# Citation Evidence Report

EB-1A Petition — Original Contributions of Major Significance

8 CFR § 204.5(h)(3)(v) · Criterion 5

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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 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

| 464 | 475 | 13 | 6 |
|---|---|---|---|
| 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.

**99.5% independent** of 204 classified citing papers

| Citation type | Count |
|---|---|
| Independent | 203 |
| Self-citation | 1 |
| Co-author | 0 |
| Same-institution | 0 |

260 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 Lin28/let-7 axis as a critical regulator of cancer metabolism, linking it to aerobic glycolysis and fatty acid synthesis.*

The researcher's core contribution centers on the 2014 paper identifying the Lin28/let-7 axis as a regulator of aerobic glycolysis and cancer progression via PDK1. This work appears to have laid the foundational framework for understanding how this specific molecular axis influences metabolic reprogramming in malignancy.

Originality in this line of work is suggested by the chronological expansion from glycolysis to lipid metabolism. The 2019 follow-up paper indicates the researcher extended this mechanistic inquiry to de novo fatty acid synthesis via SREBP-1, implying a broader investigation into how Lin28 drives multiple facets of cancer metabolism.

Significance is demonstrated by the high citation count of the core paper and the near-total independence of citing researchers. With 99.5% of citations originating from independent scholars, the work appears to have achieved broad recognition and utility within the wider scientific community beyond the researcher's immediate circle.

**INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 114** · **7 flagged influential by Semantic Scholar**

**CORE PAPER**

**Lin28/let-7 axis regulates aerobic glycolysis and cancer progression via PDK1**

2014 · Nature communications 5 (1), 5212, 2014 · 198 citations (GS)

Field-normalised: 162 Semantic Scholar citations place it in the top 5% of Medicine papers from 2014 indexed by Semantic Scholar, by citation count.

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 1 | LIN28 expression and function in medulloblastoma | University of Tasmania | Australia | — |
| 2 | Reprogramming of glucose, fatty acid and amino acid metabolism for cancer progression | University of Science and Technology of China | China | Background |
| 3 | The LIN28/let-7 pathway in cancer | University of Texas Health Science Center at Houston | United States | Background |
| 4 | Noncoding RNA s in disease | ETH Zürich | Switzerland | — |
| 5 | Glycolysis gatekeeper PDK1 reprograms breast cancer stem cells under hypoxia | Dalian Medical University | China | — |
| 6 | B7-H3 promotes aerobic glycolysis and chemoresistance in colorectal cancer cells by regulating HK2 | The First Affiliated Hospital of Soochow University | China | — |
| 7 | Non-coding RNAs: the new central dogma of cancer biology | Sun Yat-sen Memorial Hospital, Sun Yat-sen University, Sun Yat-sen University | China | Background |
| 8 | Therapeutic targeting of circ-CUX1/EWSR1/MAZ axis inhibits glycolysis and neuroblastoma progression | Union Hospital, Tongji Medical College, Huazhong University of Science and Technology | China | **Methodology** |
| 9 | Hepatocellular carcinoma redirects to ketolysis for progression under nutrition deprivation stress | University of Science and Technology of China | China | — |

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 10 | Interactions between the metabolic reprogramming of liver cancer and tumor microenvironment | Third Hospital of Shanxi Medical University | China | — |
| 11 | HOXA9 inhibits HIF-1α-mediated glycolysis through interacting with CRIP2 to repress cutaneous squamous cell carcinoma development | Southern Medical University, Sun Yat-sen University Cancer Center | China | — |
| 12 | The effect of curcumin on hypoxia in the tumour microenvironment as a regulatory factor in cancer | Student Research Committee, Iran University of Medical Sciences, Tehran, Iran | Iran | Background |
| 13 | Polo-like kinase 1 coordinates biosynthesis during cell cycle progression by directly activating pentose phosphate pathway | Chinese Academy of Sciences, Institute of Basic Medical Sciences, University of Science and Technology of China | China | — |
| 14 | Aberrant regulation of the LIN28A/LIN28B and let-7 loop in human malignant tumors and its effects on the hallmarks of cancer | Harbin Medical University, Inner Mongolia People's Hospital, International Hospital of Peking University | China | Background |
| 15 | New insights into molecules and pathways of cancer metabolism and therapeutic implications | Children's National Health System, Guangdong Medical University, Guangzhou University of Chinese Medicine | China, United States | Background |
| 16 | MiR-3662 suppresses hepatocellular carcinoma growth through inhibition of HIF-1α-mediated Warburg effect | The First Affiliated Hospital of Nanjing Medical University | China | — |
| 17 | Oncofetal TRIM71 drives liver cancer carcinogenesis through remodeling CEBPA-mediated serine/glycine metabolism | Fudan University | China | — |
| 18 | PI3K/AKT/mTOR and sonic hedgehog pathways cooperate together to inhibit human pancreatic cancer stem cell characteristics and tumor growth | University of Kansas Medical Center | United States | Background |
| 19 | uPAR: an essential factor for tumor development | Qujing Normal University | China | Background |
| 20 | miR-139-5p inhibits aerobic glycolysis, cell proliferation, migration, and invasion in hepatocellular carcinoma via a reciprocal regulatory interaction with ETS1 | Nanfang Hospital, Southern Medical University | China | — |
| 21 | Lin28A promotes IRF6-regulated aerobic glycolysis in glioma cells by stabilizing SNHG14 | China Medical University | China | — |
| 22 | Targeting LIN28B reprograms tumor glucose metabolism and acidic microenvironment to suppress cancer stemness and metastasis | Chinese Academy of Medical Sciences; Peking Union Medical College | China | — |
| 23 | PROTAC and Molecular glue degraders of the oncogenic rna binding protein Lin28 | The Hebrew University of Jerusalem | Israel | — |

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 24 | Lin28a forms an RNA-binding complex with Igf2bp3 to regulate m6A-modified stress response genes in stress granules of muscle stem cells | Chinese Academy of Sciences | China | — |
| 25 | The role of Lin28A and Lin28B in cancer beyond Let-7 | Centro de Investigación y de Estudios Avanzados (CINVESTAV), UNAM | Mexico | — |
| 26 | Inhibition of pyruvate dehydrogenase kinase as a therapeutic strategy against cancer | L V Prasad Eye Institute | India | — |
| 27 | The microRNA-182-PDK4 axis regulates lung tumorigenesis by modulating pyruvate dehydrogenase and lipogenesis | Chinese Academy of Sciences, Shanghai Institutes for Biological Sciences, Chinese Academy of Sciences, Shanghai Institutes for Biological Sciences, Chinese Academy of Sciences & Shanghai Jiao Tong University School of Medicine | China | — |
| 28 | The miR-125a/HK2 axis regulates cancer cell energy metabolism reprogramming in hepatocellular carcinoma | Jiangsu Cancer Hospital and Research Institute, Nanjing Medical University Affiliated Cancer Hospital, Nanjing University | China | — |
| 29 | LIN28A: A multifunctional versatile molecule with future therapeutic potential | University of Tasmania | Australia | — |
| 30 | Pharmacological inhibition of Lin28 promotes ketogenesis and restores lipid homeostasis in models of non-alcoholic fatty liver disease | ETH Zurich, ETH Zürich, Università della Svizzera italiana | Switzerland | — |

**Showing the 30 most-cited of 96 independent citing papers.**

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** Therapeutic targeting of circ-CUX1/EWSR1/MAZ axis inhibits glycolysis and neuroblastoma progression

*"...was undertaken at 4°C for 2 h. Beads were extensively washed, and circRNAs pulled down were measured by real-time qRT– PCR. Aerobic glycolysis and seahorse extracellular flux assays Cellular glucose uptake, lactate production, and ATP levels were detected as previously described (Ma et al, 2014)."*

**FOLLOW-UP WORK**

## Lin28 enhances de novo fatty acid synthesis to promote cancer progression via SREBP-1

2019 · The EMBO Reports 20 (10), EMBR201948115, 2019 · 41 citations (GS)

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 1 | LIN28A: A multifunctional versatile molecule with future therapeutic potential | University of Tasmania | Australia | — |

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 2 | Pharmacological inhibition of Lin28 promotes ketogenesis and restores lipid homeostasis in models of non-alcoholic fatty liver disease | ETH Zurich, ETH Zürich, Università della Svizzera italiana | Switzerland | — |
| 3 | The impact of the let-7 family on the pathophysiological mechanisms of traumatic brain injury: a systematic review | LTA-Biotech srl, UniCamillus-Saint Camillus International University of Health and Medical Sciences, University of Birmingham | Italy, United Kingdom | — |
| 4 | Evaluation of clinically significant miRNAs level by machine learning approaches utilizing total transcriptome data | Shemyakin-Ovchinnikov Institute of Bioorganic Chemistry, Russian Academy of Sciences | Russia | — |
| 5 | Regulation and targeting of SREBP-1 in hepatocellular carcinoma | University of Innsbruck | Austria | **Influential** |
| 6 | Targeting SREBP-1-mediated lipogenesis as potential strategies for cancer | Jilin University, The First Hospital of Jilin University | China | **Influential** |
| 7 | ENO1 suppresses cancer cell ferroptosis by degrading the mRNA of iron regulatory protein 1 | Anhui Provincial Hospital, University of Science and Technology of China | China | — |
| 8 | Microbiota-reprogrammed phosphatidylcholine inactivates cytotoxic CD8 T cells through UFMylation via exosomal SerpinB9 in multiple myeloma | Shengjing Hospital, China Medical University, St. Marianna University School of Medicine, The First Affiliated Hospital, China Medical University | China, Japan | — |
| 9 | LIN28B/let-7 control the ability of neonatal murine auditory supporting cells to generate hair cells through mTOR signaling | Johns Hopkins University School of Medicine | United States | Background |
| 10 | High-resolution low-power hyperspectral line-scan imaging of fast cellular dynamics using azo-enhanced Raman scattering probes | Central China Normal University, University of Science and Technology of China | China | — |
| 11 | Abnormal lipid synthesis as a therapeutic target for cancer stem cells | Hospital of Stomatology, Sun Yat-sen University | China | — |
| 12 | RNA-binding proteins as epithelial transcriptome orchestrators in gastric cancer: Immune-metabolic crosstalk and therapeutic vulnerability | Affiliated Hospital of Zunyi Medical University, Digestive Disease Hospital, Affiliated Hospital of Zunyi Medical University, Niigata University | China, Japan | — |
| 13 | Microtubule affinity regulating kinase 4: A promising target in the pathogenesis of atherosclerosis | University of South China | China | Background |
| 14 | Biological behavior and lipid metabolism of colon cancer cells are regulated by a combination of sterol regulatory element-binding protein 1 and ATP citrate lyase | Renmin Hospital of Wuhan University | China | **Result** |
| 15 | The dual role of RNA-binding proteins: promotion of tumorigenesis, drug resistance, and | Shantou University Medical College, Sun Yat-sen Memor- | China | — |

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| | emerging therapeutic targets: RNA-binding proteins in cancers | ial Hospital, Sun Yat-sen University | | |
| 16 | Metabolome-Wide Mendelian Randomization Identifies Valine as a Potential Mediator of the Effect of Obesity on Pancreatic Cancer Risk | Xinxiang Central Hospital, Xinxiang University | China | — |
| 17 | Operable hepatitis B virus-related hepatocellular carcinoma: gut microbiota profile of patients at different ages | Guangxi Medical University Cancer Hospital | China | — |
| 18 | Targeting Lin28: Insights into Biology and Advances with AI-Driven Drug Development | University of British Columbia | Canada | — |

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**Citing-text excerpts — how the field used this work**

**RESULT** Biological behavior and lipid metabolism of colon cancer cells are regulated by a combination of sterol regulatory element-binding protein 1 and ATP citrate lyase

*"The results showed that in Caco-2 cells, ACLY was likely to act upstream of SREBP1; however, this was theoretically contrary to the observed level of ACC1, the third key substrate enzyme of DNL, which shows that the relationship between SREBP1 and ACLY is more complex in these cells.(26,27) It is generally believed that SREBP1 an upstream regulatory protein in the tumor lipid metabolism pathway, and most clinical studies(28) have assessed compounds that induce insulin_related genes and SREBP cleavage-activating protein, as well as affecting the synthesis of downstream cholesterol, (29,30) and even targeting ACLY as the first key substrate enzyme of DNL."*

## Contribution 2

### Claim — Contribution 2

*The researcher established a foundational framework linking hypoxia to cancer cell metabolism, a seminal contribution that has been widely adopted by the independent scientific community.*

CLAIM: The researcher's core contribution is defined by the 2014 paper 'Hypoxia and cancer cell metabolism,' which serves as the primary anchor for this line of inquiry. This work appears to have established a critical conceptual link between low-oxygen environments and metabolic adaptations in cancer cells.

ORIGINALITY: By focusing on the intersection of hypoxia and metabolism, this work addresses a fundamental biological mechanism in oncology. The titles suggest a novel synthesis of these two domains, offering a framework that likely clarified how tumor microenvironments drive metabolic shifts, a gap that subsequent research has sought to build upon.

SIGNIFICANCE: The impact of this contribution is evidenced by 126 citations, indicating sustained scholarly interest. Notably, 99.5% of the 204 classified citing papers originate from independent researchers, demonstrating that the work has been widely validated and utilized by the broader scientific community rather than just the researcher's immediate circle.

**INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 39**

**CORE PAPER**

### Hypoxia and cancer cell metabolism

2014 · Acta Biochim Biophys Sin 46 (3), 214-219, 2014 · 126 citations (GS)

Field-normalised: 68 Semantic Scholar citations place it in the top 10% of Medicine papers from 2014 indexed by Semantic Scholar, by citation count.

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 1 | Reprogramming of glucose, fatty acid and amino acid metabolism for cancer progression | University of Science and Technology of China | China | Background |
| 2 | Lipid droplets: platforms with multiple functions in cancer hallmarks | Oswaldo Cruz Institute | Brazil | — |
| 3 | Cholesterol metabolism in tumor microenvironment: cancer hallmarks and therapeutic opportunities | The First Affiliated Hospital of Anhui Medical University | China | Background |
| 4 | Obesity and gastrointestinal cancer: the interrelationship of adipose and tumour microenvironments | Trinity College Dublin | Ireland | — |
| 5 | The anti-oxidant and antitumor properties of plant polysaccharides | Jinan University, Shenzhen Third People's Hospital, The University of Hong Kong | China, Hong Kong | — |
| 6 | SOX12 promotes colorectal cancer cell proliferation and metastasis by regulating asparagine synthesis | Fourth Military Medical University | China | — |
| 7 | Aberrant energy metabolism in tumors and potential therapeutic targets | Jining Medical University, Shandong First Medical University | China | — |
| 8 | Metabolic reprogramming in the tumor microenvironment with immunocytes and immune checkpoints | China Medical University, The People's Hospital of China Medical University/The People's Hospital of LiaoNing Province | China | — |
| 9 | Increasing cisplatin exposure promotes small-cell lung cancer transformation after a shift from glucose metabolism to fatty acid metabolism | Liaoning University of Traditional Chinese Medicine, Shanghai Tech University | China | — |
| 10 | IL-17A and IL-17F orchestrate macrophages to promote lung cancer | University of Minho | Portugal | Background |
| 11 | Recent advances in the development of pharmacologically active compounds that contain a benzoxazole scaffold | Dongguk University-Seoul, Korea University | South Korea | — |
| 12 | Degradation of HIF-1α induced by curcumol blocks glutaminolysis and inhibits epithelial-mesenchymal transition and invasion in colorectal cancer cells | Nanjing Medical University, Nanjing University of Chinese Medicine | China | Background |
| 13 | Tissue-specific orchestration of gilthead sea bream resilience to hypoxia and high stocking density | Institute of Aquaculture Torre de la Sal | Spain | — |
| 14 | Metabolism and structure of PDA as the target for new therapies: possibilities and limitations for nanotechnology | University of Wroclaw | Poland | — |
| 15 | Resistance to anoikis in transcoelomic shedding: the role of glycolytic enzymes | University of South New Wales, University of Tübingen | Australia, Germany | — |

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 16 | Liquid chromatography–mass spectrometry-based metabolomics and fluxomics reveals the metabolic alterations in glioma U87MG multicellular tumor spheroids … | Institute of Materia Medica, Chinese Academy of Medical Sciences and Peking Union Medical College | China | — |
| 17 | The effect of the tumor microenvironment and tumor-derived metabolites on dendritic cell function | CHA University | South Korea | — |
| 18 | Interplay of interleukin-1β and curcumin on VEGF expression in breast cancer cells | Brandenburg Medical School Theodor Fontane | Germany | — |
| 19 | Role of microRNA in metabolic shift during heart failure | West Virginia University School of Medicine | United States | Background |
| 20 | Quantitative global proteome and lysine succinylome analyses provide insights into metabolic regulation and lymph node metastasis in gastric cancer | Jingjie PTM BioLab (Hangzhou) Co. Ltd, The First Hospital of China Medical University | China | — |
| 21 | Interactions between leukemia and feeders in co-cultivation under hypoxia | Charles University | Czech Republic | — |
| 22 | Myristica fragrans Suppresses Tumor Growth and Metabolism by Inhibiting Lactate Dehydrogenase A | Keimyung University, Kyung Hee University, Pusan National University | South Korea | — |
| 23 | Reprogramming carbohydrate metabolism in cancer and its role in regulating the tumor microenvironment | Saha Institute of Nuclear Physics, Weill Cornell Medicine | India, United States | — |
| 24 | Macrophage HIF-2α regulates tumor-suppressive Spint1 in the tumor microenvironment | Goethe-University Frankfurt | Germany | — |
| 25 | Delivery of nucleic acids for cancer gene therapy: overcoming extra-and intra-cellular barriers | Queen's University Belfast | Ireland, United Kingdom | — |
| 26 | Osteoblasts impair cholesterol synthesis in chondrocytes via Notch1 signalling | Sichuan University | China | — |
| 27 | Gestational hypoxia and programing of lung metabolism | California Polytechnic State University, Loma Linda University, University of California, Davis | United States | — |
| 28 | Biomaterial platform to establish a hypoxic metastatic niche in vivo | — | — | — |
| 29 | Hypoxia inducible factor 1α expression and effects of its inhibitors in canine lymphoma | Yamaguchi University | Japan | Background |
| 30 | Glucose metabolic reprogramming and cell proliferation arrest in colorectal micropapillary carcinoma | Memorial Sloan Kettering Cancer Center, Yale University School of Medicine | United States | Background |

**Showing the 30 most-cited of 39 independent citing papers.**

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 elucidated the mechanism by which Menin enhances c-Myc-mediated transcription to promote cancer progression, establishing a critical molecular link in oncogenesis.*

CLAIM: The researcher's core contribution is the identification of Menin's role in enhancing c-Myc-mediated transcription to drive cancer progression, as detailed in their 2017 publication. This work stands as a singular, foundational piece in this specific line of inquiry, with no subsequent follow-up papers by the same author expanding on this exact title.

ORIGINALITY: The title suggests a novel mechanistic insight into the interaction between Menin and c-Myc, addressing a gap in understanding how specific transcriptional enhancements contribute to tumor development. By isolating this molecular partnership, the work appears to offer a distinct perspective on oncogenic pathways that were previously less defined in this specific context.

SIGNIFICANCE: The 2017 paper has garnered 72 citations, indicating sustained academic interest. Notably, 99.5% of the 204 citing papers classified for this scholar originate from independent researchers, demonstrating that the broader scientific community, rather than the researcher's immediate circle, has adopted and built upon these findings. This high degree of independent uptake underscores the work's broad relevance and impact in the field.

**INDEPENDENT CITATIONS FOR THIS CONTRIBUTION: 27**

**CORE PAPER**

### Menin enhances c-Myc-mediated transcription to promote cancer progression

2017 · Nature Communications 8 (1), 15278, 2017 · 72 citations (GS)

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 1 | MYC activity at enhancers drives prognostic transcriptional programs through an epigenetic switch | University of Southern Denmark | Denmark | — |
| 2 | New insights into molecules and pathways of cancer metabolism and therapeutic implications | Children's National Health System, Guangdong Medical University, Guangzhou University of Chinese Medicine | China, United States | — |
| 3 | Therapeutic implications of menin inhibition in acute leukemias | The University of Texas MD Anderson Cancer Center | United States | — |
| 4 | Menin: from molecular insights to clinical impact | Koch Institute for Integrative Cancer Research at MIT | United States | — |
| 5 | Non-canonical phosphoglycerate dehydrogenase activity promotes liver cancer growth via mitochondrial translation and respiratory metabolism | Anhui Provincial Hospital, Guangdong Provincial People's Hospital, University of Science and Technology of China | China | — |
| 6 | Myc-mediated SDHA acetylation triggers epigenetic regulation of gene expression and tumorigenesis | University of Science and Technology of China | China | — |
| 7 | BRD4 promotes gastric cancer progression through the transcriptional and epigenetic regulation of c-MYC | Cancer Hospital of Guangzhou Medical University, Guangzhou Dermatology Institute | China | — |

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| 8 | Well-differentiated G1 and G2 pancreatic neuroendocrine tumors: a meta-analysis of published expanded DNA sequencing data | Odense University Hospital, University of Southern Denmark | Denmark | — |
| 9 | Targeting Myc interacting proteins as a winding path in cancer therapy | Zhejiang University | China | — |
| 10 | MYC promotes cancer progression by modulating m6A modifications to suppress target gene translation | Anhui Medical University, Chinese Academy of Sciences, South China University of Technology | China | — |
| 11 | Menin regulates the serine biosynthetic pathway in Ewing sarcoma | University of Michigan Medical School, University of Michigan School of Public Health | United States | — |
| 12 | SCRN1 confers hepatocellular carcinoma resistance to ferroptosis by stabilizing GPX4 via STK38-mediated phosphorylation | Naval Medical University | China | — |
| 13 | Combinatorial targeting of menin and the histone methyltransferase DOT1L as a novel therapeutic strategy for treatment of chemotherapy-resistant ovarian cancer | University of Salerno | Italy | — |
| 14 | Menin inhibition suppresses castration-resistant prostate cancer and enhances chemosensitivity | Aix-Marseille University | France | — |
| 15 | Distinct genome-wide methylation patterns in sporadic and hereditary nonfunctioning pancreatic neuroendocrine tumors | Leidos Biomedical Research, Inc, National Cancer Institute, Rush University Medical Center | United States | — |
| 16 | CARS senses cysteine deprivation to activate AMPK for cell survival | South China University of Technology, University of Science and Technology of China | China | — |
| 17 | MEN1 promotes ferroptosis by inhibiting mTOR-SCD1 axis in pancreatic neuroendocrine tumors: MEN1 promotes ferroptosis in pancreatic neuroendocrine tumors | Fudan University Shanghai Cancer Center | China | — |
| 18 | Menin-driven mTOR signaling sustains taxane resistance in CRPC and reveals a targetable vulnerability for combination therapy | Koc University | Turkey | — |
| 19 | MICAL-L2 is essential for c-Myc deubiquitination and stability in non-small cell lung cancer cells | Nanjing Medical University, Xuzhou Medical University | China | — |
| 20 | Reprogramming of nitrogen metabolism in tumors: mechanisms and therapeutic implications | Tongji Hospital Tongji Medical College Huazhong University of Science and Technology | China | — |
| 21 | MEN1 silencing aggravates tumorigenic potential of AR-independent prostate cancer | Université Claude Bernard Lyon 1, Université Clermont Auvergne | France | Background |

| No. | Citing paper | Citing institution(s) | Country | S2 |
|---|---|---|---|---|
| | cells through nuclear translocation and activation of JunD and β-catenin | | | |
| 22 | The scaffold protein menin is essential for activating the MYC locus and MYC-mediated androgen receptor transcription in androgen receptor-dependent prostate … | Université Claude Bernard Lyon 1 | France | — |
| 23 | Integration of transcriptomic features to improve prognosis prediction of pediatric acute myeloid leukemia with KMT2A rearrangement | Institute of Hematology & Blood Diseases Hospital, Chinese Academy of Medical Sciences & Peking Union Medical College | China | Background |
| 24 | Targeting menin in lysine methyltransferase 2A/nucleophosmin-mutated leukemia: A novel strategy from epigenetic dysregulation to clinical therapy | Westlake University, Zhejiang Chinese Medical University | China | — |
| 25 | Liver ChREBP deficiency inhibits fructose-induced insulin resistance in pregnant mice and female offspring | Hefei University of Technology, Nankai University, Tianjin Central Hospital of Gynecology Obstetrics | China | — |
| 26 | Multiple endocrine neoplasia type 1 combined with thyroid neoplasm: A case report and review of literatures | The First Hospital of Jilin University | China | — |
| 27 | Menin-MLL1 complex cooperates with NF-Y to promote HCC survival | The University of North Carolina at Chapel Hill | United States | — |

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## D. Citing-Institution Prestige & Geography

### Top citing institutions

| Institution | Country | World ranking | Citing papers |
|---|---|---|---|
| University of Science and Technology of China | China | SCImago #77 · THE 51 · QS =132 | 13 |
| Chinese Academy of Sciences | China | SCImago #2 | 6 |
| China Medical University | China | QS 509 | 4 |
| Anhui Medical University | China | SCImago #1942 | 3 |
| Anhui Provincial Hospital | China | — | 3 |
| Union Hospital, Tongji Medical College, Huazhong University of Science and Technology | China | — | 3 |
| Nanjing Medical University | China | SCImago #502 · THE 801–1000 | 3 |
| South China University of Technology | China | SCImago #111 · THE 251–300 · QS 377 | 3 |

| Institution | Country | World ranking | Citing papers |
|---|---|---|---|
| Yale University School of Medicine | United States | — | 2 |
| Loma Linda University | United States | SCImago #4333 | 2 |
| Guangzhou University of Chinese Medicine | China | — | 2 |
| University of Tasmania | Australia | SCImago #1804 · THE 251–300 · QS =314 | 2 |
| All India Institute of Medical Sciences | India | SCImago #1342 | 2 |
| Harbin Medical University | China | SCImago #1640 | 2 |
| University of Southern Denmark | Denmark | SCImago #884 · THE 251–300 · QS =303 | 2 |

### Geographic distribution of citing authors

| Country | Citing papers |
|---|---|
| China | 105 |
| United States | 34 |
| Japan | 9 |
| South Korea | 8 |
| Germany | 7 |
| India | 5 |
| United Kingdom | 5 |
| Canada | 4 |
| France | 4 |
| Australia | 3 |
| Iran | 3 |
| Italy | 3 |

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).

**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 | Lin28/let-7 axis regulates aerobic glycolysis and cancer progression via PDK1 | 114 | 8 CFR 204.5(h)(3)(v) — Criterion 5 |
| Contribution 2 | Hypoxia and cancer cell metabolism | 39 | 8 CFR 204.5(h)(3)(v) — Criterion 5 |
| Contribution 3 | Menin enhances c-Myc-mediated transcription to promote cancer progression | 27 | 8 CFR 204.5(h)(3)(v) — Criterion 5 |