REVIEW ARTICLE

Cordycepin in cancer therapy: A bibliometric analysis and review of mechanisms

Zhiwei Ouyang, Yufei Zhang, Jianghan Ning, Yayi Tu*, Bin He**

Jiangxi Key Laboratory of Natural Microbial Medicine Research, College of Life Sciences, Jiangxi Science & Technology Normal University, Nanchang 330013, Jiangxi, China

Abstract

Cordycepin (3′-deoxyadenosine), a major bioactive component derived from fungi of the genus Cordyceps, has garnered significant attention in recent years for its potent antitumor properties. Drawing on literature indexed in the Web of Science Core Collection from 2004 to 2025, this study employs bibliometric tools—specifically CiteSpace and VOSviewer—to systematically examine developmental trends, research hotspots, and emerging frontiers in the field of cordycepin-related cancer research. The analysis maps a shift in focus from early-stage pharmacological validation to more advanced investigations into molecular mechanisms, with particular emphasis on cell cycle regulation. Keyword burst analysis highlights bursts in terms such as "apoptosis," "cell cycle," "gene," and "expression," underscoring that modulating the cell cycle to induce cancer cell apoptosis has become a central research theme. Building on these findings, the review further delineates the specific molecular mechanisms by which cordycepin regulates cell cycle progression in various tumor types—primarily through downregulation of Cyclin/CDK complexes, upregulation of *p21* and *p27*, and activation of DNA damage response pathways. Additionally, growing evidence indicates that cordycepin's influence on gene expression and epigenetic modulation is emerging as a critical area of focus. Taken together, cordycepin demonstrates multitargeted potential in inhibiting tumor growth, positioning it as a promising candidate for natural anticancer drug development. Future research should prioritize pharmacokinetic characterization, investigation of combinatorial therapeutic strategies, and pathways toward clinical translation. Intracellular exposure appears to be shaped by two complementary axes: interference with 3'end polyadenylation and ENT1/ENT2-mediated uptake with ADA-catalyzed deamination.

Keywords: Adenosin deaminase (ADA), Bibliometrics, Cordycepin, Cancer, Cell cycle

1. Introduction

Cordycepin, a natural derivative of adenosine primarily isolated from fungi of the genus Cordyceps—especially *Cordyceps militaris*—was first successfully extracted and reported by Cunningham et al. in Nature in 1950 [1]. Since then, it has garnered considerable interest in natural product research due to its broad spectrum of bioactivities and promising pharmacological properties, including antitumor, anti-inflammatory, antiviral, immunomodulatory, and neuroprotective effects [2–10]. With advances in modern life sciences, increasing attention has been directed toward the molecular mechanisms and therapeutic potential of cordycepin, particularly its anticancer effects [11].

Cordycepin has demonstrated antitumor efficacy across various cancer types, including breast, lung, liver, and colon cancers, as well as gliomas and bladder cancer [12–16]. Its antitumor mechanisms include induction of apoptosis, inhibition of cell proliferation, and suppression of tumor metastasis and invasion [17,18]. Among these, regulation of the cell cycle has emerged as a central focus [11]. Disruption of cell cycle control is a hallmark of tumor development, as cancer cells frequently bypass G1/S and G2/M checkpoints to sustain uncontrolled proliferation [19]. Consequently, targeting the cell cycle has become a pivotal strategy in anticancer therapy [20].

Existing studies indicate that cordycepin interferes with tumor cell cycle progression through multiple molecular pathways. It can induce G1/S or

Received 14 July 2025; accepted 1 October 2025.
Available online 15 December 2025

* Corresponding author.
** Corresponding author.
E-mail addresses: tuyayi@126.com (Y. Tu), hebin.li@foxmail.com (B. He).

https://doi.org/10.38212/2224-6614.3566

G2/M phase arrest by upregulating cyclin-dependent kinase inhibitors (e.g., *p21* and *p27*), downregulating Cyclin D1, Cyclin B1, and associated CDK complexes, and activating DNA damage response pathways such as ATM/CHK1 [21,22]. Furthermore, cordycepin modulates key signaling pathways—including p53, JNK, and PI3K—that contribute synergistically to its cell cycle-regulatory effects, thereby highlighting its multitargeted therapeutic profile [23,24].

Despite these advances, research on the anticancer mechanisms of cordycepin remains fragmented, and a comprehensive, systematic review of its developmental and mechanistic progress is still lacking. Notably, no bibliometric studies have yet been conducted to map cordycepin-related oncology research. To address this gap, the present study employs bibliometric techniques to visualize trends in cordycepin-focused cancer research from 2004 to 2025, with a particular emphasis on cell cycle regulatory mechanisms. By integrating burst keyword analysis, temporal evolution mapping, and mechanistic exploration, this review seeks to clarify developmental trajectories, identify emerging research frontiers, and highlight key challenges—ultimately providing a theoretical foundation for future mechanistic investigations and translational research on cordycepin.

We therefore present the bibliometric trends as descriptive, methods-transparent context to organize the cordycepin literature and do not claim discovery or novelty at the mechanistic level.

2. Materials and methods

2.1. Data source and search strategy

All records were retrieved from the Web of Science Core Collection (WoSCC), which provides standardized bibliographic and citation metadata across disciplines- [25,26]. We limited the Topic field (TS: title, abstract, author keywords, and Keywords Plus) using the Boolean query: TS = (cordycepin OR "3′-deoxyadenosine") AND TS = (cancer OR tumor OR tumour OR neoplasm OR anti-tumor OR anticancer). The time span was January 1, 2004, to June 20, 2025 (data freeze: June 20, 2025). Document types were restricted to Articles and Reviews; conference papers, book chapters, editorials, meeting abstracts, corrections, and retracted items were excluded. We exported Full Record and Cited References with authors' full names, addresses, and funding fields to enable collaboration and affiliation analyses. The raw file was saved as plain text (download_1.txt).

2.2. Data cleaning and quality control

We removed duplicates by matching DOI and WoS unique identifier (UT), and resolved "Early Access" vs. final versions by retaining the latest non-duplicative record. Records explicitly flagged as retracted in WoS were excluded. Author names were normalized to full names (when available); institutional names were standardized by rule-based normalization (e.g., Univ. → University, Hosp. → Hospital) and alias merging (e.g., China Med Univ., China Medical Univ. → China Medical University). For country-level analyses, Taiwan, Hong Kong, and Macau were merged into China as per our predefined geographic policy. All steps were double-checked on random samples to ensure reproducibility.

2.3. Bibliometric tools and parameter settings

We used VOSviewer (v1.6.20) and CiteSpace (v6.1.6) [27,28]. In VOSviewer, networks were constructed under full counting with association strength normalization. Thresholds were: keywords (minimum occurrences ≥5), authors (≥2), and institutions (≥3), unless otherwise specified [29–32]. In CiteSpace, settings were time slicing 2004–2025, 1-year per slice; term sources: Title, Abstract, Author Keywords, Keywords Plus; node types: countries, institutions, authors, keywords. Node selection used g-index (k = 25) per slice with Pathfinder and pruning sliced networks enabled. Keyword bursts followed Kleinberg's algorithm [33], as adapted and implemented in CiteSpace by Chen et al. [34–36]. Cluster labeling used LLR, and timeline views summarized topic evolution.

To enhance transparency, Author Keywords (DE) and Keywords Plus (ID) were analyzed separately for frequency profiles; minimal harmonization was applied to DE (e.g., unifying "G2/M phase arrest" to "G2/M arrest"), while ID terms were left unmerged and used to complement thematic mapping. In addition, we overlaid co-word/clustering outputs with a curated cordycepin mechanism set (e.g., 3′-end processing/post-transcription, ENT1/ENT2–ADA exposure axis, DDR/checkpoints, ncRNA–EMT) to label themes as specific (cordycepin-specific) or generic (oncology-general), encoded via distinct outline styles.

2.4. Collaboration network construction

A country-country/institution–institution collaboration was counted when at least two countries

co-occurred in the C1 "Addresses" field of the same article; an institution–institution collaboration was counted when at least two standardized institutions co-occurred. Each pair was counted once per article to avoid duplication. Collaboration intensity is reported as Coauthored Papers. Visualization conventions followed mainstream bibliometric mapping practice—node size encodes output/impact and edge thickness encodes collaboration strength; color denotes temporal attributes (e.g., first-year of appearance)—as implemented in VOSviewer/CiteSpace-based analyses [31,32,37,38].

2.5. Indicators and validation

We computed within-set h-index and total citations for journals, authors, and institutions. Journal impact factors and quartiles (Q1–Q4) were taken from JCR 2024/2025 (retrieval date: 2025-08-15). Descriptive patterns from VOSviewer/CiteSpace were cross-checked against frequency-based tables (e.g., top keywords, top collaboration pairs). Sensitivity checks (e.g., varying keyword thresholds from 3 to 8) yielded qualitatively consistent hotspot structures. We did not run an independent Bibliometrix (R-package) pipeline; instead, we cross-checked descriptive counts against VOSviewer/CiteSpace outputs and performed threshold sensitivity checks.

3. Results

3.1. Publication quantity analysis

From January 2004 to June 2025, we retrieved 432 articles on cordycepin and cancer. As shown in Fig. 1, the annual publication count remained below 10 during 2004–2013, reflecting an exploratory phase. Starting in 2014, yearly output stabilized above 20, with the cumulative total reaching 81.09% of all publications, indicating sustained growth in scholarly attention to cordycepin in oncology.

3.2. National output and international collaboration

As shown in Fig. 2A and B and Table 1, 36 countries contributed publications to cordycepin–oncology research. China led the field with 247 papers (58.39%), followed by South Korea (n = 65, 15.37%) and the United States (n = 36, 8.51%). This distribution aligns with the historical prominence of Cordyceps research in East Asia. Beyond volume-based rankings, an address co-occurrence analysis delineated the structure of cross-border collaboration. We identified 87 country–country coauthorship links, with the top ten dyads accounting for 50.6% of all international collaborations (Fig. 3A; Supplementary Fig. S1 (https://doi.org/10.38212/2224-6614.3566)). The most active dyads were China–United States, South Korea–United States, and China–

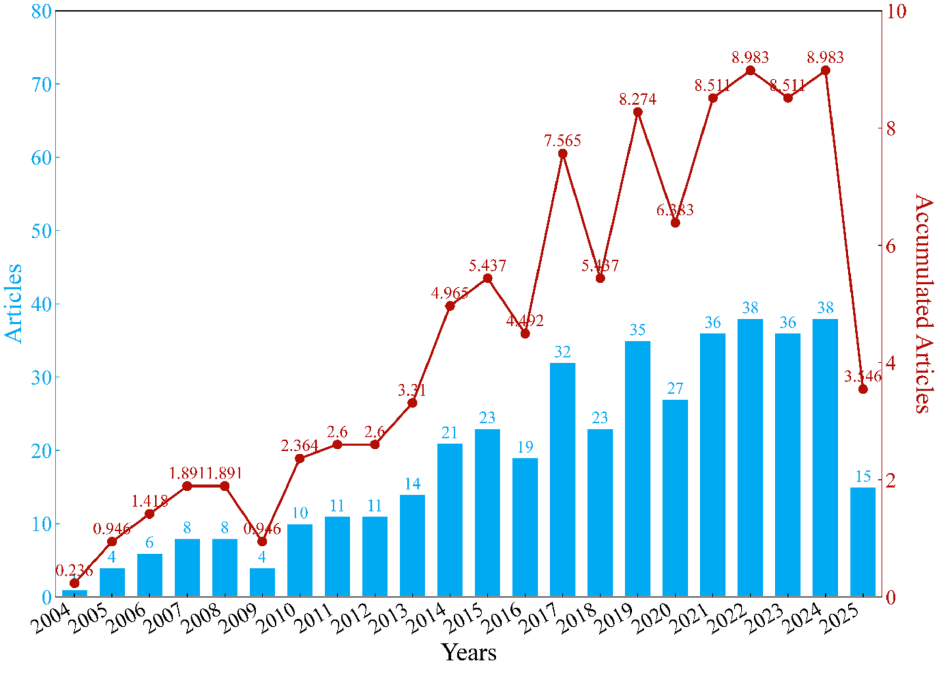

Fig. 1. Annual publication volume and percentage.

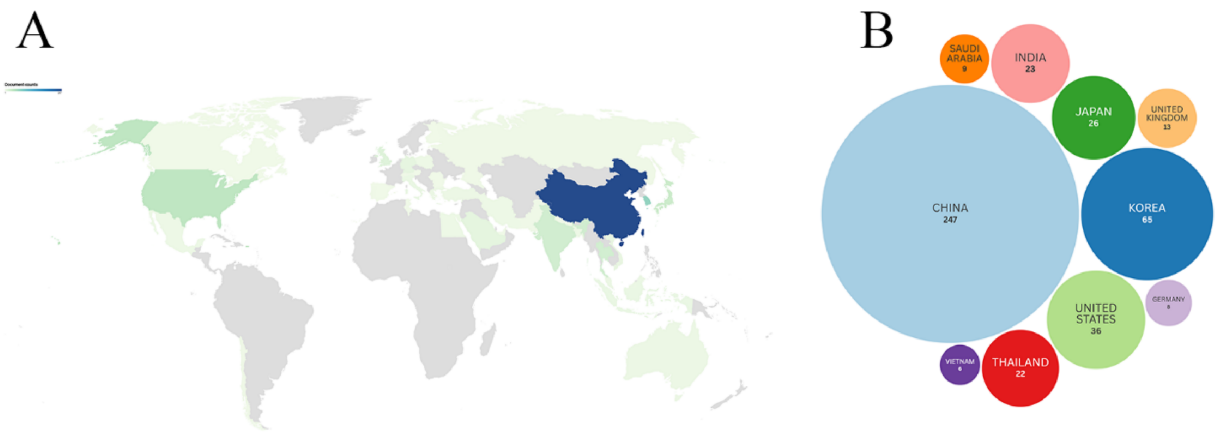

Fig. 2. Country output. (A) Global distribution by number of publications; (B) Top-10 countries by number of publications.

Table 1. Specific publication data for the top 10 countries.

Country	Articles	Fre = articles/total articles
China	247	58.392
Korea	65	15.366
United States	36	8.511
Japan	26	6.147
India	23	5.437
Thailand	22	5.201
United Kingdom	13	3.074
Saudi Arabia	9	2.128
Germany	8	1.891
Vietnam	6	1.418

Japan, indicating strong East Asia–North America linkages and a robust intra-Asia collaboration backbone. Notably, despite its comparatively lower topic-specific output, the United States appeared as a frequent partner to multiple countries, suggesting a bridging role in multinational teams. In accordance with the Methods, Taiwan, Hong Kong, and Macau were merged into China for country-level statistics. Collectively, these findings indicate that while scholarly output is concentrated in a few countries, international collaboration is organized along a limited number of high-traffic corridors, which complements simple volume rankings.

3.3. Institutional collaboration

In total, 350 institutions contributed to cordycepin–oncology publications during 2004–2025. The top 12 institutions (Table 2) are predominantly located in China (n = 7), followed by South Korea (n = 4) and Japan (n = 1), with China Medical University ranking first (23 publications). Beyond output counts, the inter-institutional collaboration network exhibits a

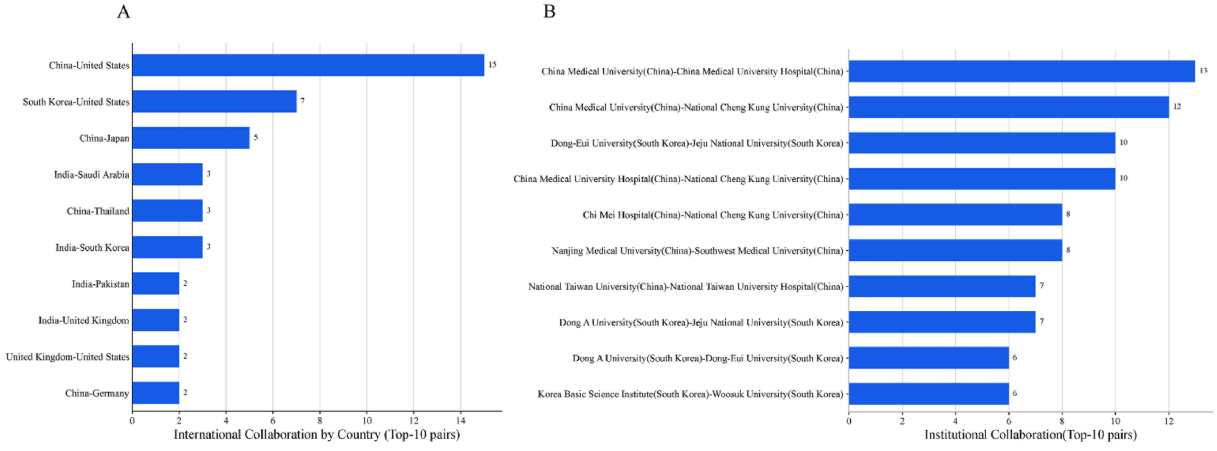

Fig. 3. Collaboration landscape in cordycepin–oncology research (2004–2025). (A) International collaboration by country (Top-10 pairs), showing coauthored papers and their share of all cross-border collaborations. (B) Institutional collaboration (Top-10 pairs, with country annotations), highlighting the long-tail pattern at the institutional level. Note: Taiwan, Hong Kong, and Macau were merged into China; institutional names were standardized prior to pairing.

Table 2. Top 12 institutions in terms of number of publications.

Institution	Articles
China Med Univ	23
Natl Cheng Kung Univ	18
Southwest Med Univ	16
Dong A Univ	10
Dong Eui Univ	10
Chungbuk Natl Univ	10
Nanjing Med Univ	9
Jilin Univ	9
Jeju Natl Univ	8
Chi Mei Med Ctr	8
Nanjing Tech Univ	8
Mukogawa Womens Univ	8

Table 3. Top 11 authors by number of publications.

Authors	Articles
Huang, Bu-Miin	15
Cheng, Jingliang	13
Fu, Junjiang	13
Fu, Jiewen	10
Choi, Yung Hyun	9
Liu, Xiaoyan	7
Li, Dabing	7
Zhang, Lianmei	6
Kim, Gi-Young	6
Du, Jiaman	6
Wei, Chunli	6

marked long-tail structure: we identified 2585 institution–institution coauthorship edges in total, whereas the Top-10 pairs together account for only 2.6% of all institutional collaborations (Fig. 3B; Table S2 (https://doi.org/10.38212/2224-6614.3566)). This indicates that collaborations are broadly distributed across numerous dyads rather than concentrated among a small set of recurrent partners. To improve fidelity, institutional names were standardized and merged across common aliases prior to pairing (e.g., China Med Univ. and China Medical Univ. → China Medical University), although minor residual heterogeneity due to address formatting cannot be fully excluded and is acknowledged in the Limitations. A CiteSpace visualization illustrates the overall community structure (Supplementary Fig. S2 (https://doi.org/10.38212/2224-6614.3566)), in which several regional clusters are apparent, while cross-regional ties remain comparatively sparse—suggesting headroom for strengthening multi-center collaboration across institutions.

3.4. Author influence and collaboration analysis

From 2004 to June 2025, 561 authors contributed to publications related to cordycepin and tumors. As shown in Table 3, Huang, Bu-Miin led with 15 publications (since 2007), followed by Cheng, Jingliang and Fu, Junjiang (both n = 13). Affiliations were standardized from the C1 address field; notably, Huang, Bu-Miin is affiliated with the Department of Cell Biology and Anatomy, College of Medicine, National Cheng Kung University, Tainan, Taiwan, whereas Cheng and Fu are affiliated with Southwest Medical University, Luzhou, China. Supplementary Fig. S3 (https://doi.org/10.38212/2224-6614.3566) depicts author collaboration communities centered on Huang, Bu-Miin, Cheng, Jingliang, Choi, Yung Hyun, Chen, Lujun, and Cho, Jae Youl. The relatively dispersed inter-cluster structure suggests headroom for strengthening cross-cluster, multi-center collaboration.

3.5. Research hotspots with DE/ID stratification

3.5.1. Frequency profiles of DE vs. ID

Across the corpus, we identified 459 unique keywords. To decouple author-asserted foci from database-generated expansions, we report Author Keywords (DE) and Keywords Plus (ID) separately.

DE (Fig. 4A): The top 15 are dominated by mechanism-oriented terms, with cell-cycle-related entries (e.g., "cell cycle," "G2/M arrest," "G1/S arrest," "p21/p27," "CDK1/Cyclin B1") alongside "apoptosis," "DNA damage," and checkpoint mediators (e.g., ATM/CHK1).

ID (Fig. 4B): The Top-15 surface broader thematic contexts (e.g., organism/source terms and pathway neighbors), complementing DE by revealing regulatory neighborhoods beyond author-asserted foci. Thus, although the hotspot labels overlap with general oncology themes, the DE-side weighting and ID-side bridging delineate a field-specific checkpoint/DDR → cycle arrest → cell death cascade (Figs. 4 and 5).

3.5.2. Co-occurrence structures (DE-focused)

We constructed keyword co-occurrence networks under full counting with association-strength normalization (Methods 2.3). Fig. 5 displays the DE-based network, which organizes into modules centered on cell-cycle control, DNA-damage response, and cell-death programs (apoptosis/autophagy). Relative to DE, an ID-based network (Supplementary Fig. S1) (https://doi.org/10.38212/2224-6614.3566) exhibits wider bridging to metabolism/inflammation-related terms and organism/source nodes, underscoring the complementary roles of DE and ID.

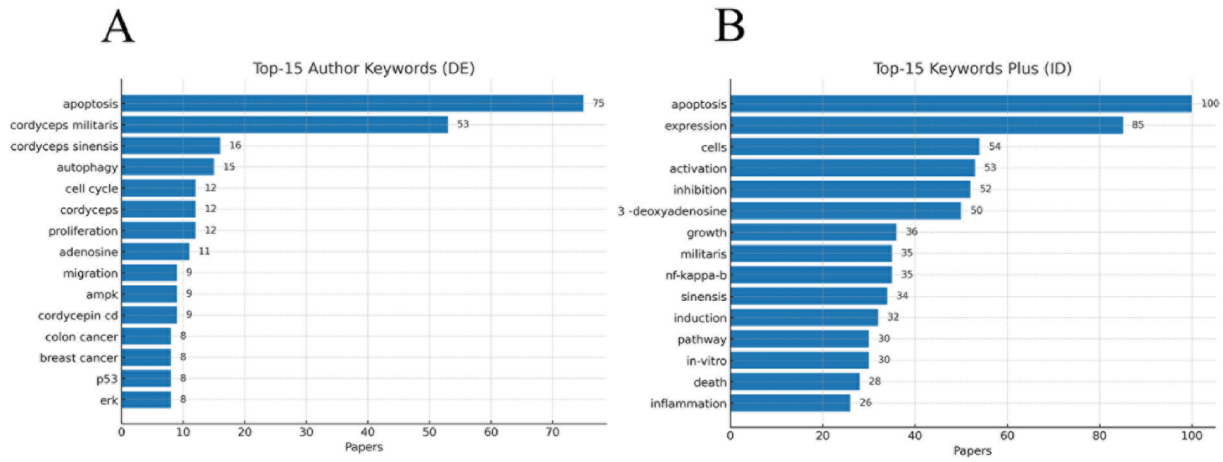

Fig. 4. High-frequency keywords by source: Author Keywords (DE) vs. Keywords Plus (ID). (A) Author Keywords (DE): Top-15; (B) Keywords Plus (ID): Top-15. Threshold: minimum occurrences ≥5 (Methods 2.3), VOSviewer full counting. DE represents author-asserted foci, whereas ID offers citation-derived expansions.

3.5.3. Burst detection and periodized trends

Burst detection (CiteSpace; Methods 2.3) identified top-bursting terms (Fig. 6) with "mechanism" showing the strongest recent burst (2023–2025). To align bursts with author-asserted foci, we further inspected DE-only time series of core mechanisms (cell cycle, G2/M/G1/S arrest, p21/p27, CDK1/Cyclin B1, ATM/CHK1, DNA damage), which collectively rose markedly after 2015, consistent with the field's transition from efficacy validation to mechanistic dissection. ID-side trends expanded in parallel towards metastasis-related and gene-regulatory neighborhoods, suggesting widening downstream enquiry. Using overlay labeling, we classified the cell-cycle/DDR/cell-death modules as specific and the metabolism/inflammation

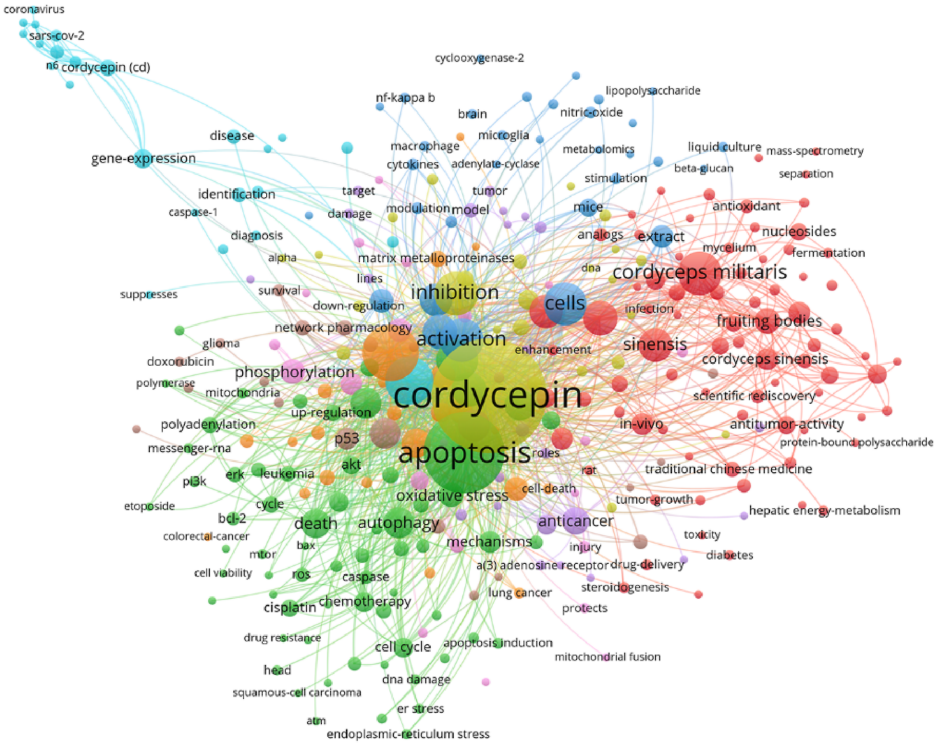

Fig. 5. Keyword co-occurrence network (DE-based; full counting, association-strength normalization).

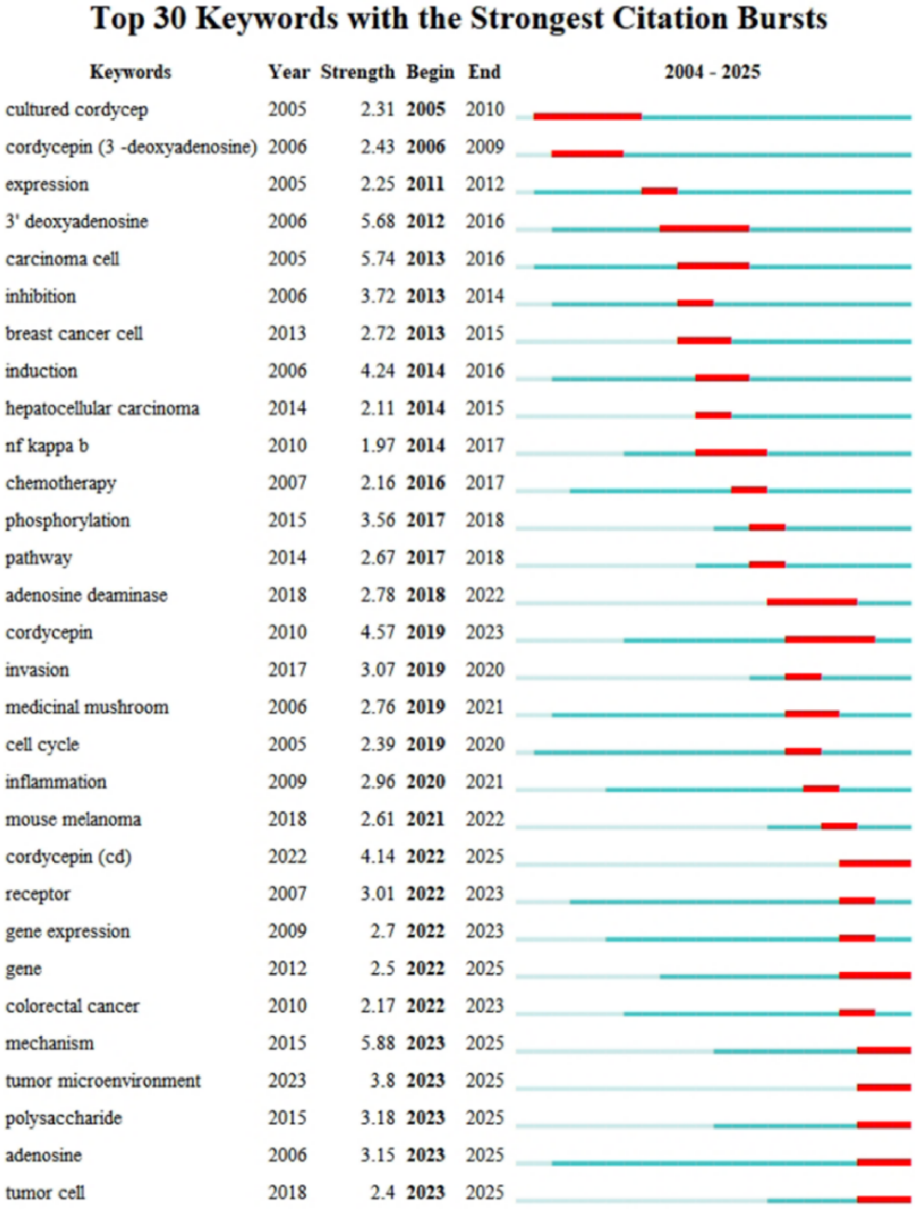

Fig. 6. Top-30 burst keywords (2004–2025).

neighborhoods on the ID side as generic. Together they outline a checkpoint/DDR → arrest → apoptosis/autophagy axis.

3.5.4. Cluster evolution with DE/ID context

Log-likelihood-ratio (LLR) clustering and timeline views resolved four periods- (2004–2009, 2010–2014, 2015–2019, and 2020–2025) (Fig. 7).

Segment 1 (2004–2009): 31 articles were clustered into 5 groups, with the largest being #0 "sinensis mycelium," followed by #1 "cell cycle," #2 "leukemia," and #3 "B16-B16 mouse melanoma cells" (Fig. 7A).

Segment 2 (2010–2014): 67 articles yielded 6 clusters, led by #0 "apoptosis," followed by #1 "*Cordyceps militaris*," #2 "expression," and #3 "synergistic" (Fig. 7B).

Segment 3 (2015–2019): 155 articles produced 6 clusters, with #0 "cell" as the largest, followed by #1 "*Cordyceps militaris*," #2 "cell migration/invasion," and #3 "ROS" (Fig. 7C).

Segment 4 (2020–2025): 191 articles were clustered into 7 groups. Cluster #0 remained "*Cordyceps militaris*," while #1 was again "cell cycle," followed by #2 "medicinal mushrooms" and #3 "cordyceps" (Fig. 7D).

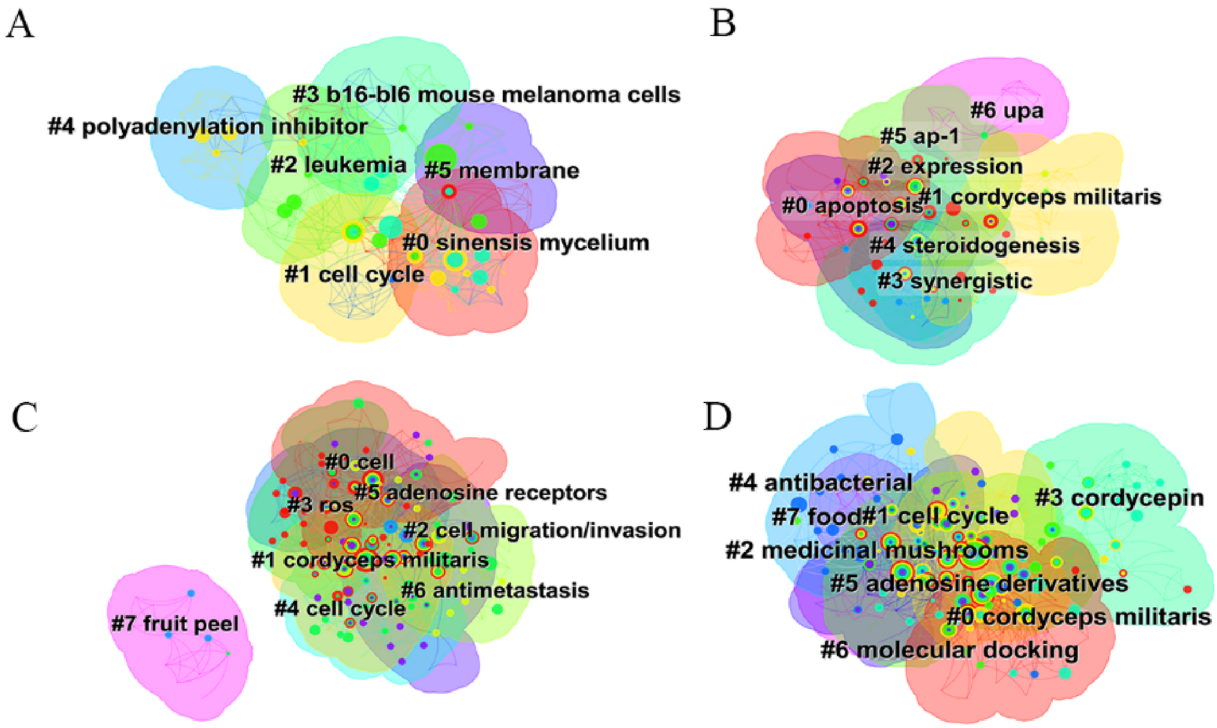

Fig. 7. Keyword clustering segmentation. A. 2004–2009, B. 2010–2014, C. 2015–2019, D. 2020–2025.

Across all periods, "cell cycle" and "*Cordyceps militaris*" consistently appeared as dominant clusters. Meanwhile, emerging clusters such as #2 "cell migration/invasion," #6 "antimetastasis," #3 "ROS," "UPA," and "expression" indicate expanding interest in metastasis-related mechanisms and downstream gene regulation.

4. Discussion

This review offers methods-transparent integration and organization of the literature, rather than experimental discovery. These perspectives move beyond volume counts to reveal a checkpoint/DNA-damage-response (DDR)-biased mechanistic emphasis and the network structure underpinning topic evolution in cordycepin oncology.

This review employed bibliometric methods to comprehensively summarize research on the antitumor effects of cordycepin over the past two decades since 2004. The earliest relevant publication in our dataset explored the role of the human metastasis inhibitor NM23-H1 in DNA repair and tumor suppression, suggesting a potential mechanistic link to cordycepin's inhibition of 3′-end exonuclease activity (Ma et al., 2004) [39]. The publication trend over the years can be divided into two distinct phases. The first phase, from 2004 to 2013, was characterized by a slow publication rate, with fewer than 10 articles published annually. The second phase, from 2014 to 2025, saw a substantial increase, averaging over 20 publications per year. This growth reflects heightened global interest in cordycepin's antitumor potential, likely driven by expanding awareness of Cordyceps, particularly *Cordyceps militaris*, as both a medicinal and dietary resource in Asian cultures [40]. Notably, Asian countries—especially China—have led in publication volume. Among the top ten publishing countries, seven are located in East or Southeast Asia. China alone accounted for 58.39% of total publications, underscoring its dominant role. This prominence is closely tied to the traditional use of Cordyceps (Dong Chong Xia Cao) in Traditional Chinese Medicine (TCM), where cordycepin, a principal bioactive compound, has been applied clinically for centuries. Consequently, institutions such as China Medical University have become prominent contributors to this research field [41,42]. Keyword burst and clustering analyses revealed that studies on cordycepin's regulation of tumor cell cycles have been persistent research focal points. Additionally, the regulation of gene expression has emerged as a growing area of interest. Based on these bibliometric insights, this review further discusses three core research directions: (1) mechanisms by which cordycepin induces apoptosis via cell cycle regulation, (2) cordycepin's role in

inhibiting tumor metastasis, and (3) its impact on gene expression. These directions are not only current research hotspots but also foundational to advancing therapeutic development.

4.1. Mechanistic studies of cordycepin's antitumor effects via cell cycle regulation

G1/S checkpoint (Cyclin D–CDK4/6, p21/p27, p53). Converging evidence indicates that cordycepin down-modulates Cyclin D–CDK4/6 while up-regulating p21/p27, often within a p53-competent context, thereby restraining G1/S transit via reduced E2F engagement. This matches the DE prominence of "cell cycle," "p21/p27," and "G1/S arrest," and the post-2015 burst intensification (Figs. 4 and 6). The cell cycle is a fundamental process controlling cellular proliferation. Its dysregulation contributes to unchecked cell growth and tumorigenesis [43]. It comprises four phases (G1, S, G2, and M), orchestrated by cyclins and cyclin-dependent kinases (CDKs) [44–46]. Because tumor cells are relatively insensitive to checkpoint regulation, they often display continuous proliferation and accelerated cycling; targeting the cycle therefore remains an important anticancer strategy [47–49].

S-phase replication stress and ATR/CHK1 signaling. Reports of γ-H2AX accumulation and ATR/CHK1 activation suggest that cordycepin elicits replication stress and checkpoint engagement, producing cytostasis and apoptosis priming [50]. This aligns with DE terms ("DNA damage," "CHK1") and ID bridges to genome-maintenance pathways (Figs. 4 and 5). Cordycepin has been shown to induce cell cycle arrest at G1/S and G2/M transitions. It achieves this by downregulating positive regulators (e.g., Cyclin D1, CDKs) and upregulating inhibitory proteins (e.g., p21, p27). Furthermore, cordycepin blocks Rb phosphorylation, thereby preventing E2F activation and subsequent DNA synthesis [51–54]. For example, in HeLa cells, cordycepin reduced CDK2 and Cyclin E expression while increasing p21 levels, leading to G1 arrest [54,55]. For completeness, we also note that cordycepin has been shown to suppress proliferative ERK signaling in tumor models (e.g., Leydig tumor cells), which provides a complementary route converging on checkpoint control and cytostasis [56].

G2/M transition (CDK1–Cyclin B1, CDC25C). Cordycepin frequently enforces a G2/M blockade via suppression of CDK1–Cyclin B1 and/or CDC25C modulation, complementing S-phase stress to curtail mitotic entry (e.g., esophageal and other solid tumors). The persistence of "G2/M arrest" in DE and across timeline clusters indicates a durable mechanistic core (Figs. 4 and 7). Cordycepin also induces G2/M arrest by suppressing CDK1 (CDC2)/Cyclin B1 activity, observed in colorectal, breast, and lung cancer models [57,58]. It enhances DNA damage responses via CHK1/CHK2 activation, contributing to delayed cell cycle progression [50,59]. Some studies further implicate p53-dependent pathways, with stronger effects noted in p53 wild-type cell lines [60–62].

In summary, cordycepin exerts anti-tumor activity by intervening in the tumor cell cycle through multiple targets and pathways, providing a mechanistic foundation for -cycle-regulating therapeutics- This G1/S–S-phase stress–G2/M sequence coheres with the DE prominence of "cell cycle/G1/S/G2/M/p21/p27/CHK1" and their post-2015 intensification (Figs. 4, 6 and 7). Importantly, this integrated cascade is corroborated jointly by DE/ID stratification and burst/timeline analyses rather than single-study inference (Figs. 4, 6 and 7).

4.2. Advances in understanding cordycepin-mediated gene expression regulation

Additional evidence indicates that as a 3′-deoxyadenosine, cordycepin shortens poly(A) tails and impairs 3′-end processing/termination, implying direct post-transcriptional interference that may cooperate with the miRNA/EMT networks herein [63,64]. Beyond cell cycle control, recent research increasingly highlights cordycepin's role in modulating gene expression, including transcription factors, non-coding RNAs, and signaling pathway remodeling.

Cordycepin has been shown to suppress oncogene expression. For instance, in HCT116 and Caco-2 colorectal cancer cells, it elevated miR-26a levels, thereby downregulating MYC mRNA and protein, which inhibited proliferation [65]. Similarly, in triple-negative breast cancer, cordycepin suppressed EMT-related transcription factors (SLUG, TWIST1, SNAIL1, ZEB1), reversing mesenchymal traits and limiting metastasis [66]. Cordycepin also influences non-coding RNAs, particularly miRNAs. Zhang et al. reported increased miR-33b levels in A375 and Lu1205 melanoma cells, leading to downregulation of HMGA2, Twist1, and ZEB1, ultimately inhibiting invasion [67].

In signaling regulation, cordycepin suppressed MEK/ERK phosphorylation and reduced Cyclin B1 and CDK1 in ECA109 and TE-1 cells, promoting G2/M arrest and apoptosis [57]. It also downregulated ERO1A via inhibition of the ERO1A/mTOR/SREBP1 axis in cholangiocarcinoma cells

[68]. High-throughput studies further support cordycepin's broad regulatory impact. RNA-seq analyses revealed altered expression of genes involved in vitamin D metabolism, lipid transport, and protein catabolism in SCLC zebrafish xenografts [69]. In AML stem cell models, cordycepin reactivated WIF1 and DKK1 while suppressing MYC and PROM1 (CD133), reducing leukemic cell viability [70].

Collectively, cordycepin modulates gene expression via multiple pathways and targets, providing a foundation for future drug development. These mechanisms align with the ID-level reinforcement of the "cell migration/invasion" and "antimetastasis" clusters (Figs. 5–7).

4.3. Mechanisms of cordycepin in inhibiting tumor migration and metastasis

Bibliometric data also identified tumor metastasis as an emerging focus, with clusters like "cell migration/invasion" and "antimetastasis" gaining prominence. Thus, this review addresses the mechanisms through which cordycepin impedes cancer cell metastasis.

Metastasis remains a major barrier in cancer treatment. Cordycepin has demonstrated antimetastatic effects by reversing epithelial-mesenchymal transition (EMT), inhibiting matrix metalloproteinases (MMPs), and disrupting cytoskeletal remodeling.

Cordycepin restored epithelial traits in BT549 and 4T1 breast cancer cells by upregulating E-cadherin and downregulating N-cadherin, SLUG, and TWIST, thereby reducing cell motility [66].

It also inhibited TPA-induced MMP-9 via AP-1 suppression, stabilizing the extracellular matrix and enhancing TIMP1/2 secretion [71,72].

Additionally, cordycepin modulated cytoskeletal signaling through the miR-33b-HMGA2/Twist1 axis, affecting RhoA, FAK, and Src activity, and impeding pseudopod formation [67]. In vivo evidence supports these findings. In MDA-MB-231 xenografts, cordycepin suppressed brain metastasis by inhibiting Hedgehog-GLI signaling [12]. In cholangiocarcinoma, it reduced ERO1A and mTOR, downregulating SREBP1 and EMT markers [68].

These results suggest that cordycepin targets multiple aspects of the metastatic cascade, positioning it as a candidate anti-metastasis agent. Concurrently, the DE/ID stratification indicates that "gene expression" is both a high-frequency DE focus and an ID bridge to broader regulatory neighborhoods (Figs. 4 and 5).

4.4. Comparative positioning among natural small molecules

Relative to curcumin and resveratrol—whose mainstream reviews depict predominantly apoptosis-centric programmed cell death, with autophagy/necroptosis as adjuncts—cordycepin more often exhibits a checkpoint/DDR-biased cascade: G1/S or G2/M arrest → ATR/CHK1–ATM/CHK2 engagement → γ-H2AX accumulation → apoptosis/autophagy. Given scaffold differences (nucleoside vs polyphenols), cordycepin's sites of action partially converge with, yet are not redundant to, those of polyphenols, implying complementarity rather than simple overlap in combinations. This conceptual comparison accords with the boundaries suggested by our DE/ID-stratified hotspots [73–75].

4.5. Limitations

Our inferences are constrained by single-database sourcing (WoSCC), potential language/publication biases, and semantic noise intrinsic to DE vs ID generation. Network topology and burst detection depend on parameterization (e.g., thresholds, g-index, pruning), although sensitivity checks indicated qualitatively stable structures (Methods 2.5). Address normalization may introduce minor noise despite rule-based standardization and spot validation. These caveats do not overturn the central axes consolidated here (cell-cycle—DDR—transcription—metastasis) but should temper interpretation. We also acknowledge that we did not run an independent bibliometrix/R-package validation, which could be explored in future work.

4.6. Research gaps and future perspectives

Despite significant progress, several limitations remain in the current body of research. First, most studies rely on in vitro assays and xenograft models, lacking comprehensive preclinical pharmacokinetic data. Parameters such as solubility, bioavailability, and metabolism in vivo remain poorly defined.

Second, mechanistic investigations are often limited to isolated pathways (e.g., PI3K/Akt, MAPK), with insufficient focus on integrated signaling networks, tumor microenvironments, and cellular heterogeneity. Research into non-coding RNAs, epigenetics, and immunomodulation is still in early stages.

Third, efficacy across tumor types remains unclear. While promising in breast, lung, and liver cancers, cordycepin's effects in other solid tumors

and hematological malignancies require further exploration.

Finally, limited studies have examined the synergy between cordycepin and conventional therapies. Although some reports suggest enhanced effects when combined with agents like cisplatin or doxorubicin, detailed evaluations of synergy, dosing, and safety are lacking [76]. Furthermore, its impact on drug-resistant tumor cells remains understudied. Structure–activity optimization has also begun to yield cordycepin derivatives (e.g., unsaturated fatty-acid conjugates) with improved stability or bioactivity profiles, warranting systematic SAR and PK evaluation.

Because ENT1/ENT2-mediated uptake and rapid ADA-catalyzed deamination jointly determine intracellular exposure, these processes likely modulate the magnitude of downstream DDR/checkpoint phenotypes; accordingly, ADA inhibition, prodrug design, and delivery strategies are mechanistically justified. Addressing these gaps will be critical for translating cordycepin into a clinically viable anticancer agent. Notably, cordycepin is rapidly deaminated by adenosine deaminase to 3′-deoxyinosine; ADA inhibition (e.g., pentostatin), prodrug strategies, and nano-delivery are active avenues to improve exposure [77,78].

Looking forward, the DE/ID-stratified trends suggest two tractable frontiers: (i) checkpoint-centric combinations (e.g., cordycepin with CDK inhibitors or ATR/CHK1 modulators) to solidify cytostasis-to-apoptosis conversion, and (ii) delivery/prodrug strategies co-targeting ADA-mediated deamination to extend exposure—both of which are testable in multi-omic pharmacology frameworks.

5. Conclusion

Beyond simple volume trends, our value-add lies in DE/ID-stratified hotspot mapping and corridor-style collaboration topology, which jointly reveal a checkpoint/DDR–centric signature aligned with a few high-traffic international links (Figs. 4–7). Our bibliometric analysis reveals that research interest in cordycepin for anticancer therapy continues to grow, particularly in cell cycle regulation and gene expression modulation. These areas have emerged as significant research hotspots. Keyword clustering and emergence analyses indicate that high-frequency terms such as “cell cycle,” “gene expression,” “apoptosis,” and “PI3K/Akt pathway” reflect concentrated thematic efforts and a clear trend toward deeper mechanistic exploration.

Building on these findings, this review synthesized recent mechanistic advances in cordycepin-related anticancer research. Cordycepin inhibits tumor cell cycle progression via multiple mechanisms, including modulation of the cyclin/CDK system, activation of checkpoint pathways, and induction of *p53* and *p21* expression. It has demonstrated significant antiproliferative activity across a variety of cancer types. Additionally, its regulatory effects on gene expression have attracted increasing attention. Some studies suggest cordycepin influences multiple signaling pathways and may also impact epigenetic regulation, indicating broader molecular targets.

The themes identified in this narrative review align closely with those uncovered through bibliometric analyses, underscoring the relevance and continuity between quantitative data and evolving research focus. Overall, cordycepin—an active natural compound with notable antitumor potential—displays multi-targeted, network-based, and mechanistically intricate pharmacological actions. Future research should aim to expand molecular mechanistic studies, enhance in vivo experimental evidence, and integrate multi-omics with systems pharmacology approaches to better characterize cordycepin's global action network and evaluate its clinical translational potential in precision oncology.

Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Zhiwei Ouyang: Conceptualization, Investigation, Writing − original draft, Writing − review & editing. Yufei Zhang: Conceptualization. Jianghan Ning: Project administration. Yayi Tu: Conceptualization, Supervision. Bin He: Conceptualization, Writing - review & editing, Supervision.

Acknowledgements

This research was supported by the National Natural Science Foundation of China (No. 32260017), Jiangxi Provincial Natural Science Foundation (No. 20242BAB25334) and Youth Talent Support Program of Jiangxi Science & Technology Normal University (No. 2022QNBJRC005), Graduate Innovation Special Fund Project of Jiangxi Provincial Department of Education (YC2024-X23).

References

[1] Cunningham KG, Manson W, Spring FS, Hutchinson SA. Cordycepin, a metabolic product isolated from cultures of Cordyceps militaris (Linn.) link. Nature 1950;166:949.

[2] Das G, Shin HS, Leyva-Gómez G, Prado-Audelo MLD, Cortes H, Singh YD, et al. Cordyceps spp.: a review on its immune-stimulatory and other biological potentials. Front Pharmacol 2020;11:602364.

[3] Liu X, Zhao S, Tan L, Tan Y, Wang Y, Ye Z, et al. Frontier and hot topics in electrochemiluminescence sensing technology based on CiteSpace bibliometric analysis. Biosens Bioelectron 2022;201:113932.

[4] Luo C, Xu X, Wei X, Feng W, Huang H, Liu H, et al. Natural medicines for the treatment of fatigue: bioactive components, pharmacology, and mechanisms. Pharmacol Res 2019;148:104409.

[5] Songprakhon P, Panya A, Choomee K, Limjindaporn T, Noisakran S, Tarasuk M, et al. Cordycepin exhibits both antiviral and anti-inflammatory effects against dengue virus infection. iScience 2024;27:110711.

[6] Tuli HS, Sharma AK, Sandhu SS, Kashyap D. Cordycepin: a bioactive metabolite with therapeutic potential. Life Sci 2013;93:863–9.

[7] Wang Z, Chen Z, Jiang Z, Luo P, Liu L, Huang Y, et al. Cordycepin prevents radiation ulcer by inhibiting cell senescence via NRF2 and AMPK in rodents. Nat Commun 2019;10:2538.

[8] Wei P, Wang K, Luo C, Huang Y, Misilimu D, Wen H, et al. Cordycepin confers long-term neuroprotection via inhibiting neutrophil infiltration and neuroinflammation after traumatic brain injury. J Neuroinflammation 2021;18:137.

[9] Zhang Y, Yu W, Yuan D, Ma W, Tu Y, He B. Epigenetic therapy for leukemias: a comprehensive review from biological markers to practice. Biotechnol Adv 2025;83:108626.

[10] Yu W, Wei X, Wu Y, Jiang C, Tu Y, He B. Microbial biosynthesis of nucleos(t)ide analogs: applications, and engineering optimization. Crit Rev Microbiol 2024:1–19.

[11] Krishna KV, Ulhas RS, Malaviya A. Bioactive compounds from cordyceps and their therapeutic potential. Crit Rev Biotechnol 2024;44:753–73.

[12] Liu C, Qi M, Li L, Yuan Y, Wu X, Fu J. Natural cordycepin induces apoptosis and suppresses metastasis in breast cancer cells by inhibiting the Hedgehog pathway. Food Funct 2020;11:2107–16.

[13] Wei C, Yao X, Jiang Z, Wang Y, Zhang D, Chen X, et al. Cordycepin inhibits drug-resistance non-small cell lung cancer progression by activating AMPK signaling pathway. Pharmacol Res 2019;144:79–89.

[14] Guo Z, Chen W, Dai G, Huang Y. Cordycepin suppresses the migration and invasion of human liver cancer cells by downregulating the expression of CXCR4. Int J Mol Med 2020;45:141–50.

[15] Feng C, Chen R, Gao X, Fang W, Wu S, Chen L, et al. Cordycepin enhances the Anticancer efficacy of PD-L1 blockade by modulating the tumor microenvironment of colon cancer. Eur J Pharmacol 2024;985:177089.

[16] Lee EJ, Kim WJ, Moon SK. Cordycepin suppresses TNF-alpha-induced invasion, migration and matrix metalloproteinase-9 expression in human bladder cancer cells. Phytother Res: PTR 2010;24:1755–61.

[17] Ho SY, Wu WS, Lin LC, Wu YH, Chiu HW, Yeh YL, et al. Cordycepin enhances radiosensitivity in oral squamous carcinoma cells by inducing autophagy and apoptosis through cell cycle arrest. Int J Mol Sci 2019;20.

[18] Wang Y, Lv Y, Liu TS, Yan WD, Chen LY, Li ZH, et al. Cordycepin suppresses cell proliferation and migration by targeting CLEC2 in human gastric cancer cells via Akt signaling pathway. Life Sci 2019;223:110–9.

[19] Phan TG, Croucher PI. The dormant cancer cell life cycle. Nat Rev Cancer 2020;20:398–411.

[20] He W, Zhang MF, Ye J, Jiang TT, Fang X, Song Y. Cordycepin induces apoptosis by enhancing JNK and p38 kinase activity and increasing the protein expression of Bcl-2 pro-apoptotic molecules. J Zhejiang Univ - Sci B 2010;11:654–60.

[21] Li XY, Tao H, Jin C, Du ZY, Liao WF, Tang QJ, et al. Cordycepin inhibits pancreatic cancer cell growth in vitro and in vivo via targeting FGFR2 and blocking ERK signaling. Chin J Nat Med 2020;18:345–55.

[22] Jung SM, Park SS, Kim WJ, Moon SK. Ras/ERK1 pathway regulation of p27KIP1-mediated G1-phase cell-cycle arrest in cordycepin-induced inhibition of the proliferation of vascular smooth muscle cells. Eur J Pharmacol 2012;681:15–22.

[23] Chen Y, Yang SH, Hueng DY, Syu JP, Liao CC, Wu YC. Cordycepin induces apoptosis of C6 glioma cells through the adenosine 2A receptor-p53-caspase-7-PARP pathway. Chem Biol Interact 2014;216:17–25.

[24] Khan MA, Tania M. Cordycepin and kinase inhibition in cancer. Drug Discov Today 2023;28:103481.

[25] de Winter JCF, Zadpoor AA, Dodou D. The expansion of Google Scholar versus Web of Science: a longitudinal study. Scientometrics 2014;98:1547–65.

[26] Jiang S, Liu Y, Zheng H, Zhang L, Zhao H, Sang X, et al. Evolutionary patterns and research frontiers in neoadjuvant immunotherapy: a bibliometric analysis. Int J Surg 2023;109:2774–83.

[27] van Eck NJ, Waltman L. Software survey: VOSviewer, a computer program for bibliometric mapping. Scientometrics 2010;84:523–38.

[28] Synnestvedt MB, Chen C, Holmes JH. CiteSpace II: visualization and knowledge discovery in bibliographic databases. In: AMIA Annual Symposium proceedings AMIA Symposium. 2005; 2005. p. 724–8.

[29] van Eck NJ, Waltman L. Citation-based clustering of publications using CitNetExplorer and VOSviewer. Scientometrics 2017;111:1053–70.

[30] van Eck NJ, Waltman L. Visualizing bibliometric networks. In: Ding Y, Rousseau R, Wolfram D, editors. Measuring scholarly impact: methods and practice. Cham: Springer International Publishing; 2014. p. 285–320.

[31] Lee P-C, Su H-N. Investigating the structure of regional innovation system research through keyword co-occurrence and social network analysis. Innovation 2010;12:26–40.

[32] Bordons M, Aparicio J, González-Albo B, Díaz-Faes AA. The relationship between the research performance of scientists and their position in co-authorship networks in three fields. J Informetr 2015;9:135–44.

[33] Kleinberg J. Bursty and hierarchical structure in streams. Data Min Knowl Discov 2003;7:373–97.

[34] Chen C. CiteSpace II: detecting and visualizing emerging trends and transient patterns in scientific literature. J Am Soc Inf Sci Technol 2006;57:359–77.

[35] Chen C, Chen Y. Searching for clinical evidence in CiteSpace. 2005. p. 121–5.

[36] Chen CM. The CiteSpace manual. 2014.

[37] Katz JS, Martin BR. What is research collaboration? Res Pol 1997;26:1–18.

[38] Beaver Dd, Rosen R. Studies in scientific collaboration. Scientometrics 1978;1:65–84.

[39] Ma DQ, McCorkle JR, Kaetzel DM. The metastasis suppressor NM23-H1 possesses 3′-5′ exonuclease activity. J Biol Chem 2004;279:18073–84.

[40] Zeng J, Zhou Y, Lyu M, Huang X, Xie M, Huang M, et al. Cordyceps militaris: a novel mushroom platform for metabolic engineering. Biotechnol Adv 2024;74:108396.

[41] Chen YC. Chinese values, health and nursing. J Adv Nurs 2001;36:270–3.

[42] Yu W, Ma M, Chen X, Min J, Li L, Zheng Y, et al. Traditional Chinese medicine and constitutional medicine in China, Japan and Korea: a comparative study. Am J Chin Med 2017;45:1–12.

[43] Liu J, Peng Y, Wei W. Cell cycle on the crossroad of tumorigenesis and cancer therapy. Trends Cell Biol 2022;32: 30–44.
[44] Schafer KA. The cell cycle: a review. Vet Pathol 1998;35: 461–78.
[45] Bertoli C, Skotheim JM, de Bruin RA. Control of cell cycle transcription during G1 and S phases. Nat Rev Mol Cell Biol 2013;14:518–28.
[46] Dalton S. Linking the cell cycle to cell fate decisions. Trends Cell Biol 2015;25:592–600.
[47] Evan GI, Vousden KH. Proliferation, cell cycle and apoptosis in cancer. Nature 2001;411:342–8.
[48] Icard P, Fournel L, Wu Z, Alifano M, Lincet H. Interconnection between metabolism and cell cycle in cancer. Trends Biochem Sci 2019;44:490–501.
[49] Xu Y, Jiao Y, Liu C, Miao R, Liu C, Wang Y, et al. R-loop and diseases: the cell cycle matters. Mol Cancer 2024;23:84.
[50] Lee YP, Lin CR, Chen SS, Chen RJ, Wu YH, Chen YH, et al. Combination treatment of cordycepin and radiation induces MA-10 mouse Leydig tumor cell death via ROS accumulation and DNA damage. Am J Cancer Res 2023;13: 1329–46.
[51] Min Y, Ding Y, Huang Q, Xu Y, Li J. Cordycepin inhibited the retinoblastoma cell proliferation, migration, and invasion as well as lung metastasis via modulating c-Myc/cyclin D1 pathway. Chem Biol Drug Des 2023;101:605–13.
[52] Yoshikawa N, Yamada S, Takeuchi C, Kagota S, Shinozuka K, Kunitomo M, et al. Cordycepin (3′-deoxyadenosine) inhibits the growth of B16-BL6 mouse melanoma cells through the stimulation of adenosine A3 receptor followed by glycogen synthase kinase-3beta activation and cyclin D1 suppression. In: Naunyn-Schmiedeberg's archives of pharmacology. 377; 2008. p. 591–5.
[53] Wang XA, Xiang SS, Li HF, Wu XS, Li ML, Shu YJ, et al. Cordycepin induces S phase arrest and apoptosis in human gallbladder cancer cells. Molecules 2014;19: 11350–65.
[54] Tania M, Shawon J, Saif K, Kiefer R, Khorram MS, Halim MA, et al. Cordycepin downregulates Cdk-2 to interfere with cell cycle and increases apoptosis by generating ROS in cervical cancer cells: in vitro and in silico study. Curr Cancer Drug Targets 2019;19:152–9.
[55] Qu S, Wang Q, Wang Y, Li L, Zhu L, Kuang X, et al. Design, synthesis, antibacterial/antitumor activity and in vitro stability of novel cordycepin derivatives with unsaturated fatty acid chain. In: European journal of pharmaceutical sciences: official journal of the European Federation for Pharmaceutical Sciences. 187; 2023. p. 106466.
[56] Chen LC, Chen CY, Lee YP, Huang BM. Cordycepin inhibits ERK pathway to suppress FGF9-induced tumorigenesis with MA-10 mouse Leydig tumor cells. J Food Drug Anal 2023;31:485–501.
[57] Xu JC, Zhou XP, Wang XA, Xu MD, Chen T, Chen TY, et al. Cordycepin induces apoptosis and G2/M phase arrest through the ERK pathways in esophageal cancer cells. J Cancer 2019;10:2415–24.
[58] Lee SJ, Moon GS, Jung KH, Kim WJ, Moon SK. c-Jun N-terminal kinase 1 is required for cordycepin-mediated induction of G2/M cell-cycle arrest via p21WAF1 expression in human colon cancer cells. In: Food and chemical toxicology: an international journal published for the British Industrial Biological Research Association. 48; 2010. p. 277–83.
[59] Su NW, Wu SH, Chi CW, Tsai TH, Chen YJ. Cordycepin, isolated from medicinal fungus Cordyceps sinensis, enhances radiosensitivity of oral cancer associated with modulation of DNA damage repair. In: Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association. 124; 2019. p. 400–10.
[60] Lee MJ, Lee JC, Hsieh JH, Lin MY, Shih IA, You HL, et al. Cordycepin inhibits the proliferation of malignant peripheral nerve sheath tumor cells through the p53/Sp1/tubulin pathway. Am J Cancer Res 2021;11:1247–66.
[61] Seong da B, Hong S, Muthusami S, Kim WD, Yu JR, Park WY. Cordycepin increases radiosensitivity in cervical cancer cells by overriding or prolonging radiation-induced G2/M arrest. Eur J Pharmacol 2016;771:77–83.
[62] Chen LS, Stellrecht CM, Gandhi V. RNA-directed agent, cordycepin, induces cell death in multiple myeloma cells. Br J Haematol 2008;140. 682-391.
[63] Kondrashov A, Meijer HA, Barthet-Barateig A, Parker HN, Khurshid A, Tessier S, et al. Inhibition of polyadenylation reduces inflammatory gene induction. RNA (New York, NY) 2012;18:2236–50.
[64] Wong YY, Moon A, Duffin R, Barthet-Barateig A, Meijer HA, Clemens MJ, et al. Cordycepin inhibits protein synthesis and cell adhesion through effects on signal transduction. J Biol Chem 2010;285:2610–21.
[65] Zhang Z, Li K, Zheng Z, Liu Y. Cordycepin inhibits colon cancer proliferation by suppressing MYC expression. BMC Pharmacol Toxicol 2022;23:12.
[66] Wei C, Khan MA, Du J, Cheng J, Tania M, Leung EL, et al. Cordycepin inhibits triple-negative breast cancer cell migration and invasion by regulating EMT-TFs SLUG, TWIST1, SNAIL1, and ZEB1. Front Oncol 2022;12:898583.
[67] Zhang P, Huang C, Fu C, Tian Y, Hu Y, Wang B, et al. Cordycepin (3′-deoxyadenosine) suppressed HMGA2, Twist1 and ZEB1-dependent melanoma invasion and metastasis by targeting miR-33b. Oncotarget 2015;6:9834–53.
[68] Zhou X, Li Y, Yang C, Chen D, Wang T, Liu T, et al. Cordycepin reprogramming lipid metabolism to block metastasis and EMT via ERO1A/mTOR/SREBP1 axis in cholangiocarcinoma. Life Sci 2023;327:121698.
[69] Zhang SR, Pan M, Gao YB, Fan RY, Bin XN, Qian ST, et al. Efficacy and mechanism study of cordycepin against brain metastases of small cell lung cancer based on zebrafish. Phytomedicine: Intern J Phytother Phytopharmacol 2023; 109:154613.
[70] Abazari N, Stefanucci MR, Bossi LE, Trojani A, Cairoli R, Beghini A. Cordycepin (3′dA) induces cell death of AC133 (+) leukemia cells via Re-Expression of WIF1 and down-modulation of MYC. Cancers 2023;15.
[71] Noh EM, Youn HJ, Jung SH, Han JH, Jeong YJ, Chung EY, et al. Cordycepin inhibits TPA-induced matrix metalloproteinase-9 expression by suppressing the MAPK/AP-1 pathway in MCF-7 human breast cancer cells. Int J Mol Med 2010;25:255–60.
[72] Nakamura K, Shinozuka K, Yoshikawa N. Anticancer and antimetastatic effects of cordycepin, an active component of Cordyceps sinensis. J Pharmacol Sci 2015;127:53–6.
[73] Yu C, Yang B, Najafi M. Targeting of cancer cell death mechanisms by curcumin: implications to cancer therapy. Basic Clin Pharmacol Toxicol 2021;129:397–415.
[74] Costantino M, Corno C, Colombo D, Perego P. Curcumin and related compounds in cancer cells: new avenues for old molecules. Front Pharmacol 2022;13:889816.
[75] Jang JY, Im E, Kim ND. Mechanism of resveratrol-induced programmed cell death and new drug discovery against cancer: a review. Int J Mol Sci 2022;23.
[76] Wang C-W, Lee B-H, Tai C-J. The inhibition of cordycepin on cancer stemness in TGF-beta induced chemo-resistant ovarian cancer cell. 2017. p. 8.
[77] Zhang J, Yang Z, Zhao Z, Zhang N. Structural and pharmacological insights into cordycepin for neoplasms and metabolic disorders. Front Pharmacol 2024;15:1367820.
[78] Lee JB, Radhi M, Cipolla E, Gandhi RD, Sarmad S, Zgair A, et al. A novel nucleoside rescue metabolic pathway may be responsible for therapeutic effect of orally administered cordycepin. Sci Rep 2019;9:15760.