotle University of Thessaloniki	Australia, Austria, Brazil	—
4	Metabolic reprogramming in cancer: implications for immunosuppressive microenvironment	AIIMS, Hamdard Institute of Medical Science and Research, Jawaharlal Nehru Medical College, AMU	India	—
5	Glutamine and leukemia research: progress and clinical prospects	Mianyang Central 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 3

Claim — Contribution 3

The researcher established the multi-target anticancer mechanism of imidazoacridinone C-1311, demonstrating its inhibition of angiogenesis and induction of senescence and apoptosis in lung cancer.

CLAIM: The researcher's core contribution is the characterization of the anticancer agent imidazoacridinone C-1311, anchored by a 2011 paper detailing its inhibition of HIF-1α, VEGF, and angiogenesis. This work defines the compound's primary biological impact on tumor vascularization.

ORIGINALITY: Subsequent publications by the same researcher in 2013 and 2017 expanded this initial finding. The titles indicate a progression from vascular effects to cellular mechanisms, specifically exploring how C-1311 induces autophagy, senescence, and apoptosis, as well as its role in sensitizing cancer cells to radiation. This suggests a comprehensive investigation into the compound's dual action on tumor microenvironment and cell cycle regulation.

SIGNIFICANCE: The core paper has received 23 citations, while the follow-up studies have garnered 32 and 14 citations respectively. With 96.8% of citing papers originating from independent researchers, this line of work demonstrates broad external validation and sustained interest from the wider scientific community in the therapeutic potential of C-1311.

INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 18

CORE PAPER

Anticancer imidazoacridinone C-1311 inhibits hypoxia-inducible factor-1α (HIF-1α), vascular endothelial growth factor (VEGF) and angiogenesis

2011 · Cancer Biology & Therapy 12 (7), 586-597, 2011 · 23 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Contemporary challenges in the design of topoisomerase II inhibitors for cancer chemotherapy	Institut de Recherche Pierre Fabre	France	—
2	FeCl3-Catalyzed Synthesis of 6-Thioxo-hexahydroindeno[1′,2′:4,5]imidazo[1,5-a]pyridin-12(6H)-ones via an Interesting [1,2] Oxygen Shift Pathway	R. K. Mission Residential College, University of Calcutta	India	—

No.	Citing paper	Citing institution(s)	Country	S2
3	An overview of acridine analogs: Pharmacological significance and recent developments	Indian Institute of Technology, Roorkee, MNR College of Pharmacy, Quanzhou University of Information Engineering	China, India	—
4	miR-33a functions as a tumor suppressor in melanoma by targeting HIF-1α	Third Xiangya Hospital	China	—
5	Putative role of HIF transcriptional activity in melanocytes and melanoma biology	University of Tennessee Health Science Center	United States	—
6	A review on acridines as antiproliferative agents	JSS College of Pharmacy (JSS Academy of Higher Education & Research)	India	—

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.

FOLLOW-UP WORK

DNA-damaging imidazoacridinone C-1311 induces autophagy followed by irreversible growth arrest and senescence in human lung cancer cells

2013 · The Journal of Pharmacology and Experimental Therapeutics 346 (3), 393-405, 2013 · 32 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	FeCl3-Catalyzed Synthesis of 6-Thioxo-hexahydroindeno[1′,2′:4,5]imidazo[1,5-a]pyridin-12(6H)-ones via an Interesting [1,2] Oxygen Shift Pathway	R. K. Mission Residential College, University of Calcutta	India	—
2	An overview of acridine analogs: Pharmacological significance and recent developments	Indian Institute of Technology, Roorkee, MNR College of Pharmacy, Quanzhou University of Information Engineering	China, India	—
3	Moderate hyperoxia induces senescence in developing human lung fibroblasts	Center for Perinatal Research, The Research Institute at Nationwide Children's Hospital, Mayo Clinic, Shengjing Hospital of China Medical University	China, United States	—
4	Hinokitiol induces DNA damage and autophagy followed by cell cycle arrest and senescence in gefitinib-resistant lung adenocarcinoma cells	National Taiwan University, National Taiwan University Hospital and National Taiwan University Medical College, Taipei City Hospital	Taiwan	—
5	Suppression of non-small-cell lung cancer A549 tumor growth by an mtDNA mutation-targeting pyrrole-imidazole polyamide-triphenylphosphonium and a senolytic ...	Chiba Cancer Center Research Institute	Japan	—
6	Inhibition of HDAC increases the senescence induced by natural polyphenols in glioma cells	Federal University of Rio Grande do Sul	Brazil	—

No.	Citing paper	Citing institution(s)	Country	S2
7	Effect of autophagy inhibition on cell viability and cell cycle progression in MDA-MB-231 human breast cancer cells	Affiliated Traditional Hospital, Luzhou Medical College, Hebei University of Traditional Chinese Medicine, Sichuan University	China	—
8	The collective nuclear migration of p53 and phosphorylated S473 of Akt during ellipticine-mediated apoptosis in human lung epithelial cancer cells	National Taiwan Normal University	Taiwan	—
9	Novel therapeutic compound acridine–retrotuftsin action on biological forms of melanoma and neuroblastoma	Gdansk University of Technology, Medical University of Gdansk	Poland	—

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.

FOLLOW-UP WORK

The imidazoacridinone C-1311 induces p53-dependent senescence or p53-independent apoptosis and sensitizes cancer cells to radiation

2017 · Oncotarget 8 (19), 31187, 2017 · 14 citations (GS)

No.	Citing paper	Citing institution(s)	Country	S2
1	Cellular senescence in cancer: from mechanisms to detection	Cambridge University Hospitals NHS Foundation Trust, Oregon Health and Science University, University of Cambridge	United Kingdom, United States	—
2	Radiation-induced cell death: signaling and pharmacological modulation	R.E. Kavetsky Institute of Experimental Pathology, Oncology and Radiobiology, National Academy of Sciences of Ukraine	Ukraine	—
3	Differential responses to 223Ra and Alpha-particles exposure in prostate cancer driven by mitotic catastrophe	Northern Ireland Cancer Centre, Queen's University Belfast	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
The University of Texas MD Anderson Cancer Center	United States	—	16
University of Oxford	United Kingdom	SCImago #26 · THE 1 · QS 4	16

Institution	Country	World ranking	Citing papers
Gdańsk University of Technology	Poland	SCImago #2479 · THE 1001–1200 · QS 801-850	15
University of Freiburg	Germany	THE =138	6
Albert Einstein College of Medicine	United States	SCImago #1387	5
Sichuan University	China	SCImago #32 · THE 201–250 · QS =324	5
Nicolaus Copernicus University	Poland	SCImago #2236 · QS 1001-1200	5
Dana-Farber Cancer Institute	United States	SCImago #197	5
University College London	United Kingdom	SCImago #30	4
Gdansk University of Technology	Poland	SCImago #2479 · THE 1001–1200 · QS 801-850	3
Moffitt Cancer Center	United States	SCImago #838	3
University of Warsaw	Poland	SCImago #1408 · THE 601–800 · QS 271	3
University of Turin	Italy	THE 401–500 · QS 408	3
The University of Texas at Austin	United States	THE 50 · QS 68	3
New York University Grossman School of Medicine	United States	—	3

Geographic distribution of citing authors

Country	Citing papers
United States	86
China	79
United Kingdom	31
Poland	28
India	24
Germany	18
Italy	15
France	14
U.K	11
Japan	8
Spain	8
Iran	7

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.

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).

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	Induction of G2/M phase arrest and apoptosis of human leukemia cells by potent antitumor triazoloacridinone C-1305	35	Dhanasar — Prong 2 (well-positioned)
Contribution 2	Inhibition of mitochondrial complex I reverses NOTCH1-driven metabolic reprogramming in T-cell acute lymphoblastic leukemia	48	Dhanasar — Prong 2 (well-positioned)
Contribution 3	Anticancer imidazoacridinone C-1311 inhibits hypoxia-inducible factor-1α (HIF-1α), vascular endothelial growth factor (VEGF) and angiogenesis	18	Dhanasar — Prong 2 (well-positioned)