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About this journal  $\vee$ 

Articles V

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Journal updates

#### Overview

Cellular and Molecular Life Sciences (CMLS) is a multidisciplinary Open Access journal covering the latest aspects of biological and biomedical research.

### Artificial Intelligence (AI)

Springer Nature is monitoring ongoing developments in this area closely and will review (and update) these policies as appropriate.

- 1. Al authorship
- 2. Generative AI images
- 3. Al use by peer reviewers

#### **AI Authorship**

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#### **Journal metrics**



Journal Impact Factor 6.2 (2024)

#### **Editorial Policies**

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### Frontiers in Cell and Developmental Biology

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### Artificial intelligence

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#### For authors

Why submit?

Article types

#### Author guidelines

Editor guidelines

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Submission checklist

Contact editorial office

# **Article Review**

An article review is a critical evaluation summarizing the main points, strengths, and weaknesses of a scholarly article.

### **Types:**

- Critical Review
- Literature Review
- Systematic Review
- Narrative Review
- Theoretical Review
- Integrative Review
- Comparative Review
- Scoping Review



#### **Article Review**

Title: The Impact of Climate Change on Global Agriculture

Author: Jane Smith

Journal: Journal of Environmental Studies, Vol. 25, Issue 4, 2023

#### Introduction

Jane Smith's article, "The Impact of Climate Change on Global Agriculture," examines how climate change affects agricultural practices worldwide, focusing on temperature changes, precipitation patterns, and extreme weather events.

#### Summary

Smith discusses the impact of climate change on food security, presenting data on crop yield shifts and agricultural productivity. She uses global case studies to illustrate these effects.

#### Analysis

The article is well-researched, featuring both qualitative and quantitative data. While the global perspective is strong, the discussion on adaptive strategies is limited.

#### Strengths

- Comprehensive analysis
- Diverse case studies
- Clear writing

#### Weaknesses

- Limited focus on adaptation
- Underrepresentation of some regions

#### Conclusion

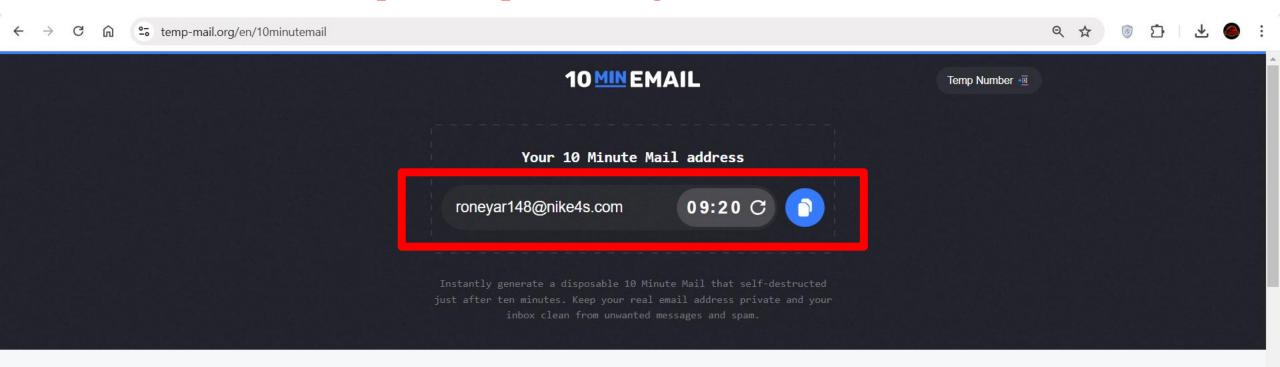
Smith's article highlights the urgent need for action to mitigate climate change's effects on agriculture. Future research should emphasize adaptive strategies and regional analysis.

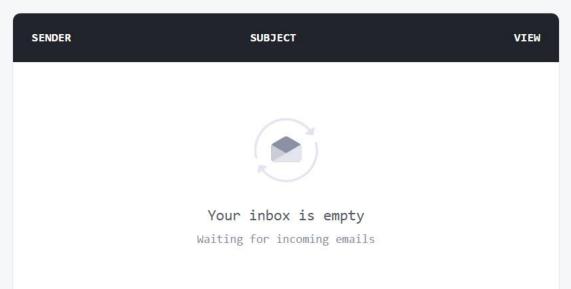
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Smith, J. (2023). The Impact of Climate Change on Global Agriculture. Journal of Environmental Studies, 25(4), 123-145.



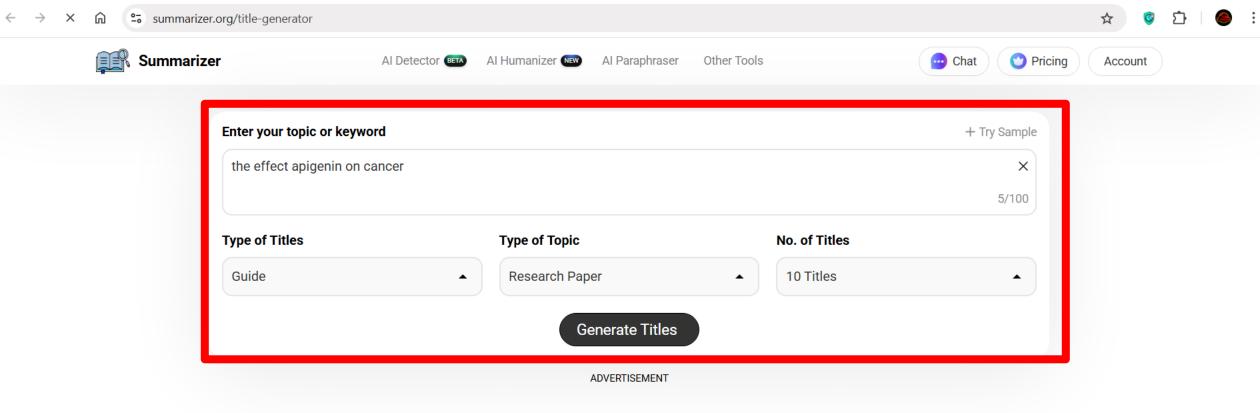
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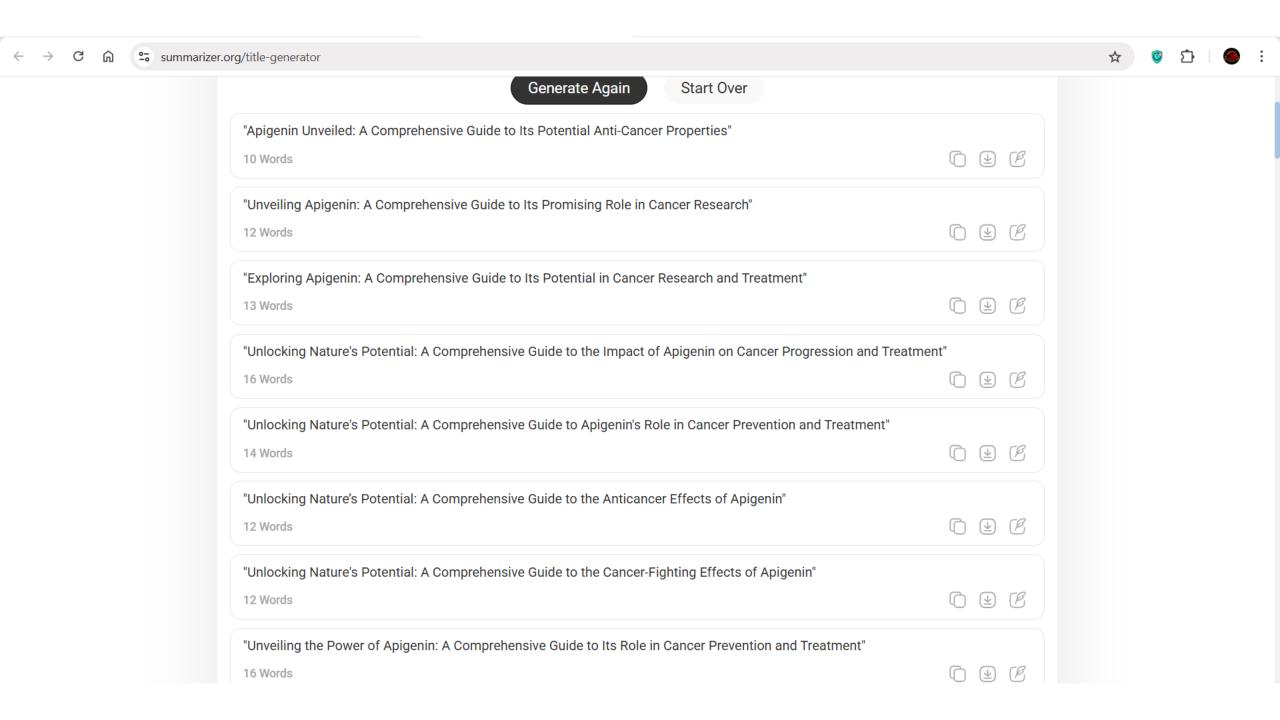




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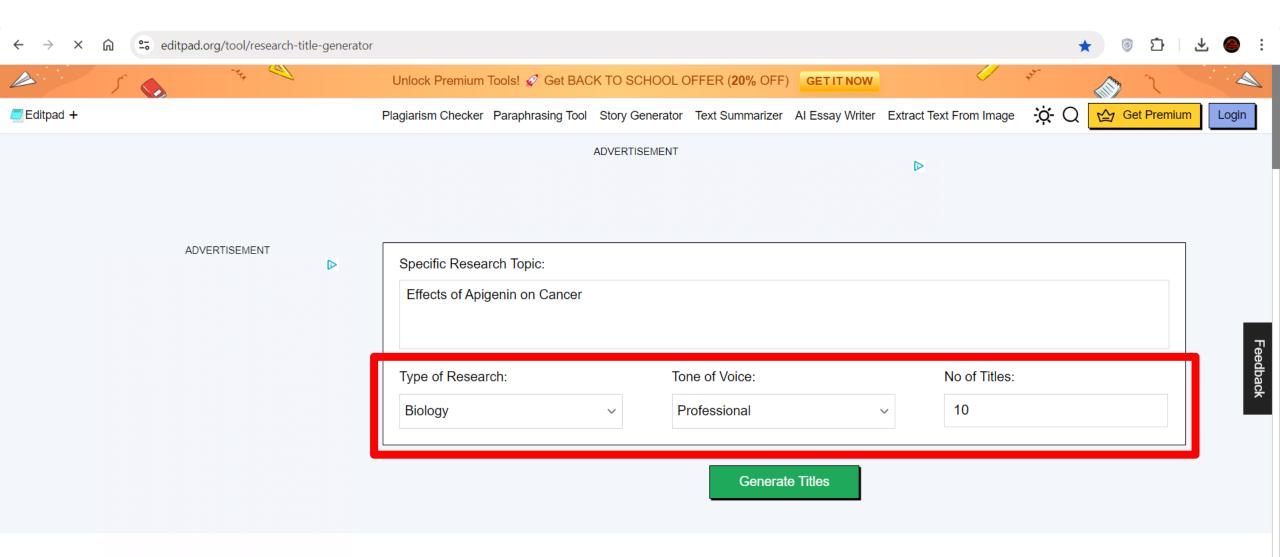






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#### Results

1. The Anti-Cancer Properties of Apigenin: Mechanisms of Action and Potential Therapeutic Applications	
2. Apigenin as a Novel Chemopreventive Agent in Cancer Treatment	
3. Understanding the Role of Apigenin in Inhibiting Cancer Cell Growth and Metastasis	
4. The Significance of Apigenin in Targeting Multiple Signaling Pathways in Cancer Cells	
5. Apigenin and Its Effects on Apoptosis and Autophagy in Cancer Cells	
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10. The Future of Apigenin-Based Therapies in Precision Medicine for Cancer Patients	

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	State your main idea about this topic: (optional)		
	Therapy		Feed
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	Excessive use of social media can lead to anxiety, depression, and low self-esteem	Teenagers and young adults	

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- 1. Innovative therapies targeting cancer stem cells show promise in improving treatment efficacy and reducing tumor relapse, ultimately revolutionizing cancer management strategies.
- 2. Understanding the unique characteristics of cancer stem cells is essential for developing therapies that can effectively eradicate tumors and prevent cancer recurrence.
- 3. Combining traditional cancer treatments with novel therapies aimed at cancer stem cells could enhance patient outcomes and provide a more comprehensive approach to cancer therapy.
- 4. Research into cancer stem cell biology offers insights that may lead to the identification of novel therapeutic targets, paving the way for more personalized and effective cancer treatment options.
- 5. The development of therapies that specifically target cancer stem cells could significantly improve survival rates and quality of life for patients battling aggressive forms of cancer.

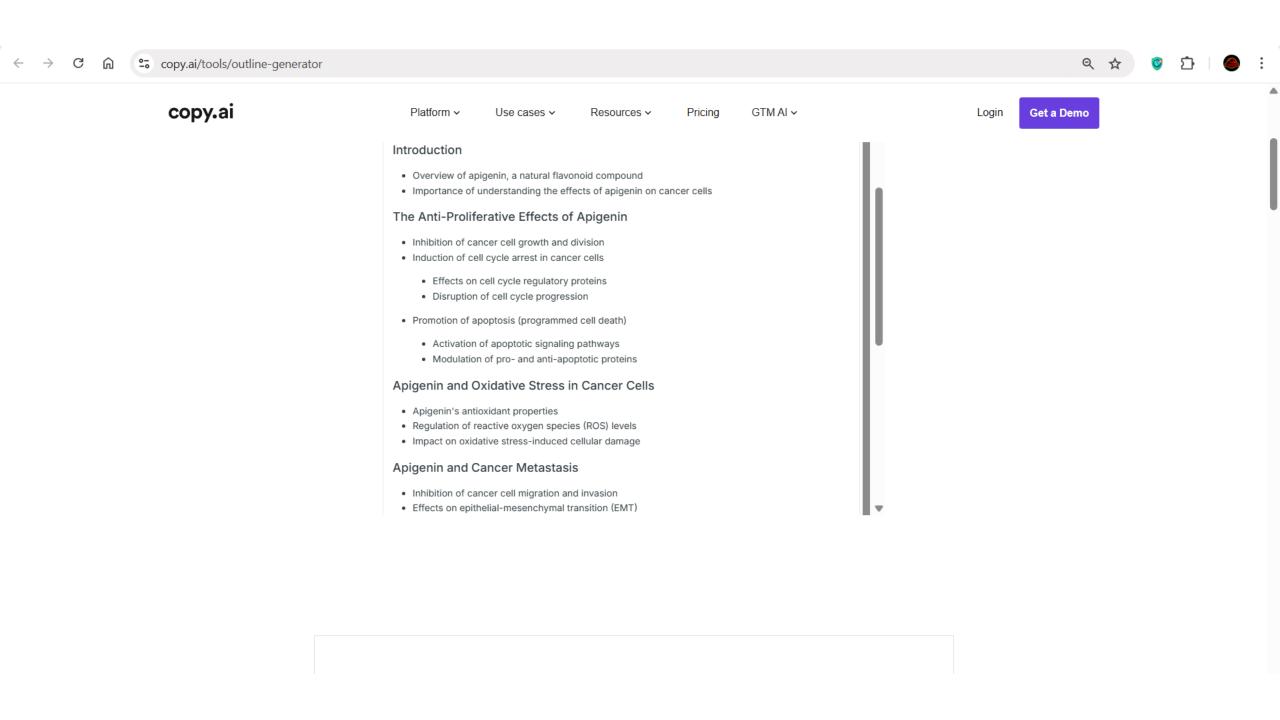
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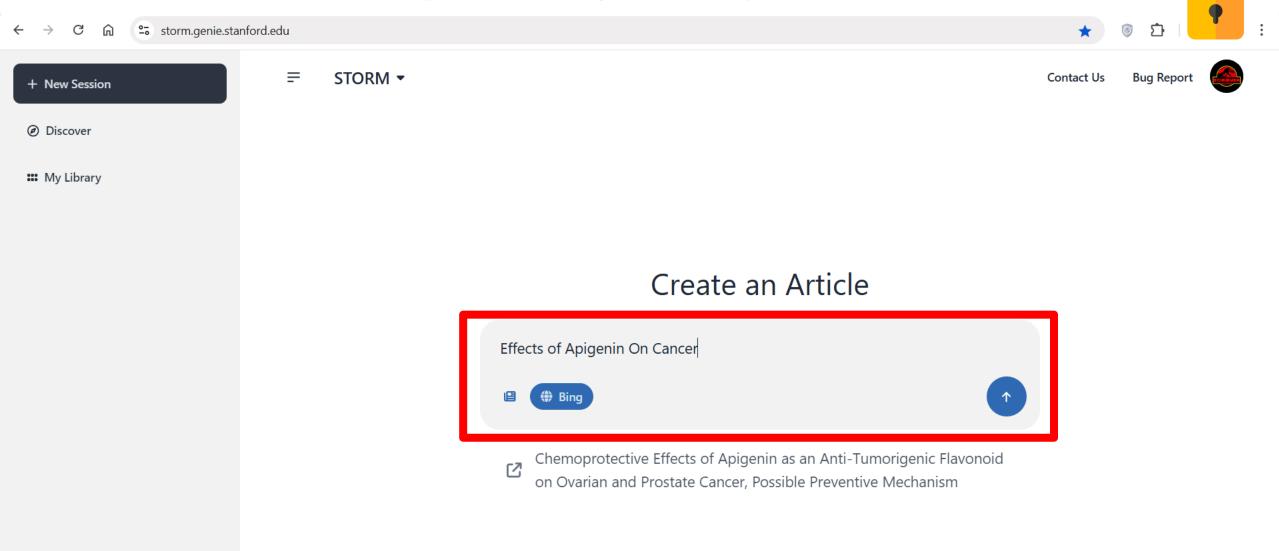
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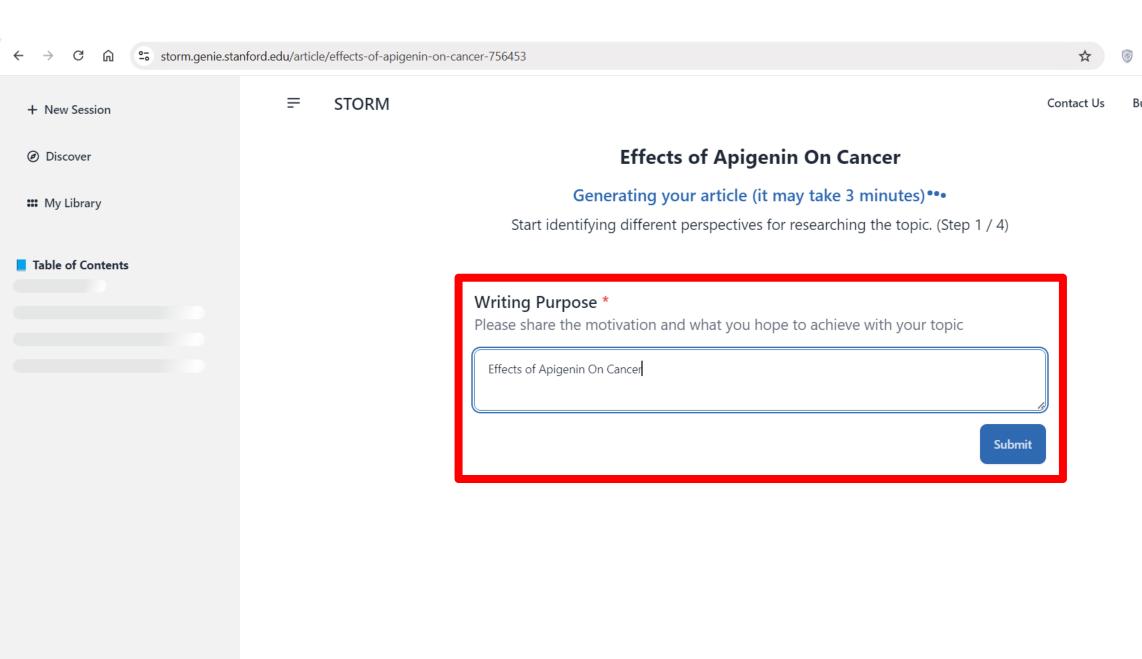
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Colorectal Cancer

Lung Cancer

Ovarian Cancer

Renal Cancer

Head and Neck Cancer





#### **Effects of Apigenin On Cancer**

See BrainSTORMing Process

#### summary

Apigenin is a naturally occurring flavonoid, chemically known as 4',5,7-trihydroxyflavone, found abundantly in various fruits, vegetables, and herbs such as parsley, celery, and chamomile. Notable for its diverse biological activities, apigenin has attracted significant interest in the field of cancer research due to its potential anticancer properties and mechanisms of action that target multiple pathways associated with tumor growth and progression. [1][2] Its reputation as a promising therapeutic agent stems from preclinical

#### Reference [1]

**Title:** Mechanism of Apigenin against breast cancer stem cells: network ...

**Url:** https://www.frontiersin.org/journals/pharmacology/articles/10.3389/fphar.2024.1496664/full

**Highlights:** . The cartoon representation enhances visualization of the ligand-protein binding residues and illustrates the molecule's propensity to form hydrogen bonds (Figure 3).

strating its ability to induce apoptosis, inhibit angiogenesis, and modulate cell on across several cancer types, including prostate, colorectal, lung, and ovarian he effectiveness of apigenin is underscored by its selective cytotoxic effects on ile sparing normal cells, a characteristic that enhances its appeal as a potential ventional chemotherapy. Studies have shown that apigenin can enhance the motherapeutic agents, such as cisplatin and ABT-263, by sensitizing cancer cells prosurvival signaling pathways.[3][4] However, despite the promising results eclinical models, apigenin's clinical application is limited by challenges related pility and the need for more rigorous clinical trials to validate its therapeutic an subjects.[5][2] Controversies surrounding apigenin's use include concerns ple bioavailability, as its effectiveness may be diminished by poor solubility and



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#### Apigenin and Specific Cancer Types

Prostate Cancer

Colorectal Cancer

Lung Cancer

Ovarian Cancer

Renal Cancer

Head and Neck Cancer

#### **Preclinical Studies**

Anti-Metastatic Effects

Safety and Toxicity Profile

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Safety and Side Effects

### ≡ STORM

### **Apigenin and Specific Cancer Types**

Apigenin, a naturally occurring flavonoid, has been shown to possess various anticancer properties across multiple cancer types. Its mechanisms of action include the modulation of cell cycle progression, induction of apoptosis, and inhibition of oncogenic signaling pathways.

#### **Prostate Cancer**

In prostate cancer, apigenin has demonstrated selective cytotoxic effects on cancer cells while sparing normal cells. It has been observed to enhance cisplatin's cytotoxic effects through p53-mediated apoptosis, highlighting its potential as a chemosensitizing agent [4][3]. Additionally, apigenin treatment in mouse models significantly suppressed prostate cancer progression by inhibiting IGF signaling and modulating AKT and ERK pathways [3].

#### **Colorectal Cancer**

Studies have indicated that apigenin can effectively inhibit growth in colorectal cancer cell lines, such as HCT116 and DLD1, by inducing cell cycle arrest at the G2/M phase and upregulating cell cycle inhibitors like p21WAF1/CIP1[3][5]. The flavonoid also enhances the efficacy of other chemotherapeutic agents, like ABT-263, through the inhibition of prosurvival regulators AKT and ERK[3].

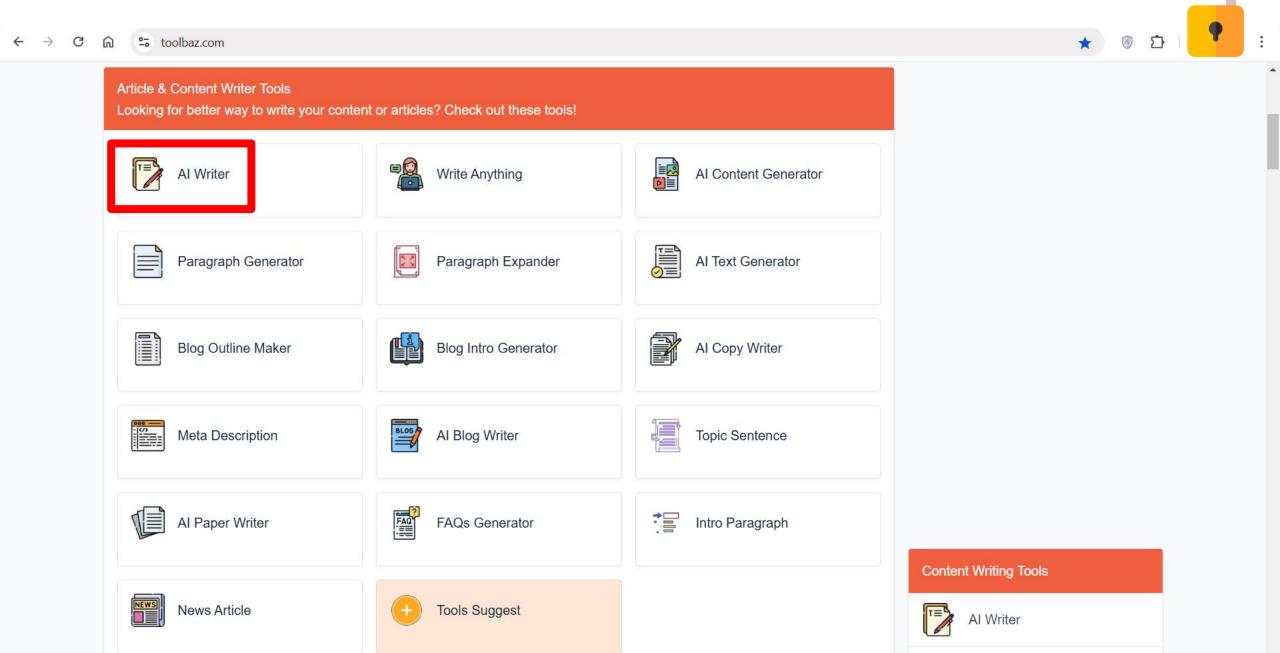
#### **Lung Cancer**

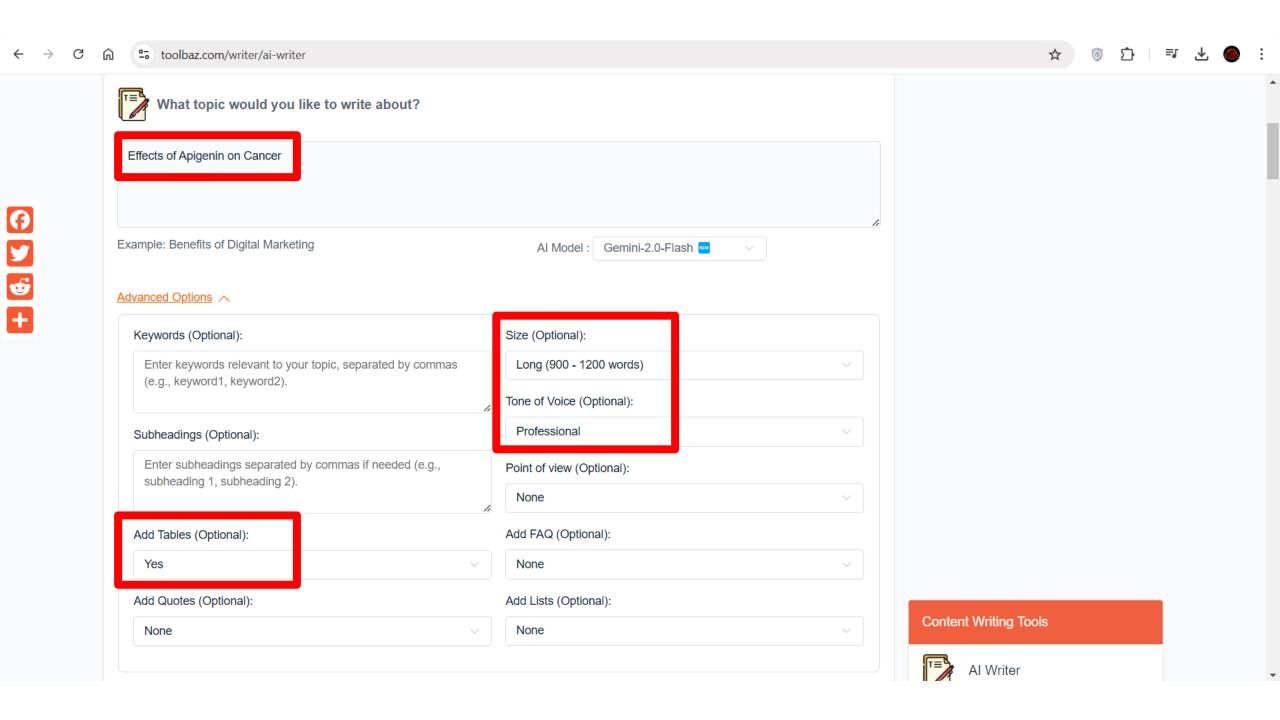
In non-small cell lung cancer (NSCLC), apigenin has been found to inhibit the translocation of NF-κB, a key regulator of survival signaling in cancer cells. This inhibition results in decreased expression of anti-apoptotic proteins such as Bcl-2 and Mcl-1, thereby promoting apoptosis[3]





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#### The Multifaceted Effects of Apigenin on Cancer: A Comprehensive Review

Apigenin, a naturally occurring flavonoid found abundantly in fruits, vegetables, and herbs such as parsley, celery, chamomile, and onions, has garnered significant attention in the scientific community for its potential chemopreventive and chemotherapeutic properties. Its ability to modulate multiple cellular pathways involved in cancer development and progression makes it a promising candidate for further investigation and potential inclusion in cancer treatment strategies. This article delves into the multifaceted effects of apigenin on cancer, exploring its mechanisms of action, preclinical evidence, potential clinical applications, and future research directions.

Flavonoids, like apigenin, are a diverse group of plant-derived polyphenols known for their antioxidant, anti-inflammatory, and anti-cancer properties. Apigenin, specifically, belongs to the flavone subclass and is characterized by its unique chemical structure, which allows it to interact with various cellular targets. Unlike some synthetic chemotherapeutic agents that can cause significant side effects, apigenin exhibits relatively low toxicity in normal cells, making it an attractive prospect for cancer prevention and therapy.

#### Mechanism of Action: A Multi-Targeted Approach

Apigenin exerts its anti-cancer effects through a variety of mechanisms, impacting multiple stages of cancer development, from initiation and promotion to progression and metastasis. This multi-targeted approach is a key advantage, as it addresses the complexity of cancer, which often involves the interplay of several signaling pathways.

Here's a breakdown of the key mechanisms:

- Induction of Apoptosis (Programmed Cell Death): One of the most well-documented effects of apigenin is its ability to induce apoptosis in cancer cells. Apoptosis is a crucial process that eliminates damaged or abnormal cells, preventing uncontrolled proliferation. Apigenin can trigger apoptosis through several pathways, including:
  - Activation of Caspases: Apigenin activates caspases, a family of proteases that play a central role in executing the apoptotic program.
  - Regulation of Bcl-2 Family Proteins: The Bcl-2 family of proteins includes both pro-apoptotic (e.g., Bax, Bak) and anti-apoptotic (e.g., Bcl-2, Bcl-xL) members. Apigenin can shift the balance



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Cancer Type	Mechanism(s) of Action	Study Type	Key Findings
Prostate Cancer	Apoptosis induction, cell cycle arrest, inhibition of angiogenesis, suppression of EMT	In vitro and in vivo	Reduced cell viability, increased apoptosis, decreased tumor growth, inhibited metastasis
Breast Cancer	Apoptosis induction, inhibition of cell proliferation, modulation of estrogen signaling	In vitro and in vivo	Reduced cell proliferation, induced apoptosis, decreased tumor size, inhibited angiogenesis, modulated estrogen receptor expression
Colon Cancer	Apoptosis induction, cell cycle arrest, inhibition of angiogenesis, anti-inflammatory	In vitro and in vivo	Reduced cell viability, increased apoptosis, decreased tumor growth, inhibited angiogenesis, suppressed inflammatory markers
Pancreatic Cancer	Apoptosis induction, inhibition of cell proliferation, chemosensitization	In vitro and in	Reduced cell viability, increased apoptosis, enhanced sensitivity to chemotherapy drugs, decreased tumor growth
Lung Cancer	Apoptosis induction, cell cycle arrest, inhibition of metastasis	In vitro and in vivo	Reduced cell viability, increased apoptosis, decreased tumor growth, inhibited metastasis, modulated expression of metastasis-related genes
Ovarian Cancer	Apoptosis induction, inhibition of cell proliferation, chemosensitization	In vitro and in vivo	Reduced cell viability, increased apoptosis, enhanced sensitivity to chemotherapy drugs, decreased tumor growth, inhibited angiogenesis
Leukemia	Apoptosis induction, cell cycle arrest, differentiation induction	In vitro and in vivo	Reduced cell viability, increased apoptosis, induced differentiation of leukemia cells, decreased leukemia cell burden



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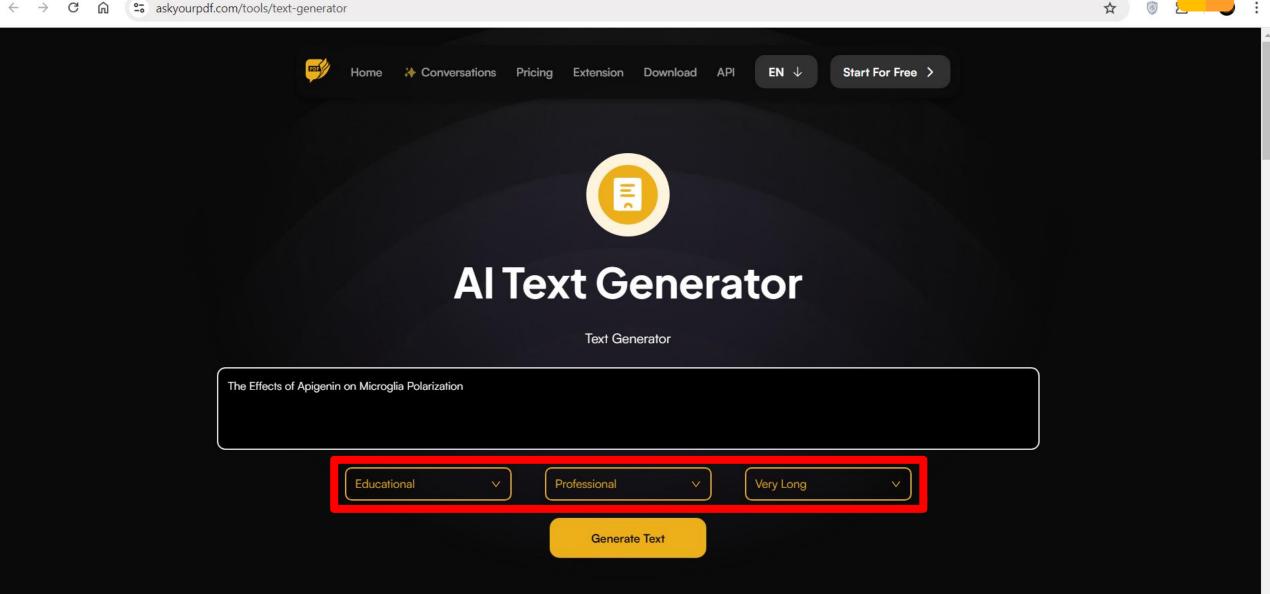
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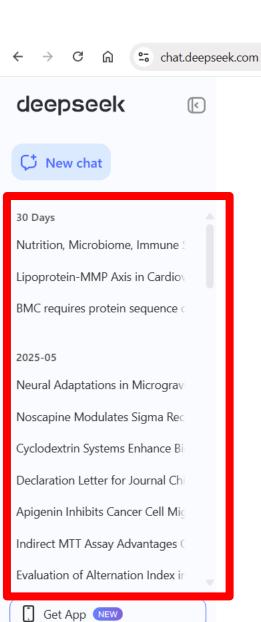


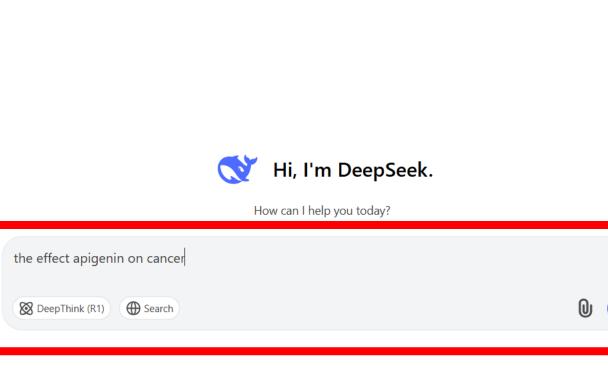
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Text Generator **♦**<sup>†</sup> Edit using our Editor Generate text Fill in the field (s) below to generate your text The Effects of Apigenin on Microglia Polarization The Effects of Apigenin on Microglia Polarization Microglia, the resident immune cells of the central nervous system (CNS), play a crucial role in maintaining brain homeostasis and Select Purpose responding to injury or infection. These cells are highly plastic and can adopt different phenotypes in response to various stimuli, a Select Purpose process known as microglia polarization. The polarization of microglia has been implicated in various neurological disorders, including Alzheimer's disease, Parkinson's disease, and multiple sclerosis. Recently, apigenin, a flavonoid compound found in Select Tone various plant-based foods, has gained attention for its potential therapeutic effects on microglia polarization. Select Tone Select Length Apigenin, a natural flavonoid, is found in high concentrations in fruits, vegetables, and herbs, such as parsley, chamomile, and celery. It has been shown to possess anti-inflammatory, antioxidant, and anti-cancer properties, making it a promising therapeutic Select Length  $\vee$ agent for various diseases. Recent studies have investigated the effects of apigenin on microglia polarization, with promising results. **Generate Text** 

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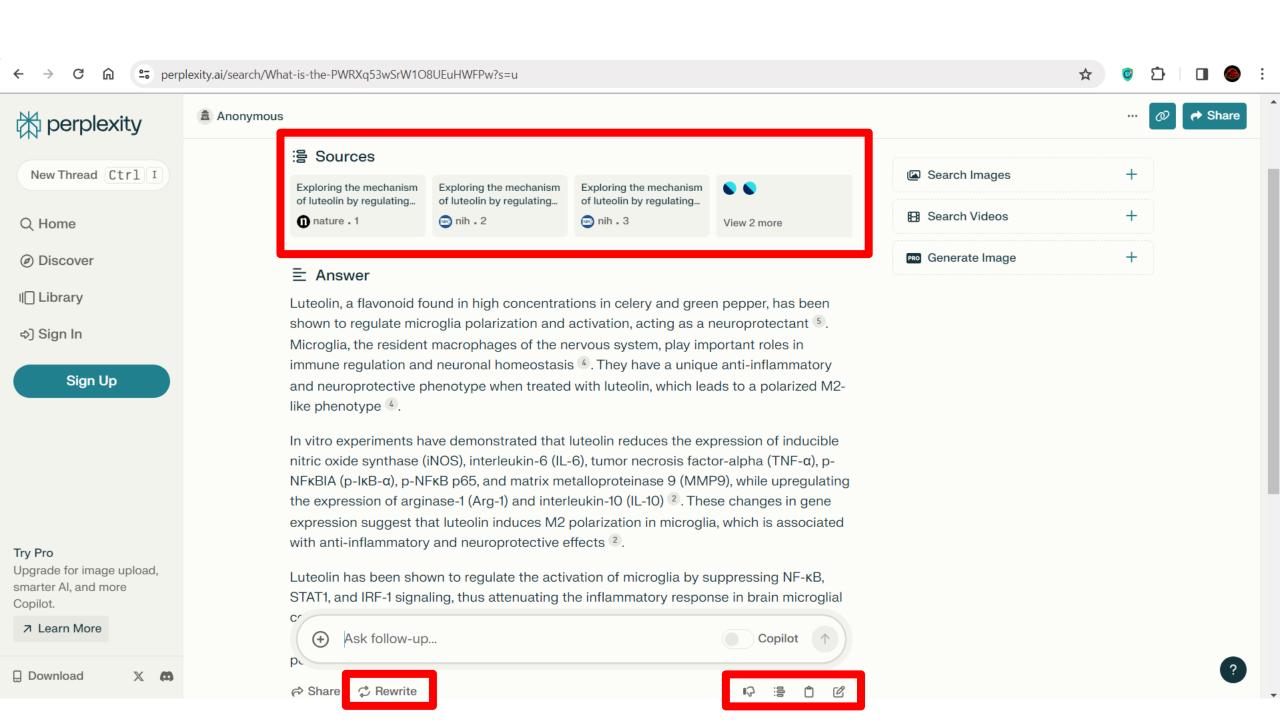


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#### ANSWER I PHIND V9 MODEL

Luteolin is a flavonoid found in many plants, including green tea, and has been studied for its potential health benefits. It has been shown to interact with various proteins and enzymes, including those involved in inflammatory responses and cell signaling pathways. However, the specific effect of Luteolin on microglia polarization is not directly mentioned in the provided sources.

Microglia are a type of immune cell in the brain that play a crucial role in maintaining brain homeostasis. They can become activated and migrate to sites of injury or infection, where they can release pro-inflammatory cytokines and other mediators. This process, known as polarization, is thought to contribute to neurological diseases such as Alzheimer's disease and multiple sclerosis.

From the given sources, one can infer that Luteolin might influence microglia polarization based on its known interactions with various proteins and enzymes. For example, it has been shown to bind to proteins like Interleukin-6 (IL-6), Interleukin-10 (IL-10), Matrix Metalloproteinase 9 (MMP9), Nuclear Factor kappa B Induced Apoptosis Protein (NFKBIA), and Tumor Necrosis Factor (TNF) with relatively low binding energies, indicating that these proteins may be potential targets for Luteolin 2.

However, without direct studies examining the effect of Luteolin on microglia polarization, it's difficult to definitively say what the impact would be. More research is needed to fully understand this interaction.









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SOURCES ☑ ▽

1 www.nature.com > articles > s41598-...

Exploring the mechanism of luteolin by regulating microglia ... - Nature

GO and KEGG analyses revealed that luteolin affects microglia polarization in regulation of inflammatory response as well as the interleukin (IL)-17 and tumor n..

2 www.ncbi.nlm.nih.gov > pmc > articles...

Exploring the mechanism of luteolin by regulating microglia ...

GO and KEGG analyses revealed that luteolin affects microglia polarization in regulation of inflammatory response as well as the interleukin (IL)-17 and tumor n..

3 neuroinflammation.biomedcentral.co...

Luteolin triggers global changes in the microglial transcriptome ...

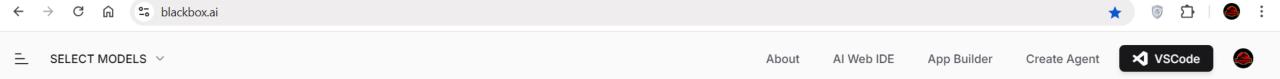
Luteolin dose-dependently suppressed proinflammatory marker expression in LPSactivated microglia and triggered global changes in the microglial transcriptome ..

4 www.ncbi.nlm.nih.gov > pmc > articles... Immunopharmacological Activities

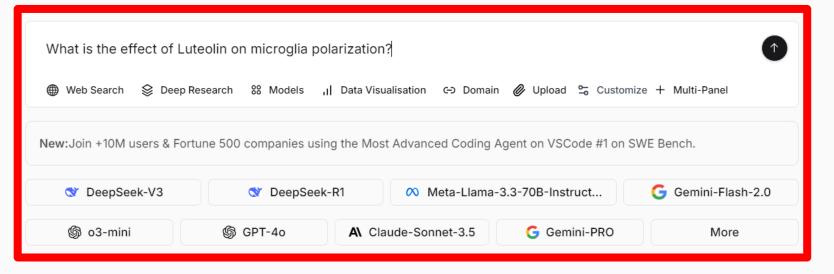
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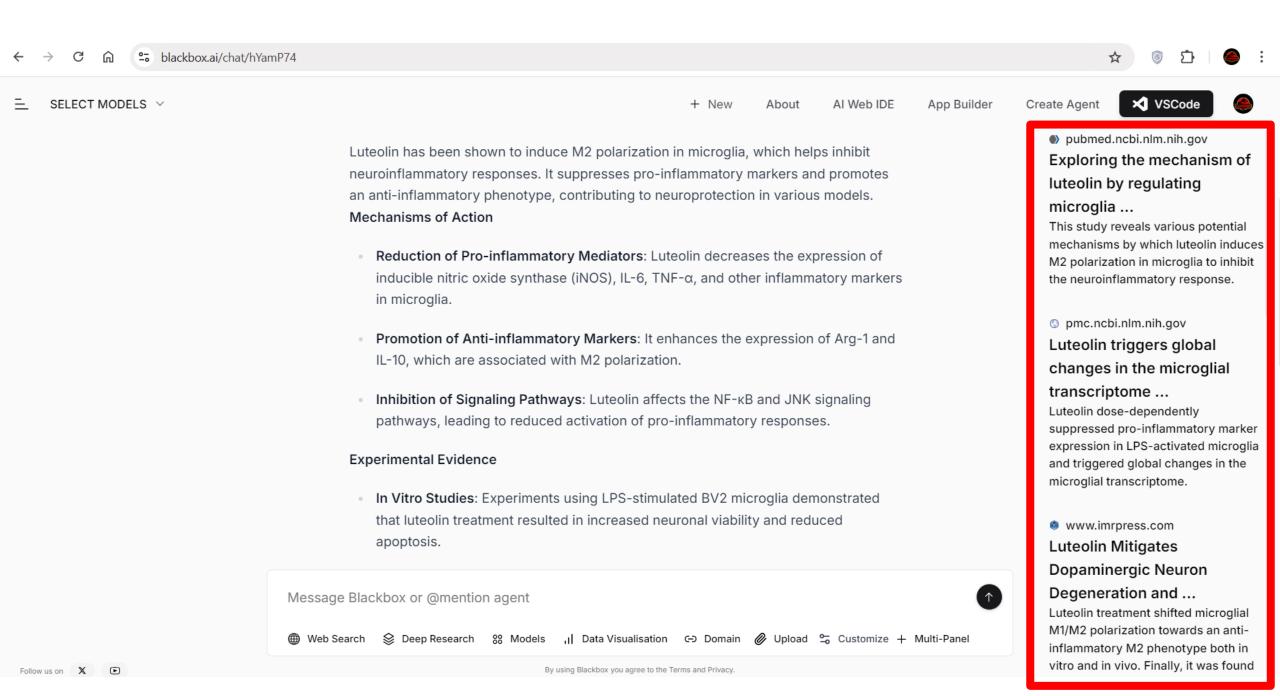
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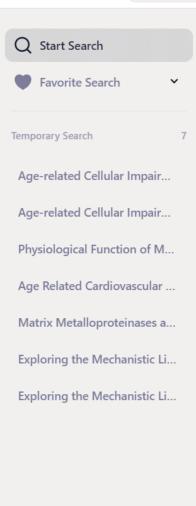












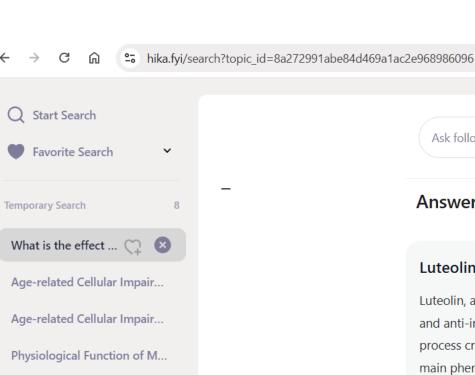
# Any answer It all starts with a question.

What is the effect of Luteolin on microglia polarization?

English

Suggestions & Feedback





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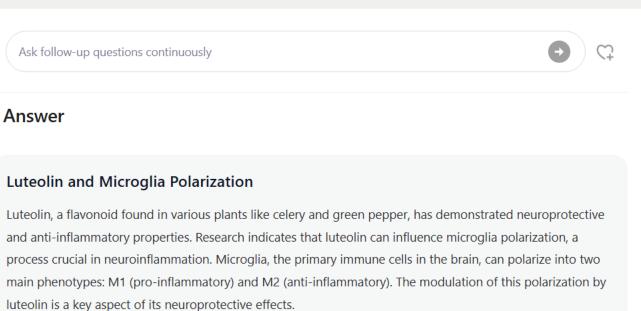
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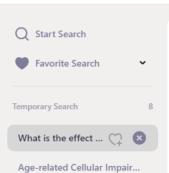
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#### **Mechanisms of Action**

Luteolin's effect on microglia polarization involves several mechanisms:

- **Regulation of inflammatory pathways:** Luteolin can suppress NFκB, STAT1, and IRF1 signaling, which are vital in the inflammatory response of brain microglial cells.
- M1/M2 Polarization: Luteolin can alter the M1/M2 polarization of macrophages, promoting an antiinflammatory response by downregulating pSTAT3 and upregulating pSTAT6 pathways.
- Direct anti-inflammatory effects: Preincubation of microglia with luteolin diminishes neurotoxic effects, owing to its direct antiinflammatory effects.



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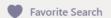
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Category	Key Points	Details
Luteolin Overview	Flavonoid Source	Luteolin is a flavonoid found in plants like celery and green pepper, known for its neuroprotective and anti-inflammatory properties.
Microglia Polarization	M1 and M2 Phenotypes	Microglia can polarize into M1 (pro-inflammatory) and M2 (anti-inflammatory) phenotypes, which is crucial in neuroinflammation.
Mechanisms of Action	Inflammatory Pathway Regulation	Luteolin suppresses NFkB, STAT1, and IRF1 signaling, vital for the inflammatory response in microglial cells.
	M1/M2 Polarization Alteration	It promotes an anti-inflammatory response by downregulating pSTAT3 and upregulating pSTAT6 pathways.
	Direct Anti-inflammatory Effects	Preincubation with luteolin reduces neurotoxic effects due to its direct anti-inflammatory properties.
Evidence from Studies	Target Identification	77 intersecting targets between luteolin and microglia polarization have been identified, indicating potential drug action targets.
	In Vitro Experiments	Experiments have validated cell culture systems and examined microglia polarization and related target protein expression.
	Model System Validation	Dose-response curves for luteolin were performed to study its genome-wide transcriptional effects on activated microglia.
Neuroprotective Effects	Neuroinflammation Attenuation	Luteolin promotes M2 polarization, helping to resolve neuroinflammation linked to neurodegenerative diseases.
	Cognitive Impairment Protection	It shows protective effects against cognitive impairment, likely due to its anti-inflammatory and antioxidant properties.

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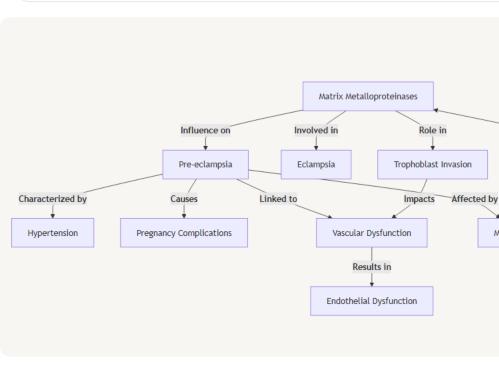
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#### Matrix Metallopro...



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Ask follow-up questions continuously

- Matrix Metalloproteinases (MMPs): A group of enzymes crucial for remodeling the extracellular matrix, significantly impacting pregnancy outcomes.
- Pre-eclampsia: A pregnancy complication characterized by hypertension and organ damage, affecting 3-8% of pregnancies globally. It is influenced by MMP activity.
- Eclampsia: The severe progression of pre-eclampsia, leading to seizures and serious complications.

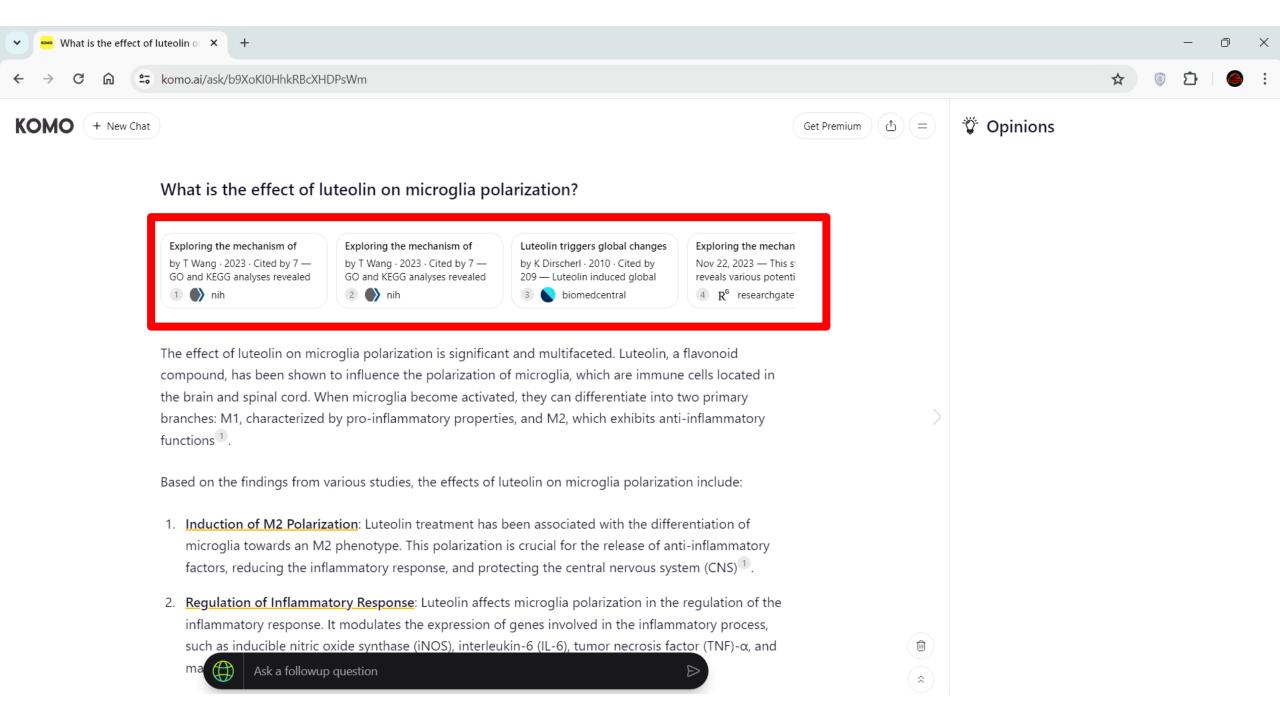


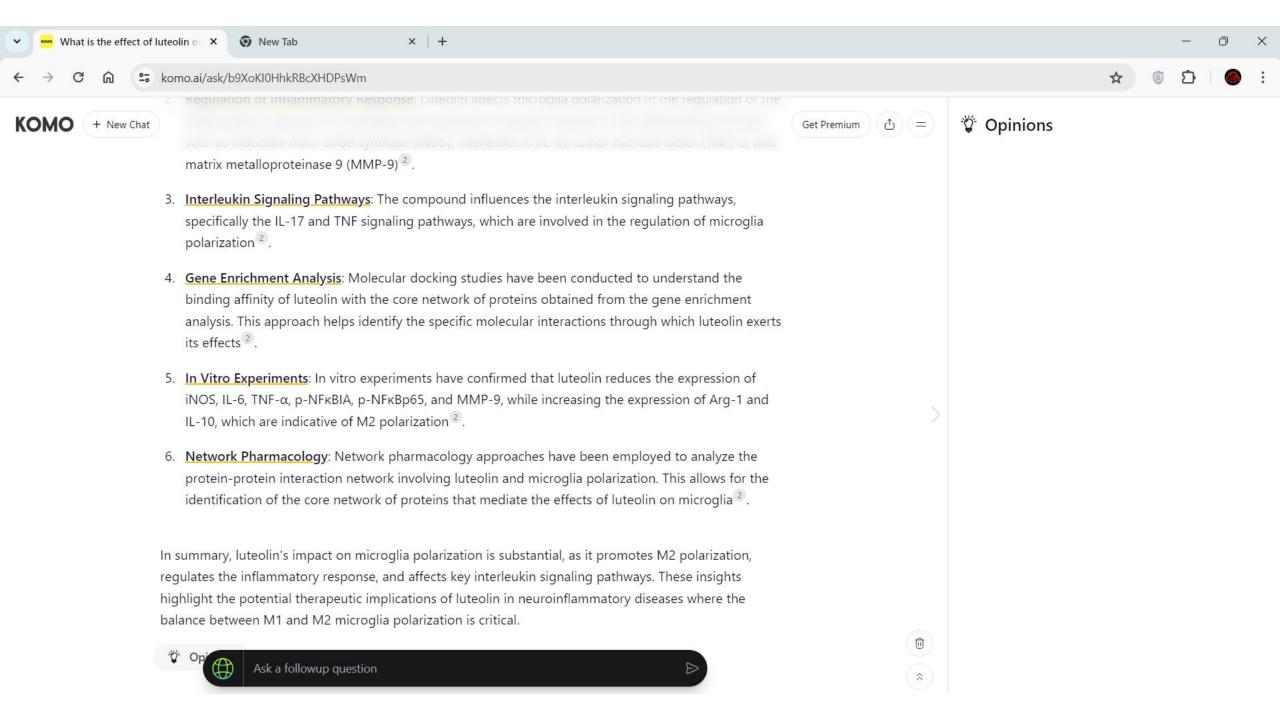
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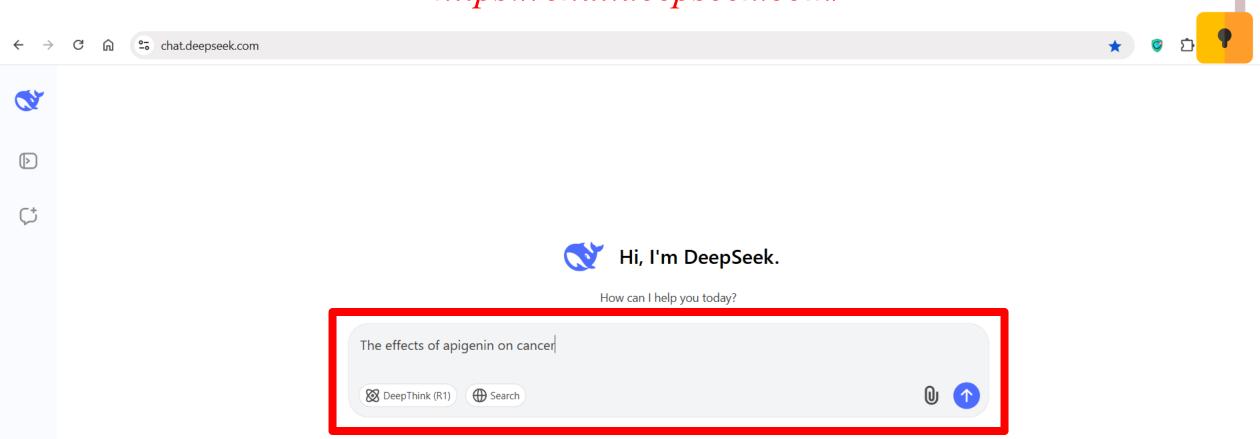


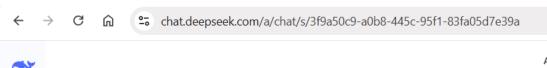






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#### Apigenin's Potential in Cancer Prevention and Treatment

### 1. Antiproliferative Effects

Apigenin inhibits cancer cell proliferation by:

- Cell Cycle Arrest: Induces G2/M or G1/S phase arrest by modulating cyclins (e.g., cyclin D1, cyclin B) and cyclin-dependent kinases (CDKs).
- Downregulation of Oncogenes: Suppresses MYC, RAS, and other pro-proliferative signals.

#### 2. Pro-Apoptotic Effects

Apigenin promotes apoptosis (programmed cell death) through:

- Activation of Intrinsic Pathways: Upregulates pro-apoptotic proteins (Bax, Bak) and downregulates anti-apoptotic proteins (Bcl-2, Bcl-xL).
- Caspase Activation: Stimulates caspase-3, -8, and -9.
- p53 Activation: Enhances tumor suppressor p53 function in p53-wildtype cancers.

#### 3. Anti-Metastatic and Anti-Invasive Effects

- . Inhibition of Epithelial-Mesenchymal Transition (EMT): Downregulates Snail, Twist, and MMPs (matrix metalloproteinases), reducing metastasis.
- Suppression of Angiogenesis: Inhibits VEGF and HIF-1α, limiting tumor blood supply.

#### 4. Anti-Inflammatory Effects

- NF-κB Inhibition: Blocks NF-κB signaling, reducing pro-inflammatory cytokines (TNF-α, IL-6) that promote tumor growth.
- COX-2 Suppression: Lowers cyclooxygenase-2, linked to inflammation-driven cancers.



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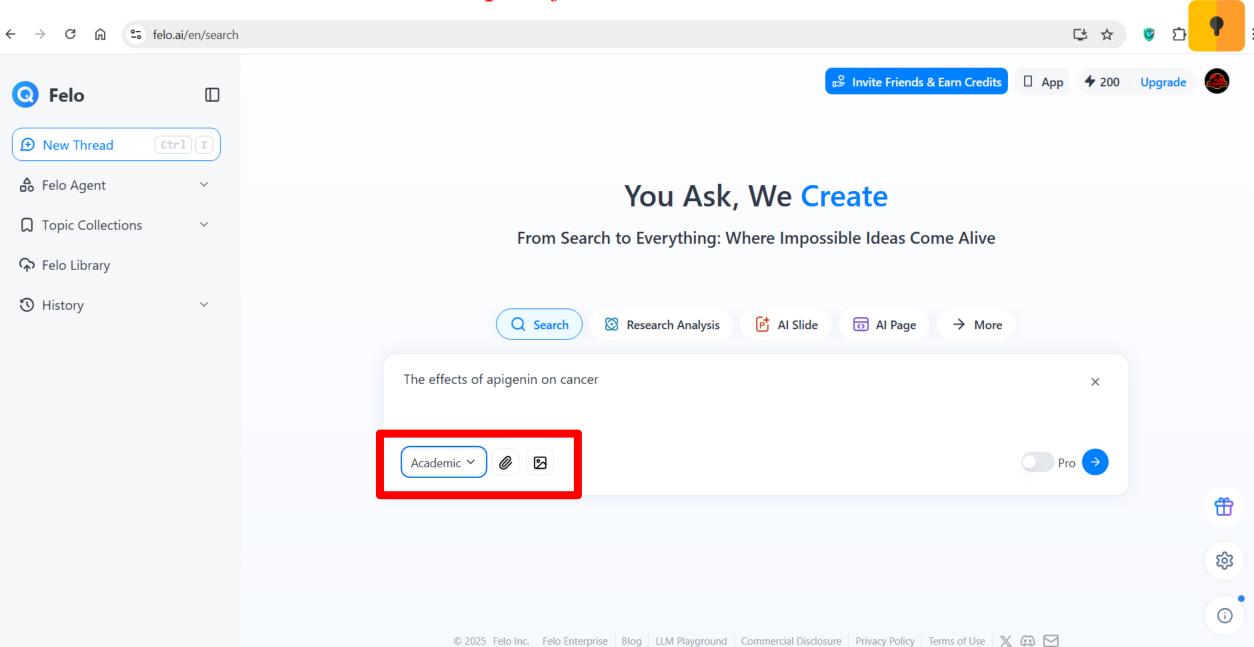


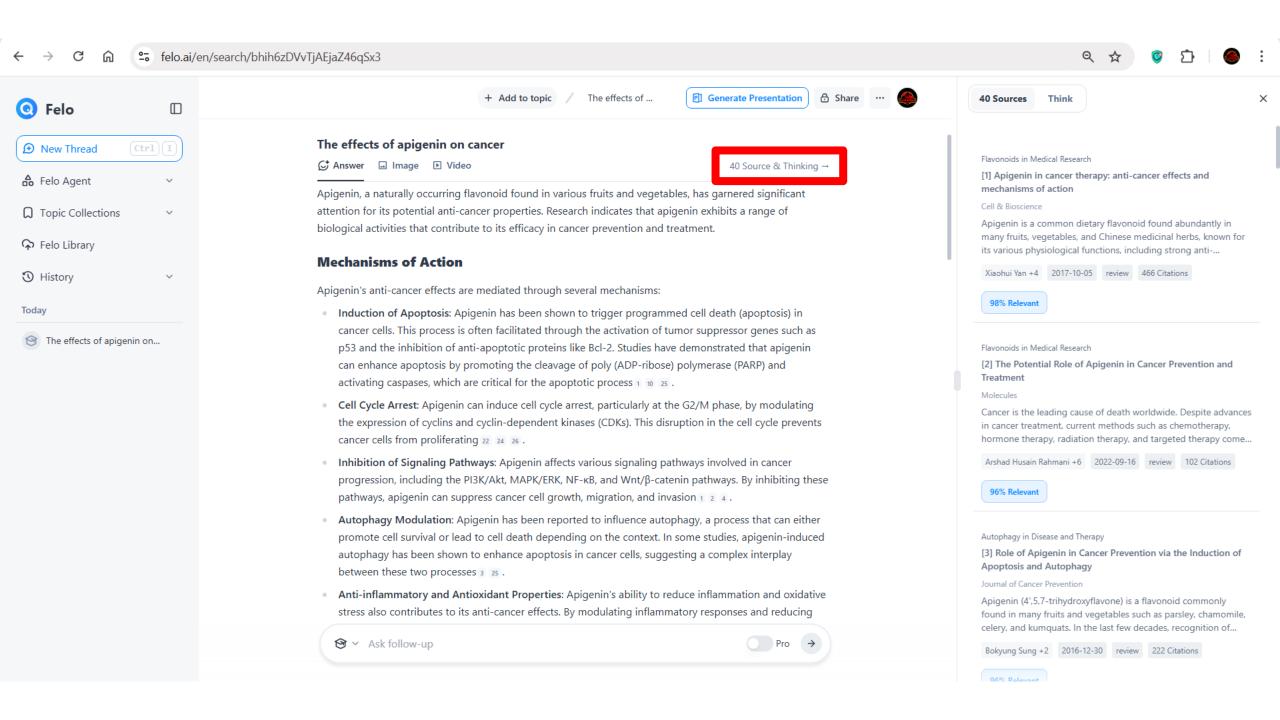




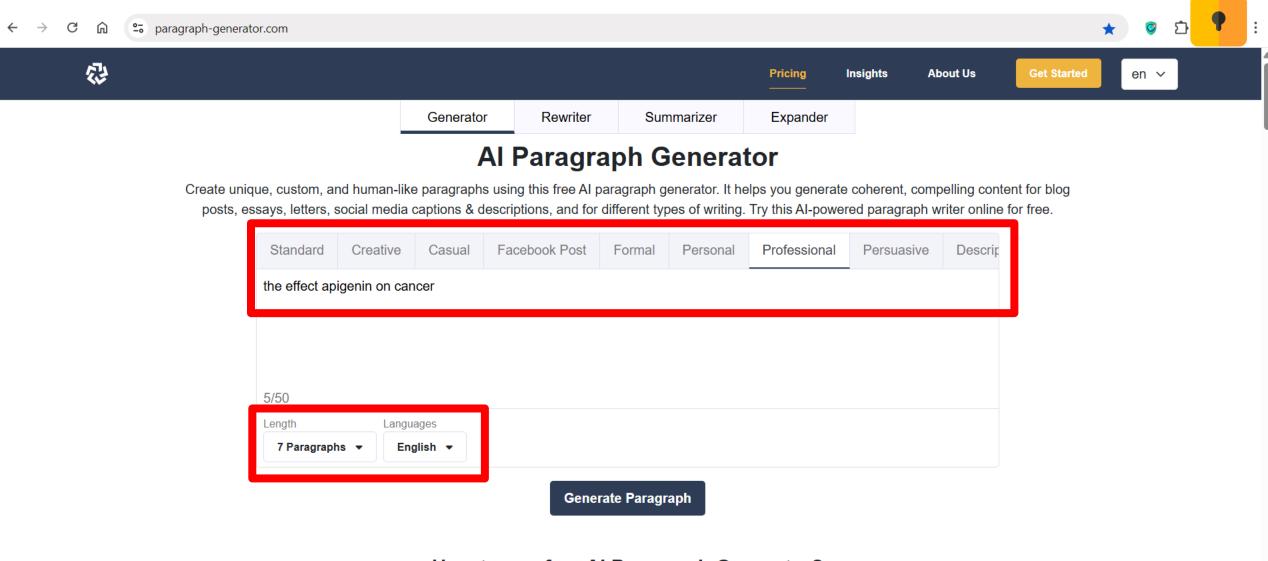


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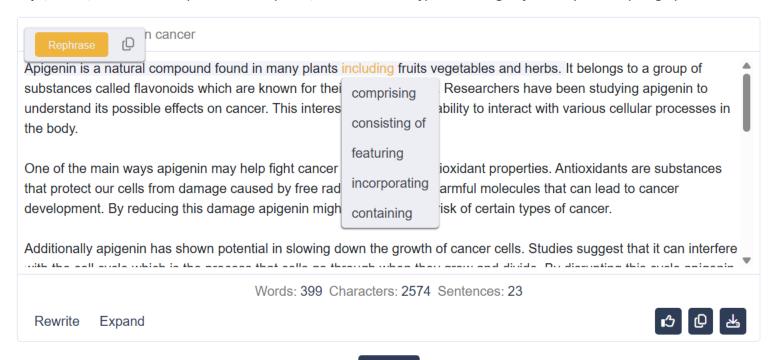






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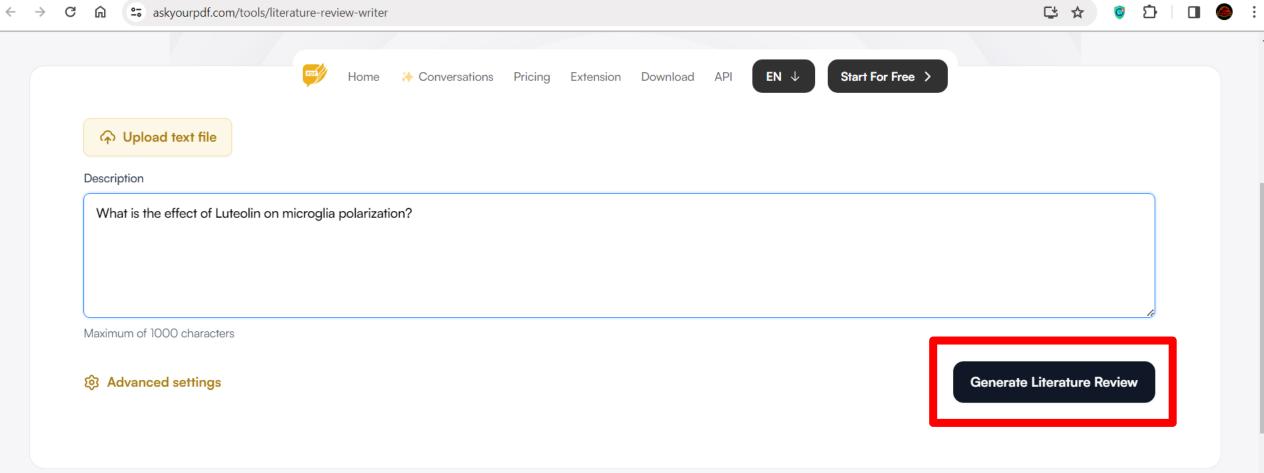
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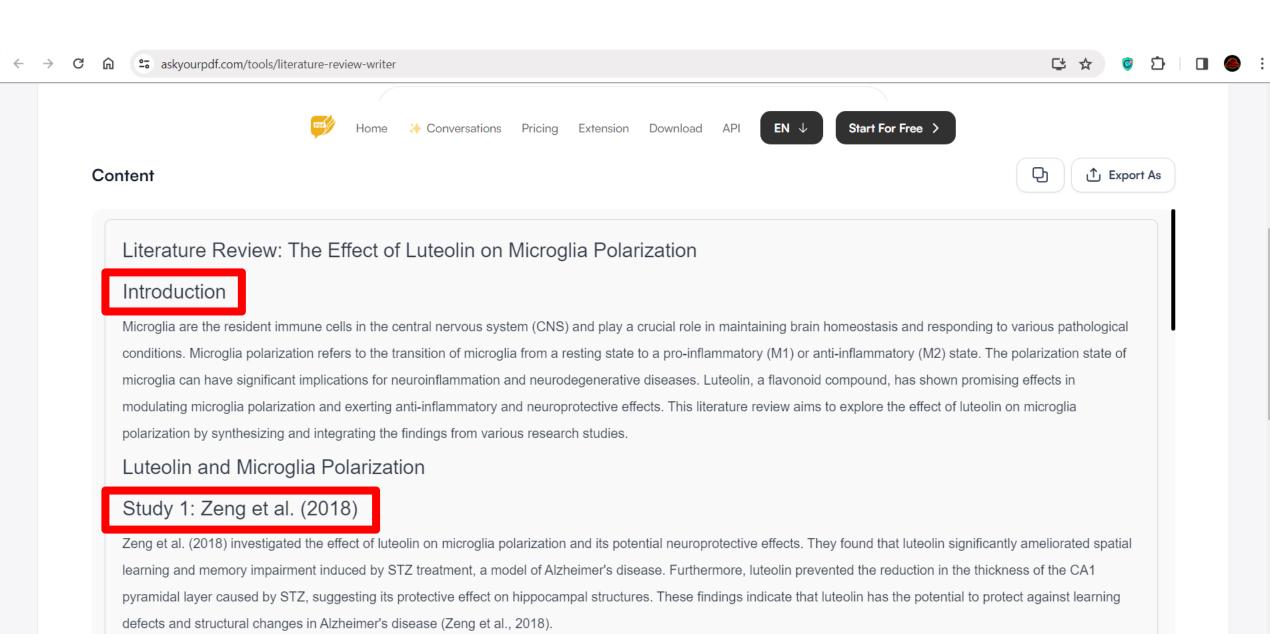




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## Study 2: Wang et al. (2016)

In an in vitro study, Wang et al. (2016) investigated the effect of luteolin on neuroinflammatory responses in lipopolysaccharide (LPS)-stimulated microglia. They found that luteolin inhibited the expression of proinflammatory mediators, reduced the production of nitric oxide and prostaglandin E2, and blocked NF-kB activation. These anti-inflammatory effects of luteolin suggest its therapeutic potential in the treatment of neuroinflammatory disorders (Wang et al., 2016).

## Study 3: Clayton et al. (2021)

Clayton et al. (2021) explored the role of luteolin in plaque-associated microglia and its effect on neurotoxicity. They found that luteolin exhibited direct anti-inflammatory effects and protected against neurotoxicity. These findings suggest that luteolin may have therapeutic potential in the treatment of neuroinflammatory disorders (Clayton et al., 2021).

## Study 4: Park et al. (2016)

Park et al. (2016) investigated the anti-neuroinflammatory effect of luteolin glucuronides, the major metabolites found in plasma after oral administration of luteolin. They found that luteolin glucuronides reduced the expression of inflammatory genes in LPS-treated microglia, suggesting their involvement in the anti-inflammatory effect of luteolin in vivo. These findings highlight the importance of luteolin metabolites in mediating its therapeutic effects (Park et al., 2016).

## Study 5: Zhu et al. (2011)

Zhu et al. (2011) studied the inhibitory effect of luteolin on microglial inflammation and its potential in improving neuron survival. They found that luteolin inhibited microglial inflammation and improved neuron survival against inflammation. This suggests that luteolin has the potential to modulate microglial activation and protect neurons from











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activation (Subedi et al., 2019).

## Knowledge Gaps and Future Research Directions

Although the existing research provides valuable insights into the effect of luteolin on microglia polarization, several knowledge gaps remain:

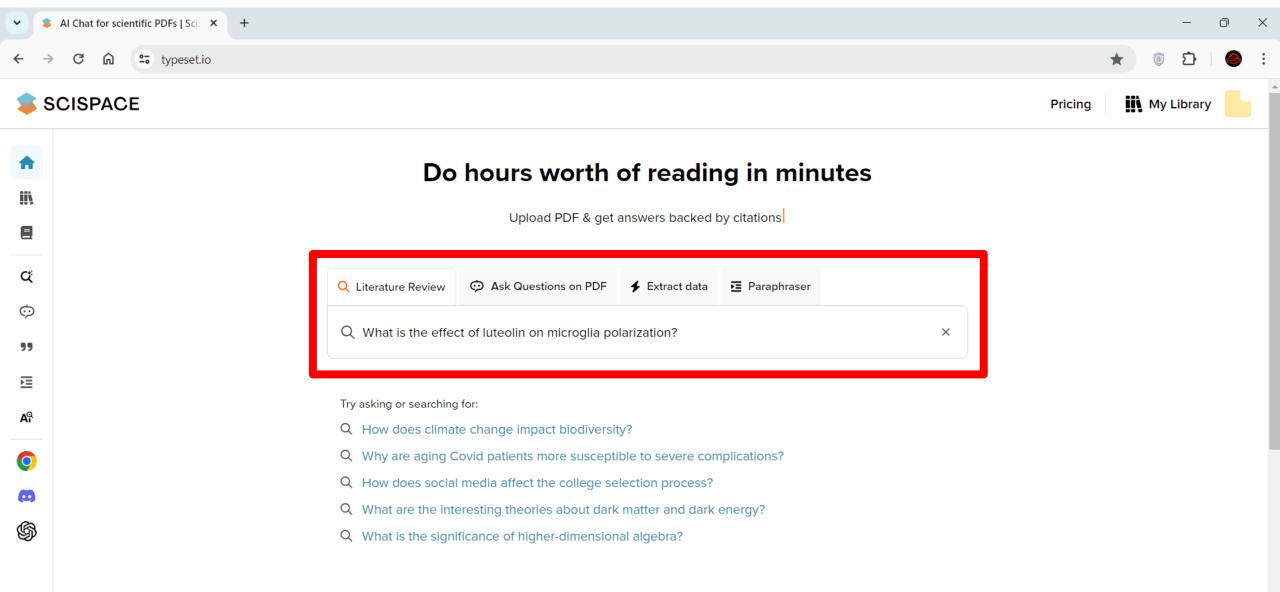
- 1. Mechanisms of action: While studies have demonstrated the anti-inflammatory and neuroprotective effects of luteolin on microglia, the precise molecular mechanisms underlying its effect on microglia polarization are not fully understood. Further research is needed to elucidate the signaling pathways and molecular targets involved.
- 2. In vivo studies: Most of the existing studies on luteolin and microglia polarization have been conducted in vitro. Future research should focus on in vivo studies to validate the findings and assess the therapeutic potential of luteolin in animal models of neurodegenerative diseases.
- 3. Metabolism and bioavailability: The metabolism and bioavailability of luteolin and its metabolites play a crucial role in determining their therapeutic effects. Further research is needed to investigate the pharmacokinetics and optimize the delivery methods of luteolin to maximize its efficacy.
- 4. Combination therapy: Luteolin has shown potential as an adjuvant therapy in combination with other therapeutics. Future research should explore the synergistic effects of luteolin with existing drugs or compounds to enhance therapeutic outcomes in neuroinflammatory diseases.

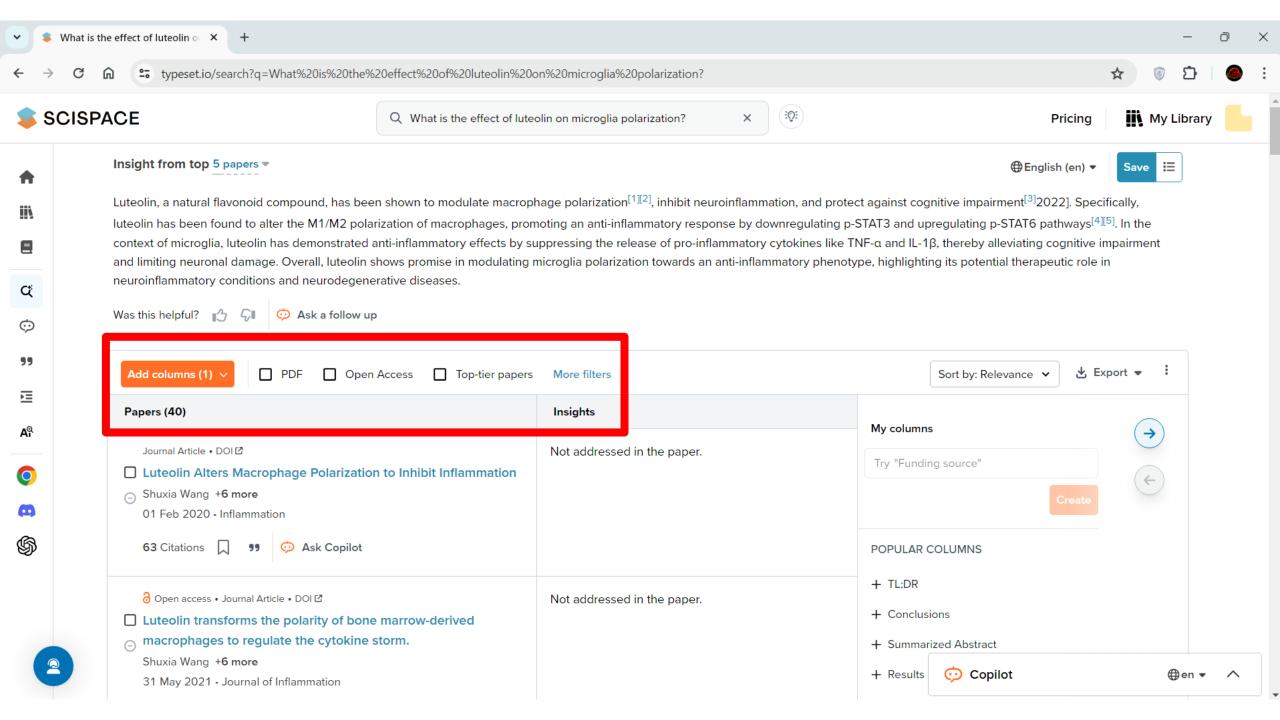
## Conclusion

In conclusion, luteolin has demonstrated beneficial effects on microglia polarization, exerting anti-inflammatory and neuroprotective effects. It has the potential to modulate microglial activation, inhibit the production of inflammatory mediators, and protect against neurotoxicity. However, further research is warranted to elucidate the underlying mechanisms, validate the findings in animal models, optimize delivery methods, and explore combination therapy approaches. Understanding the effects of luteolin on microglia polarization will contribute to the development of novel therapeutic strategies for neuroinflammatory diseases.

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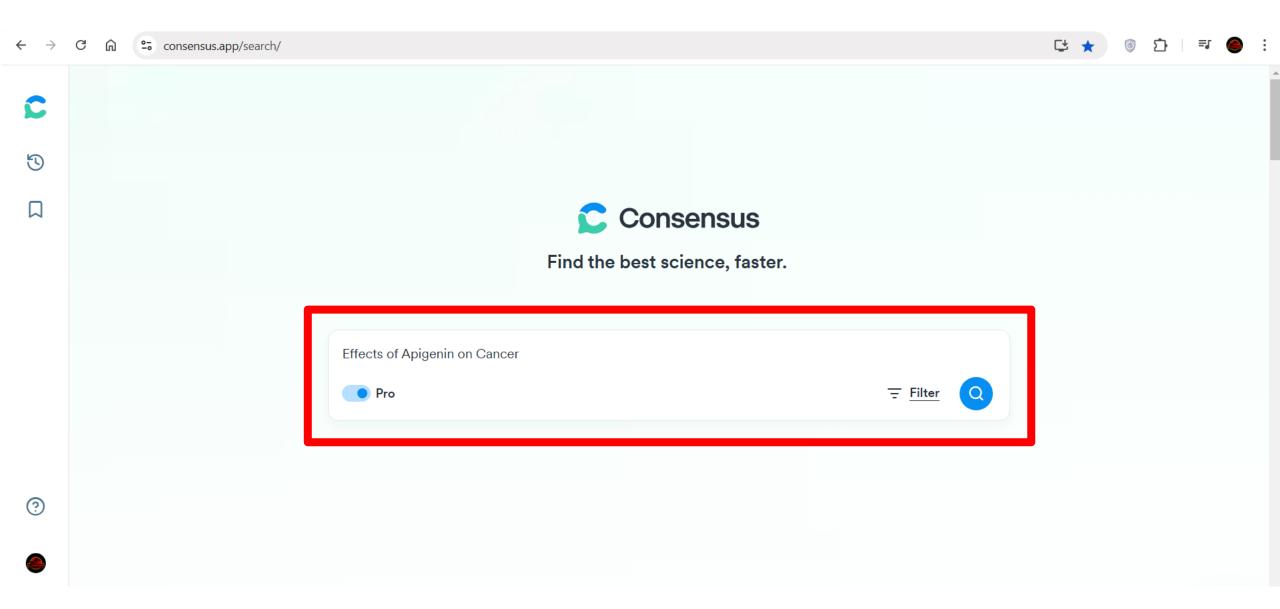


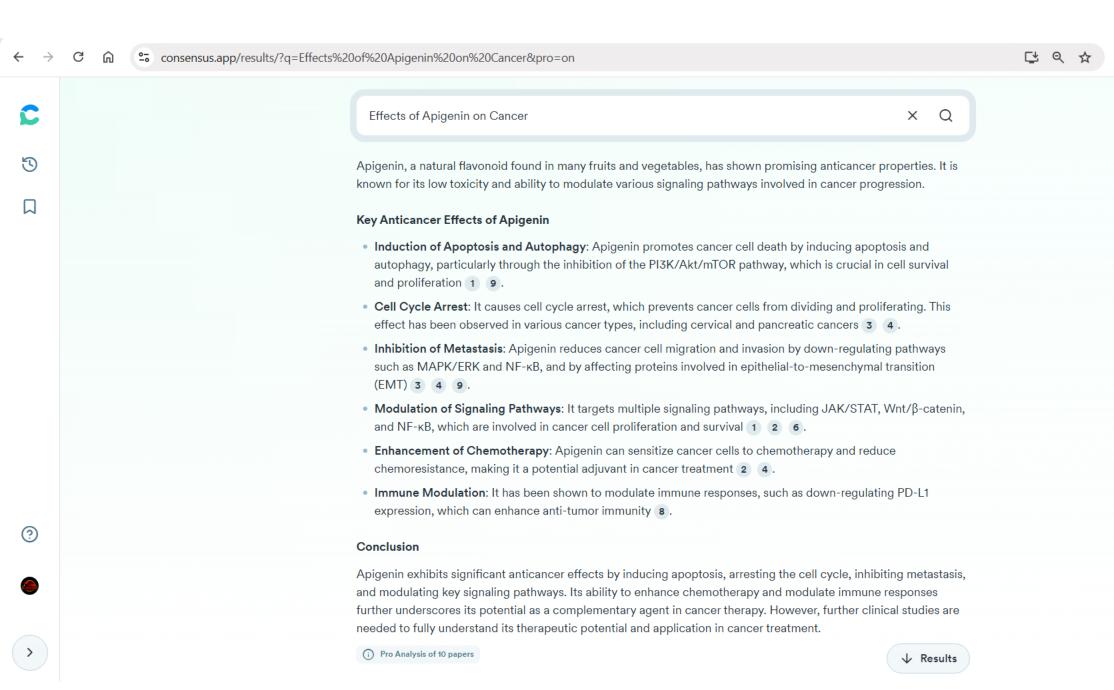


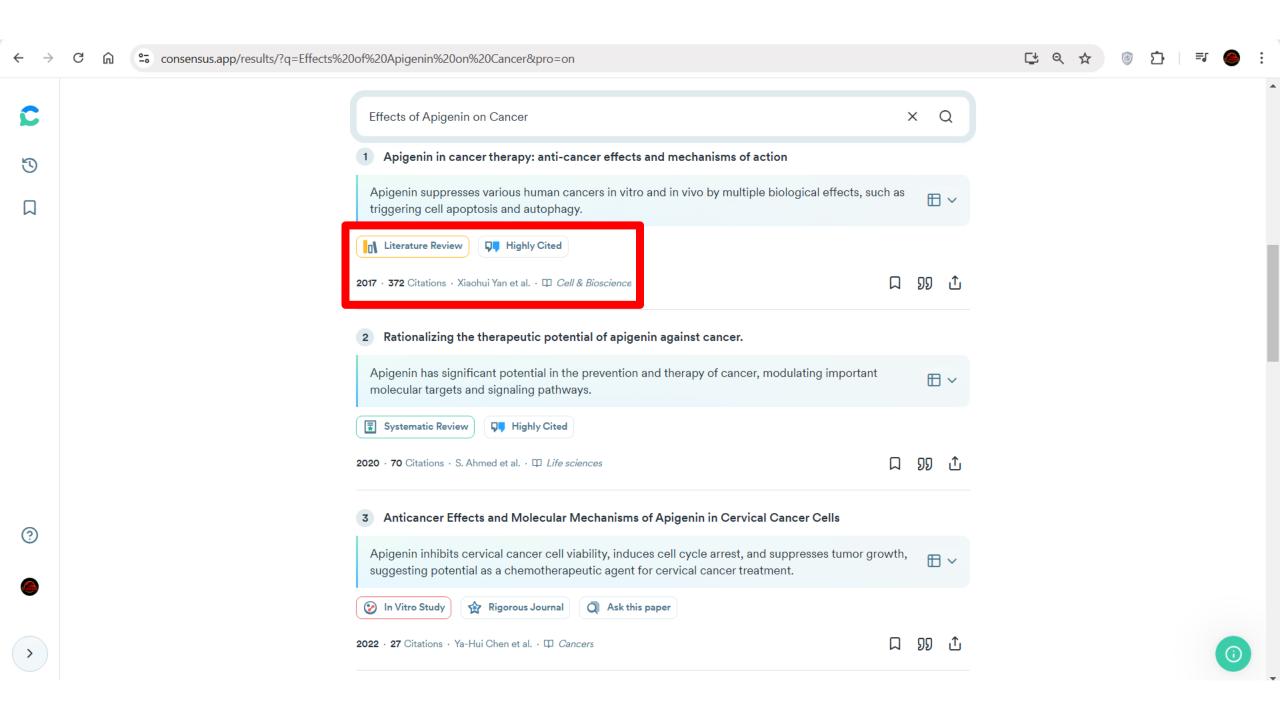


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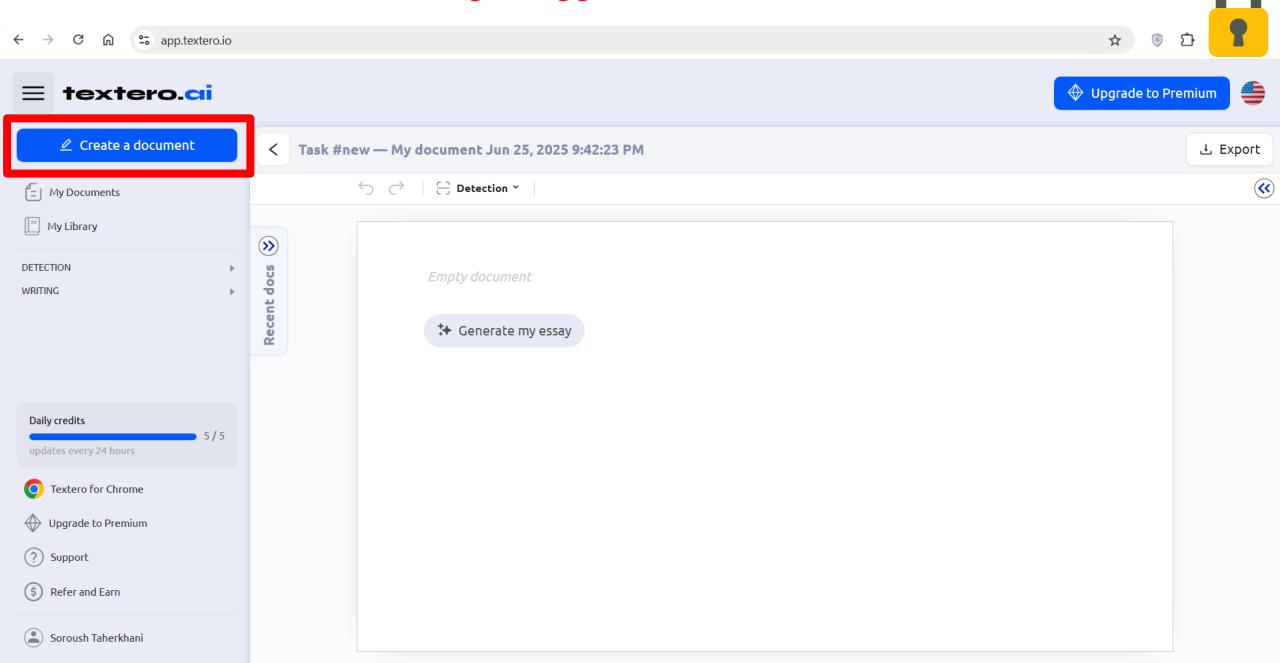


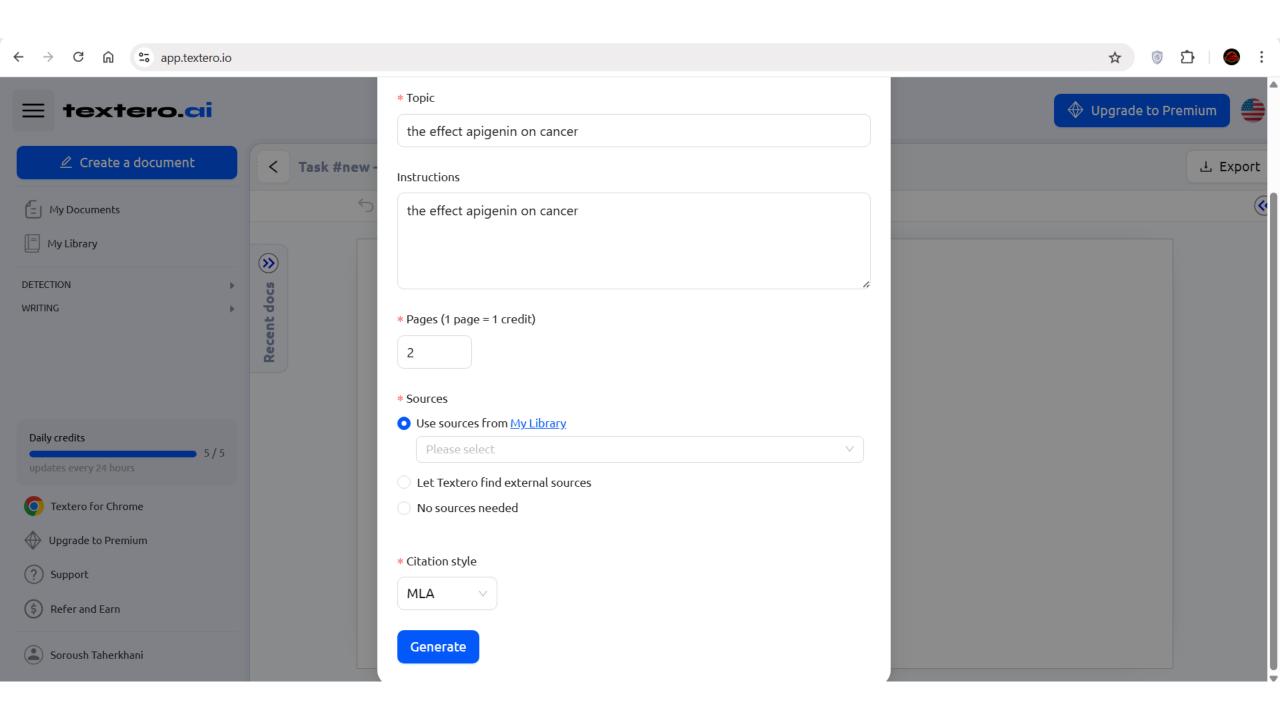


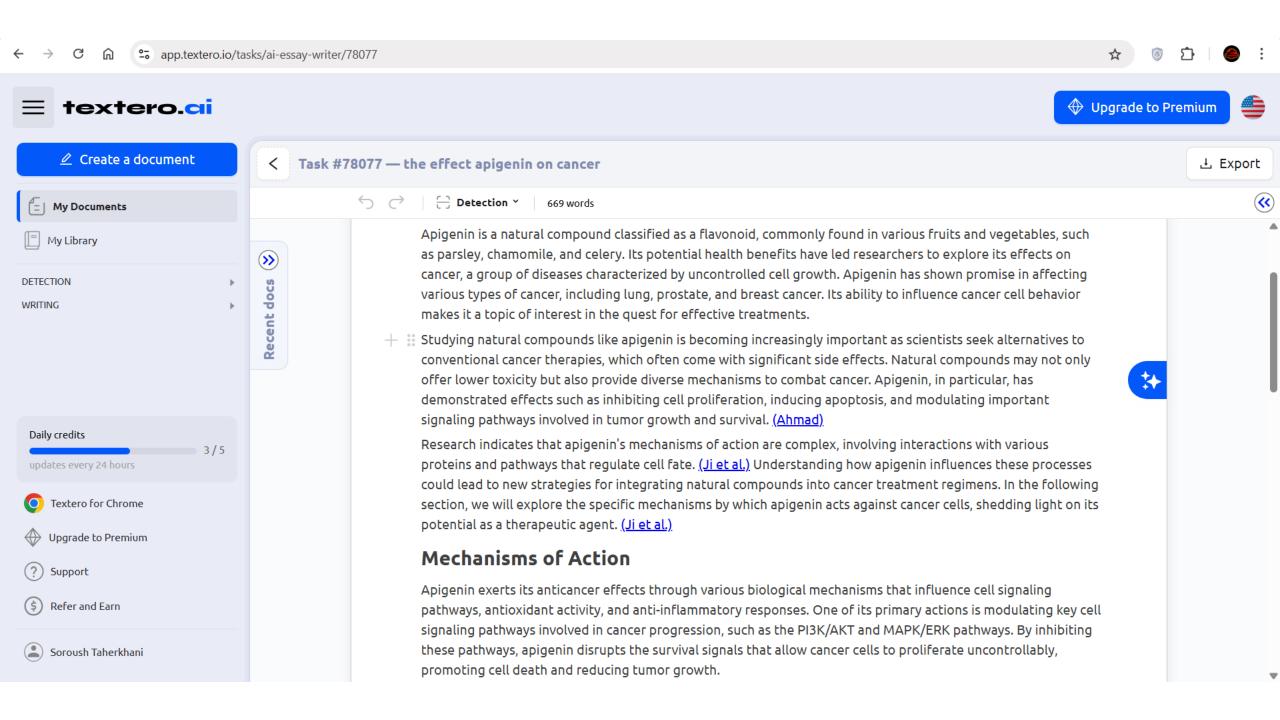


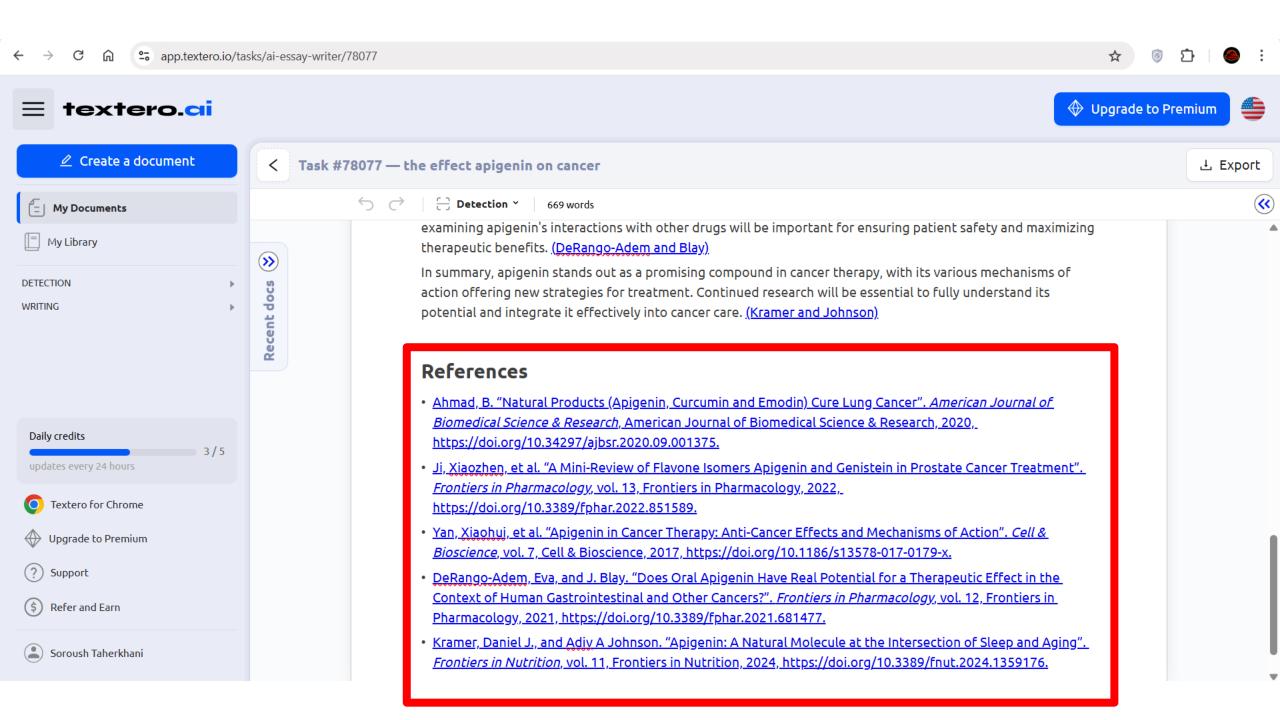


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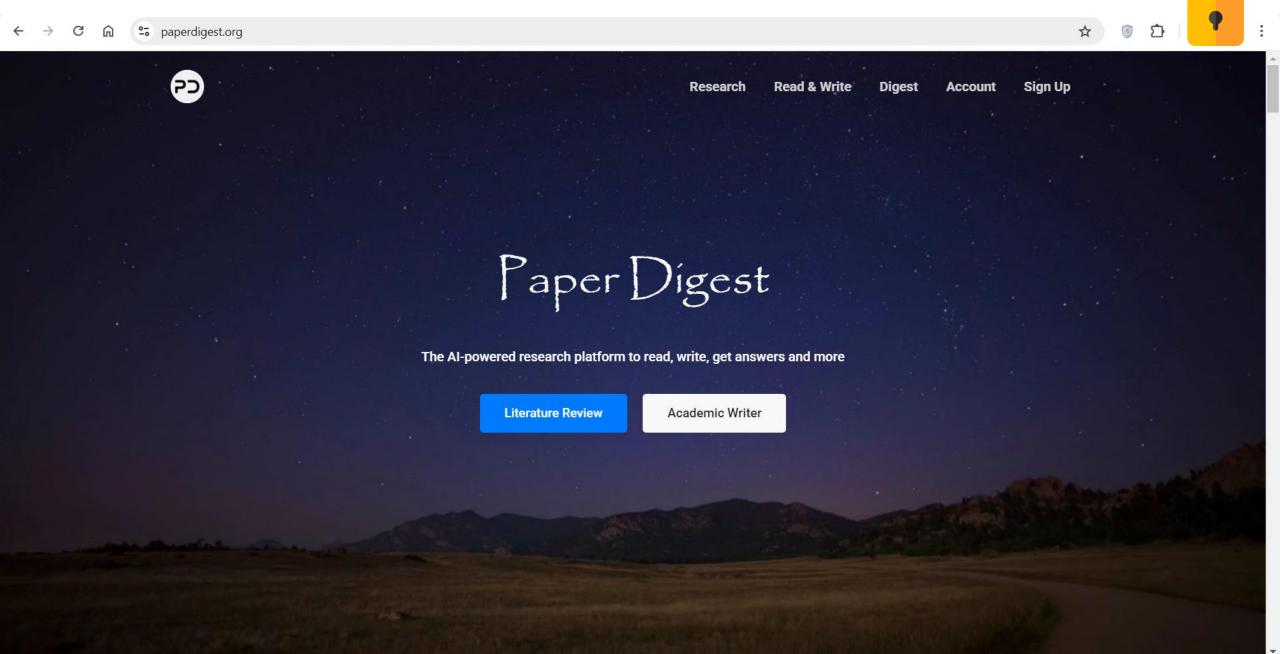








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#### **■** Review of the Related Work

Apigenin, a naturally occurring flavone, has garnered significant attention in cancer research due to its potential chemopreventive and therapeutic effects on various types of cancer. Several studies have explored the impact of apigenin on different cancer cell lines, shedding light on its mechanisms of action and its synergistic effects with other anticancer agents. (Liu et. al., 2005) demonstrated that apigenin inhibits the expression of vascular endothelial growth factor (VEGF) in lung cancer cells, suggesting its potential in lung cancer chemoprevention. The study highlighted that apigenin targets the hypoxia-inducible factor 1 (HIF-1) binding site, leading to decreased VEGF expression. Similarly, (Mirzoeva et. al., 2013) found that apigenin inhibits TGF-β-induced VEGF expression in prostate carcinoma cells through a Smad2/3- and Src-dependent mechanism, indicating its potential in prostate cancer treatment. (Lee et. al., 2008) and (Shao et. al., 2013) explored the inhibitory effects of apigenin on invasive growth and metastasis in breast and colon cancer cells, respectively. (Lee et. al., 2008) highlighted the role of apigenin in blocking the PI3K/Akt pathway and beta 4 integrin function in breast cancer cells, while (Shao et. al., 2013) observed a synergistic effect between apigenin and ABT-263 on apoptosis in colon cancer cells. These findings suggest that apigenin may have promising implications in inhibiting cancer progression and metastasis. Moreover, (Li et. al., 2009) and (Ketkaew et. al., 2016) investigated the inhibitory effects of apigenin on ovarian cancer and head and neck squamous cell carcinoma, respectively. (Li et. al., 2009) highlighted apigenin's ability to inhibit the proliferation of ovarian cancer cells through Id1, while (Ketkaew et. al., 2016) demonstrated apigenin's role in inhibiting hypoxia-induced stem cell marker expression in head and neck squamous cell carcinoma cells. Furthermore, (Abid et. al., 2022) provided an updated overview of apigenin's pharmacological properties, emphasizing its anti-inflammatory, antibacterial, antiviral, and anticancer effects. The study critically analyzed the impact of apigenin on cancer cell signaling pathways, including the PI3K/AKT/MTOR, JAK/STAT, NF-kB, and ERK/MAPK pathways, further supporting its potential as a therapeutic agent in cancer treatment. In conclusion, the literature review of the effects of apigenin on cancer highlights its diverse mechanisms of action and its potential as a chemopreventive and therapeutic agent in various types of cancer. The synergistic effects of apigenin with other anticancer agents, as demonstrated in several studies, further underscore its promising role in cancer treatment. Continued research in this field is essential to fully elucidate the therapeutic potential of apigenin and its application in clinical settings for cancer management.



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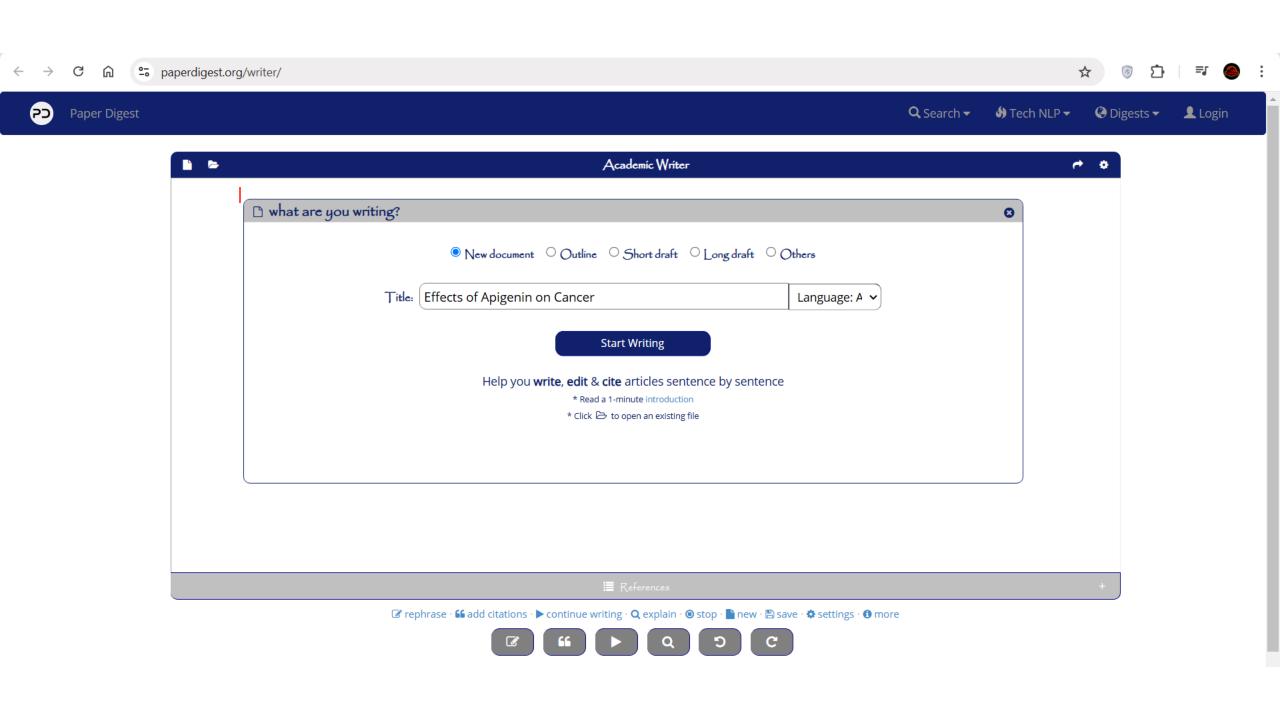


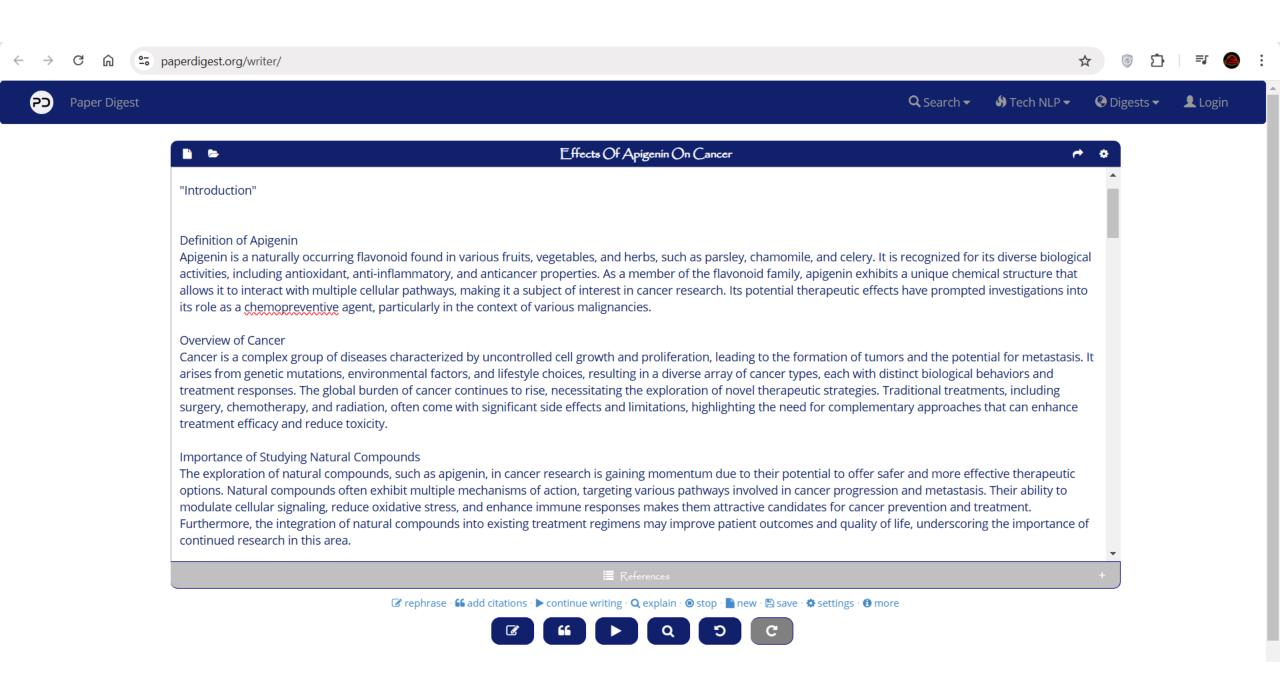






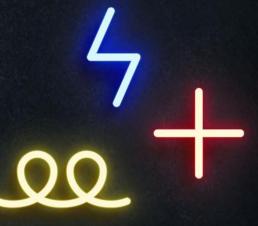
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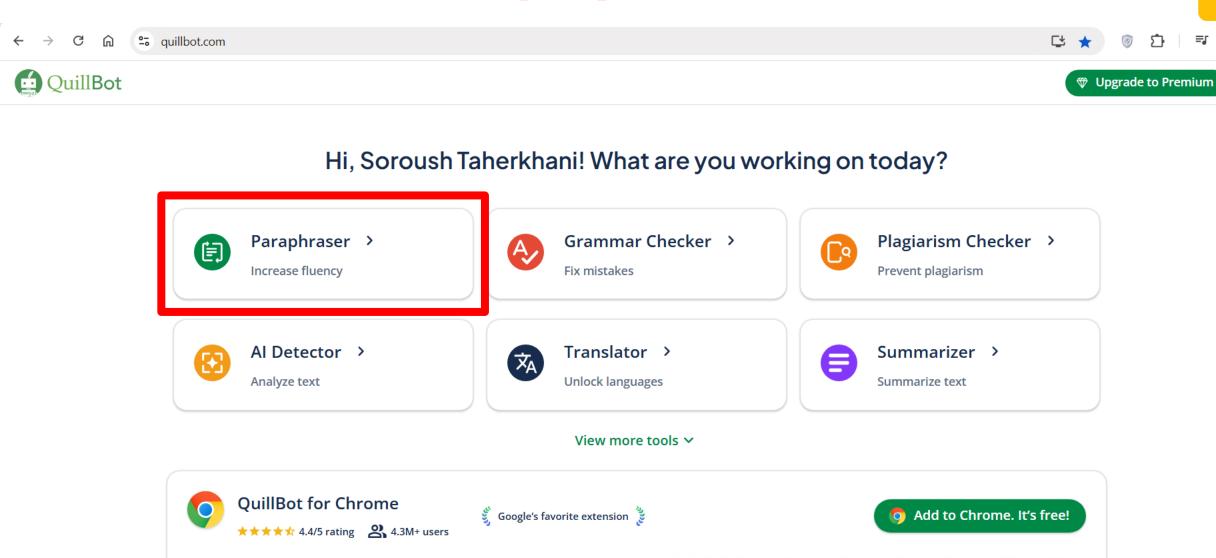






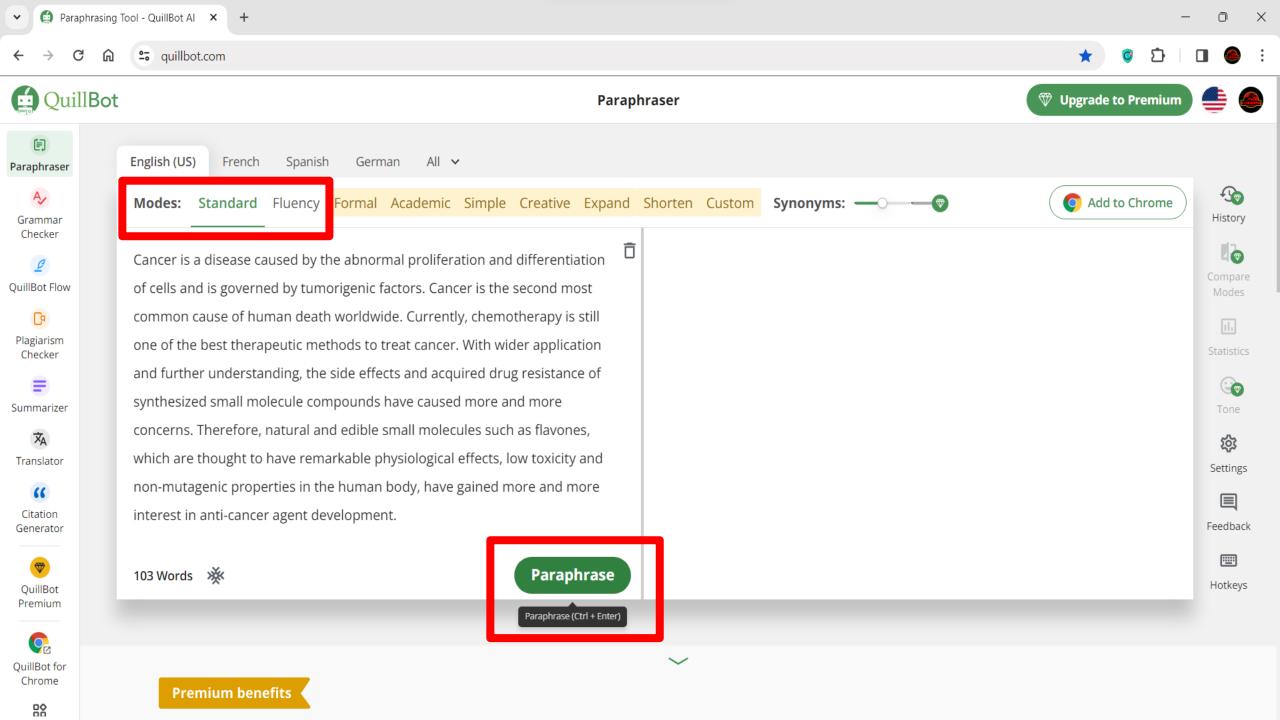
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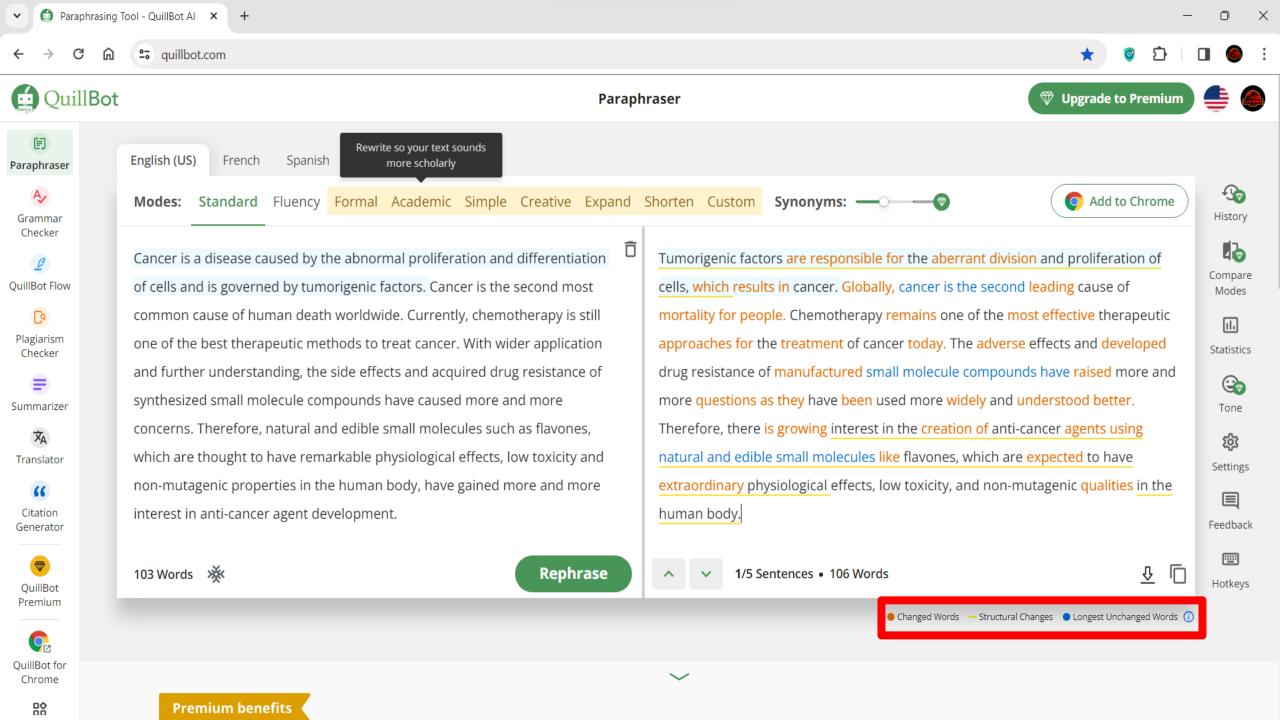




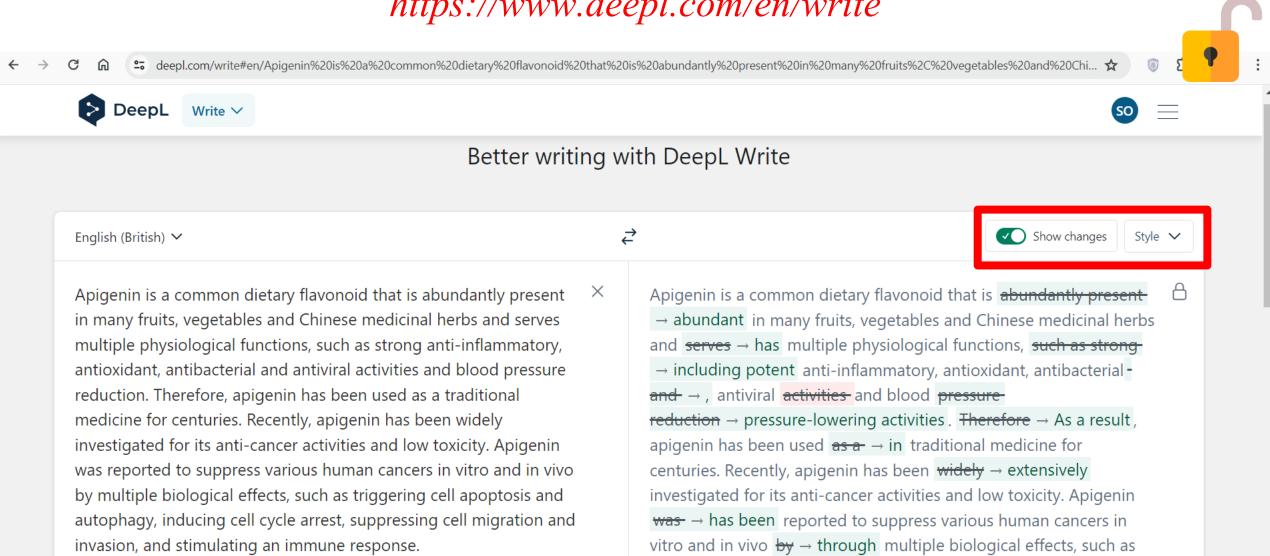
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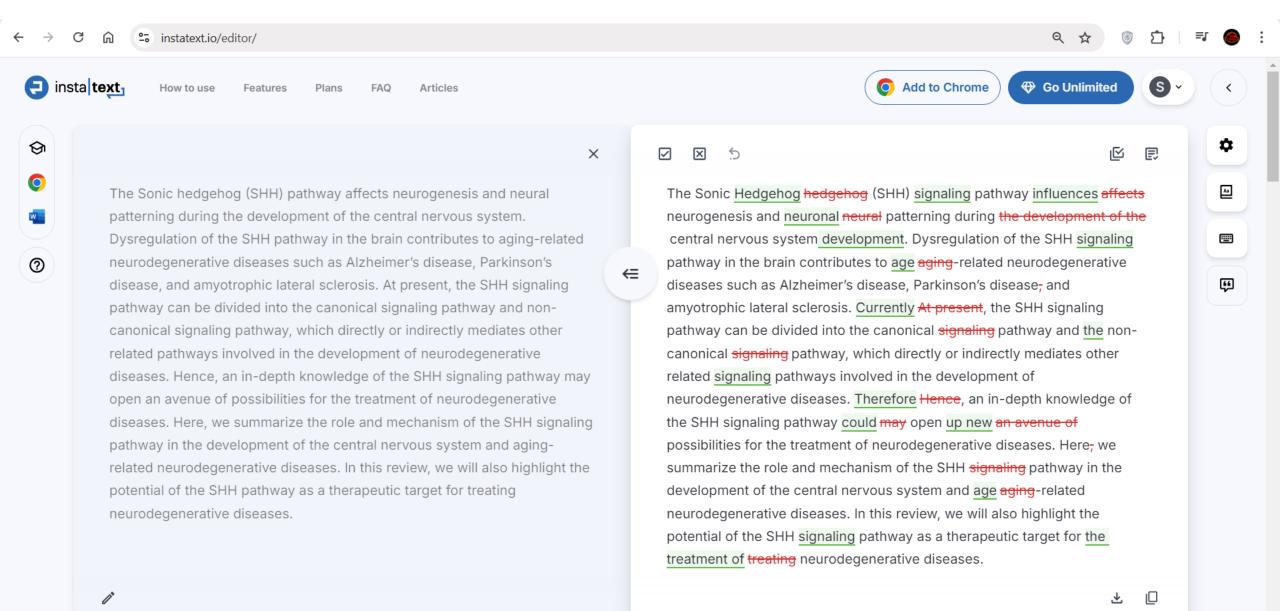


triggering → inducing cell apoptosis and autophagy, inducing cell cycle arrest, suppressing cell migration and invasion, and stimulating



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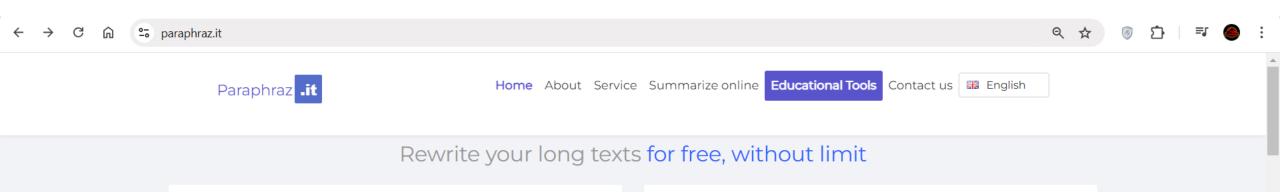


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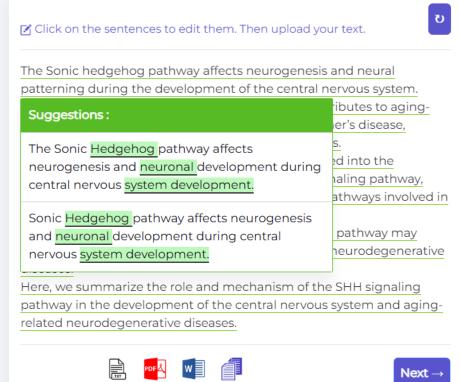
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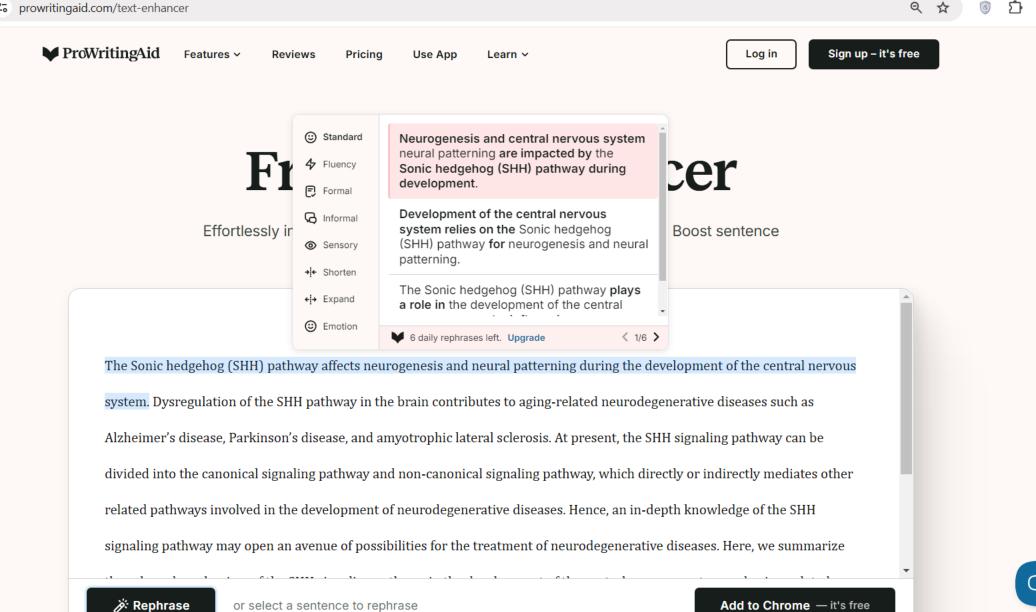
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Here, we summarize the role and mechanism of the SHH signaling pathway in the development of the central nervous system and aging-related neurodegenerative diseases.

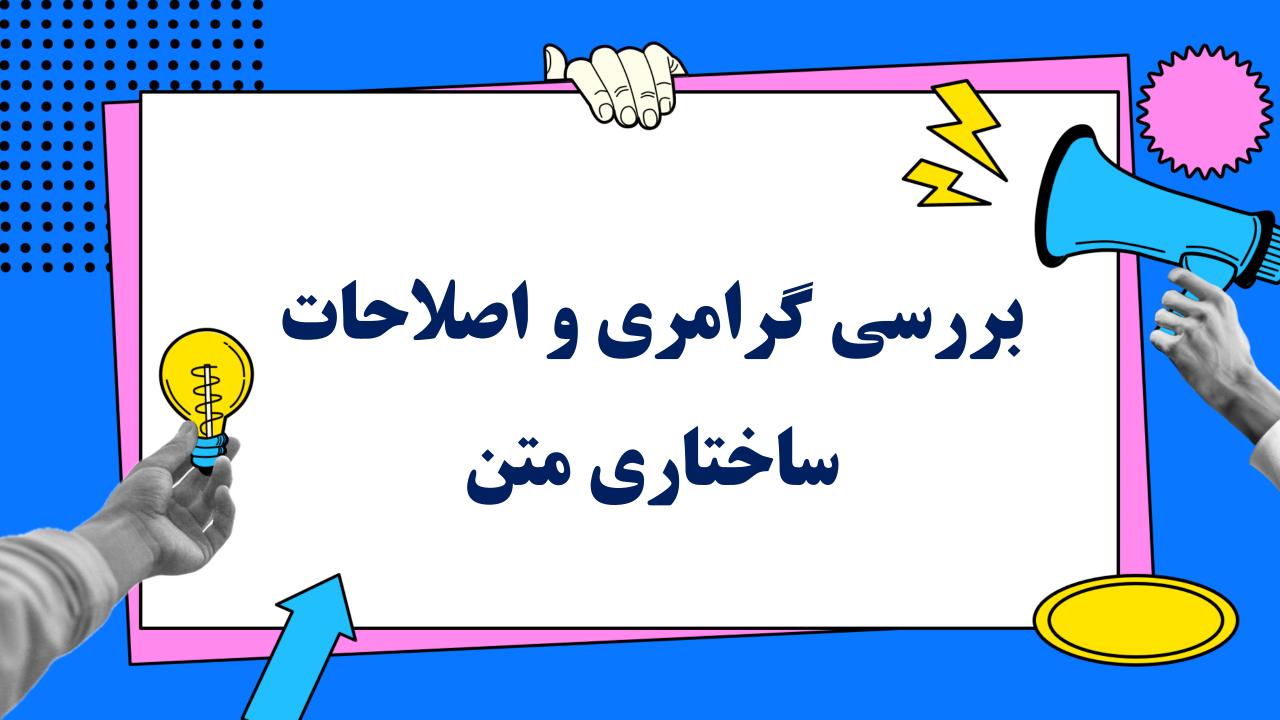


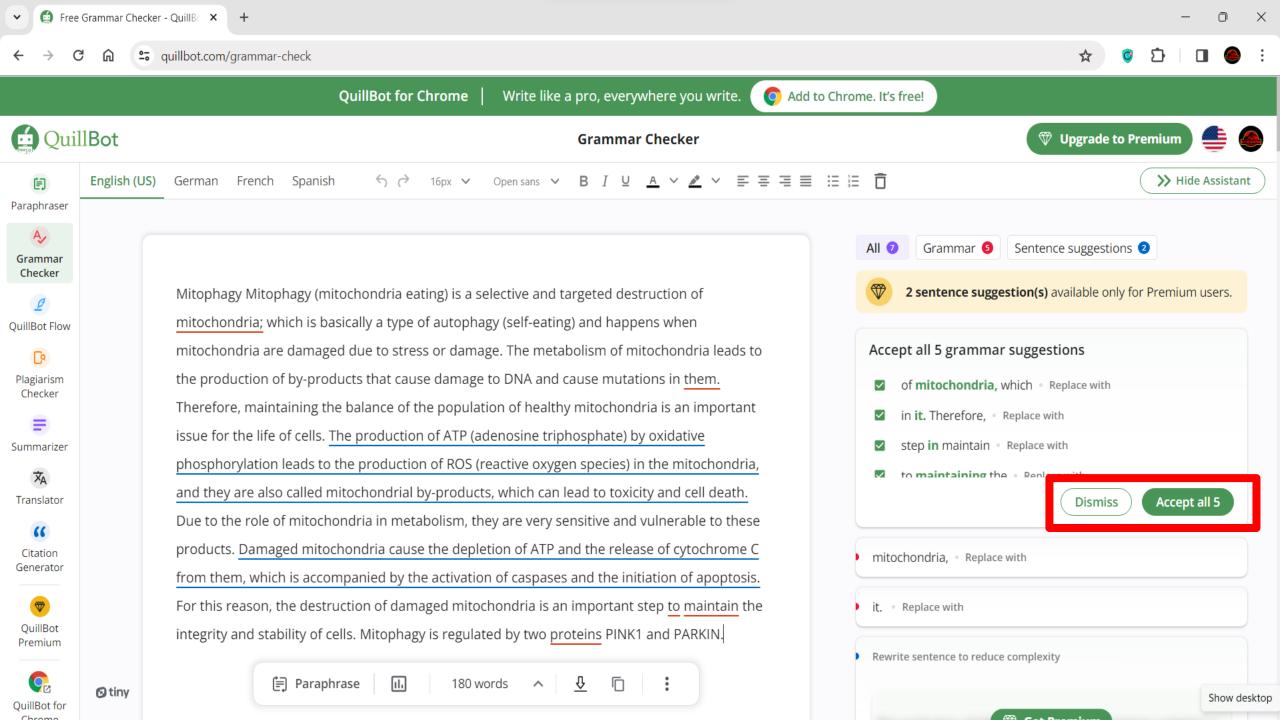
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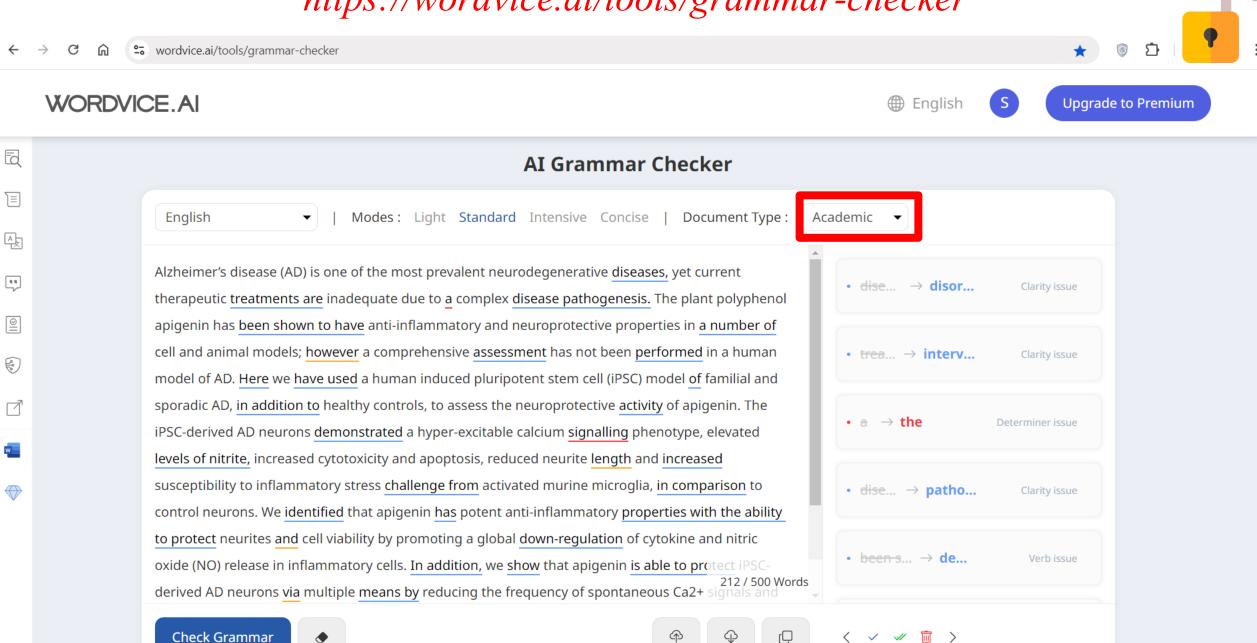








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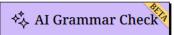




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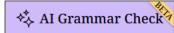
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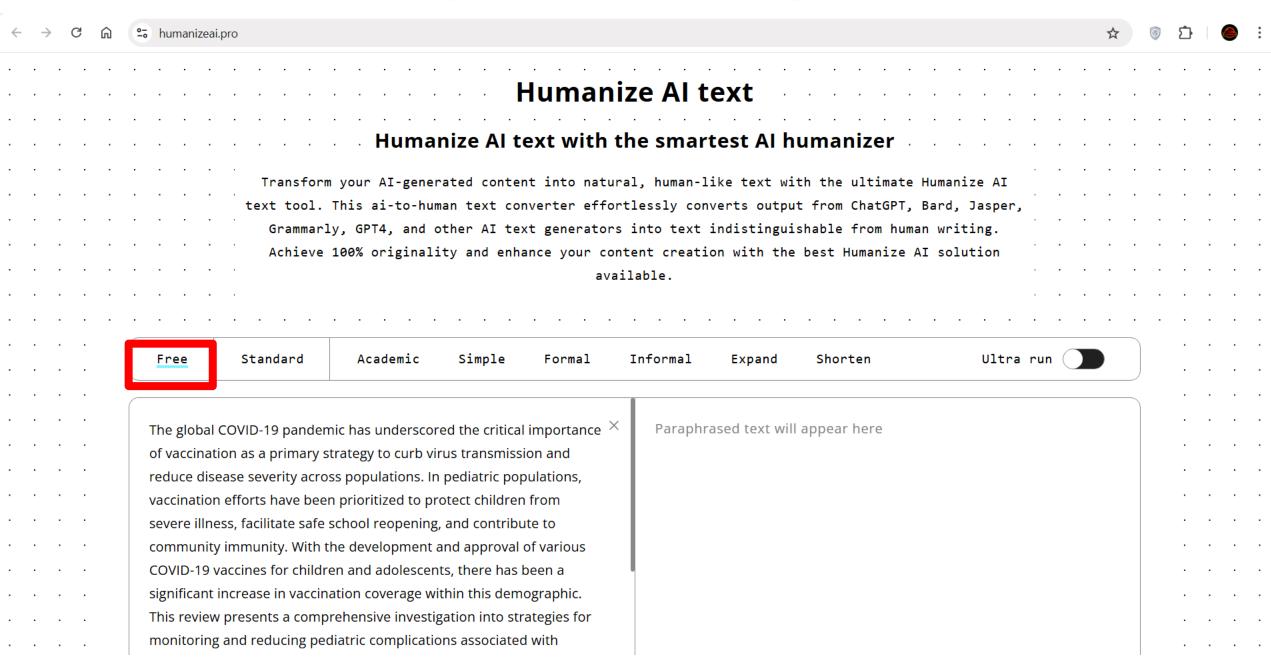


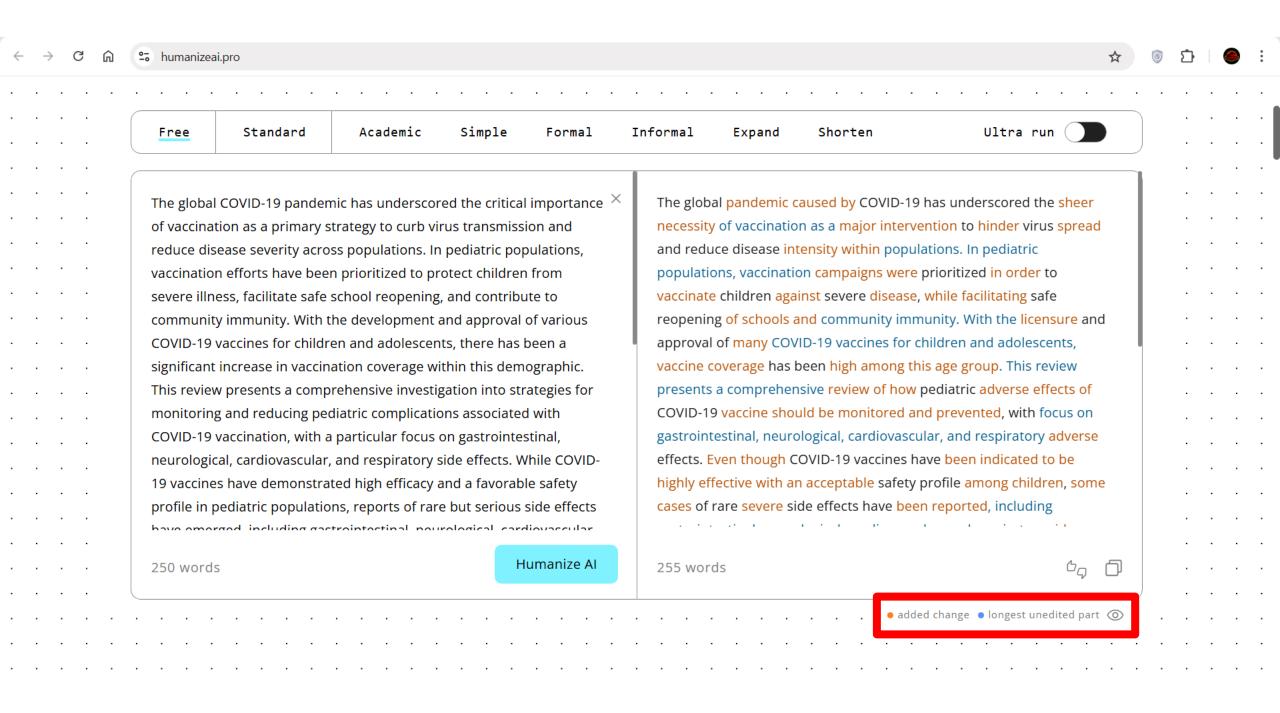


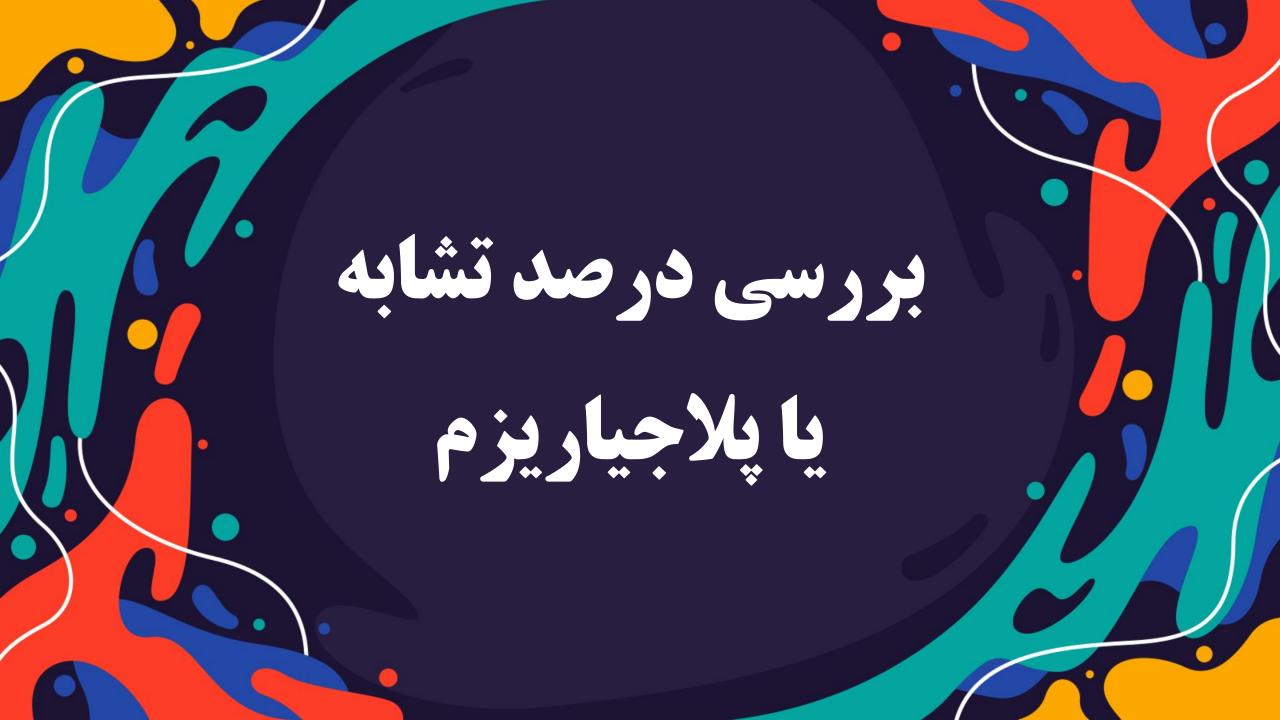




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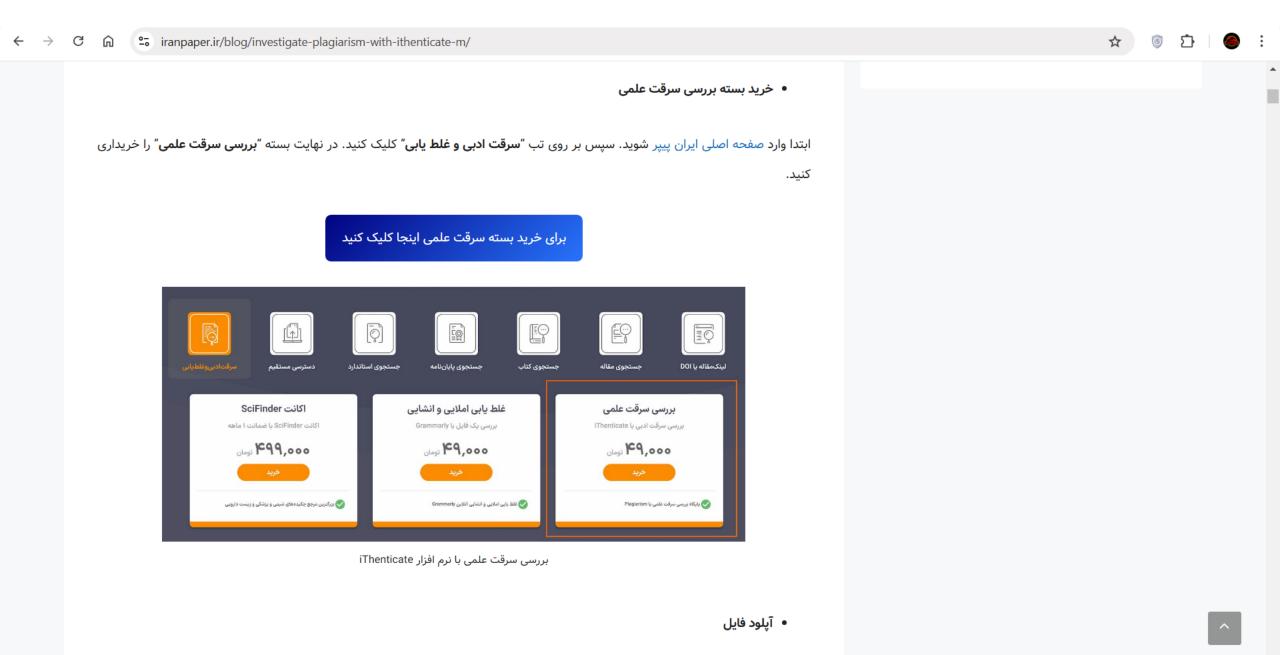
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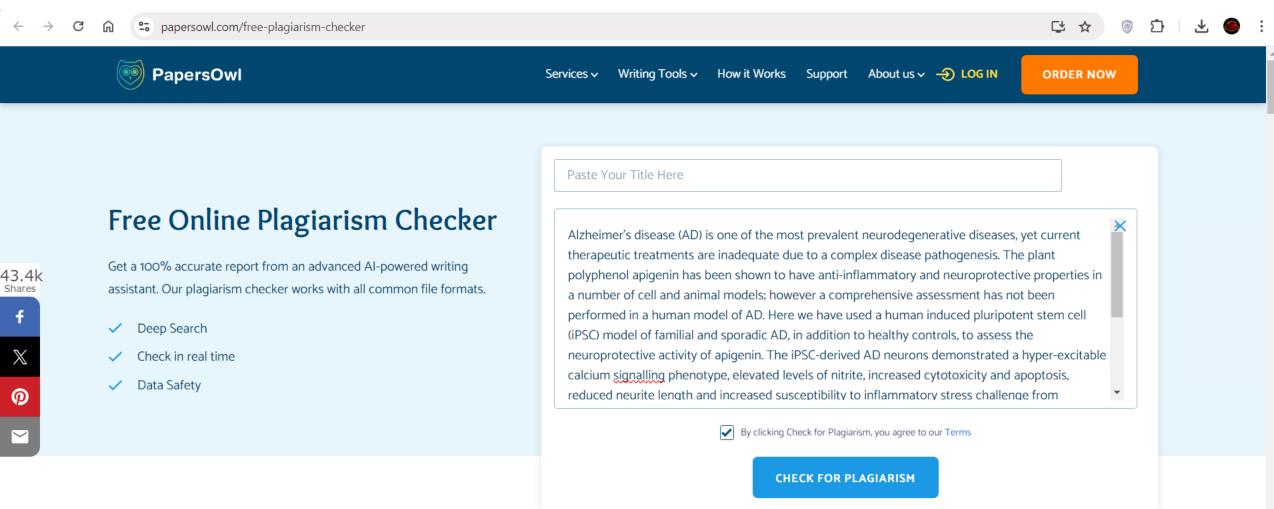
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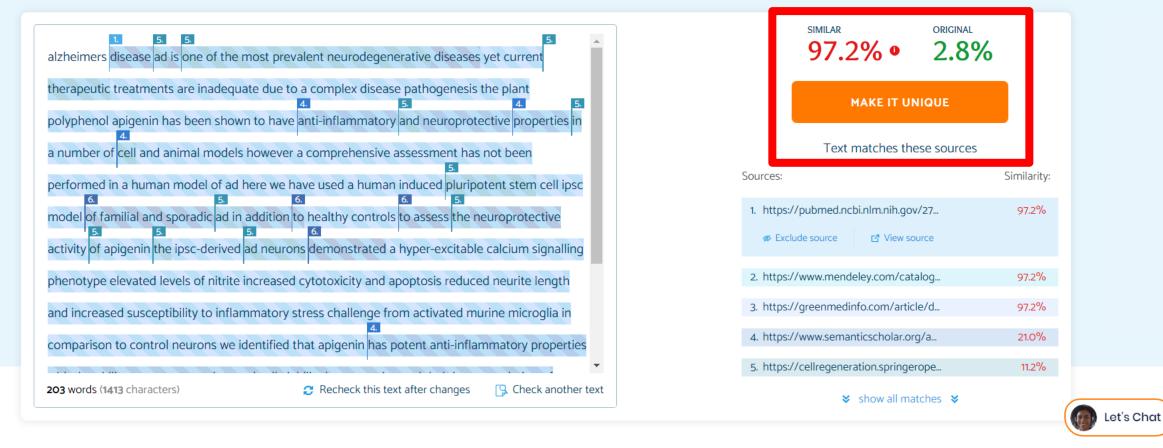


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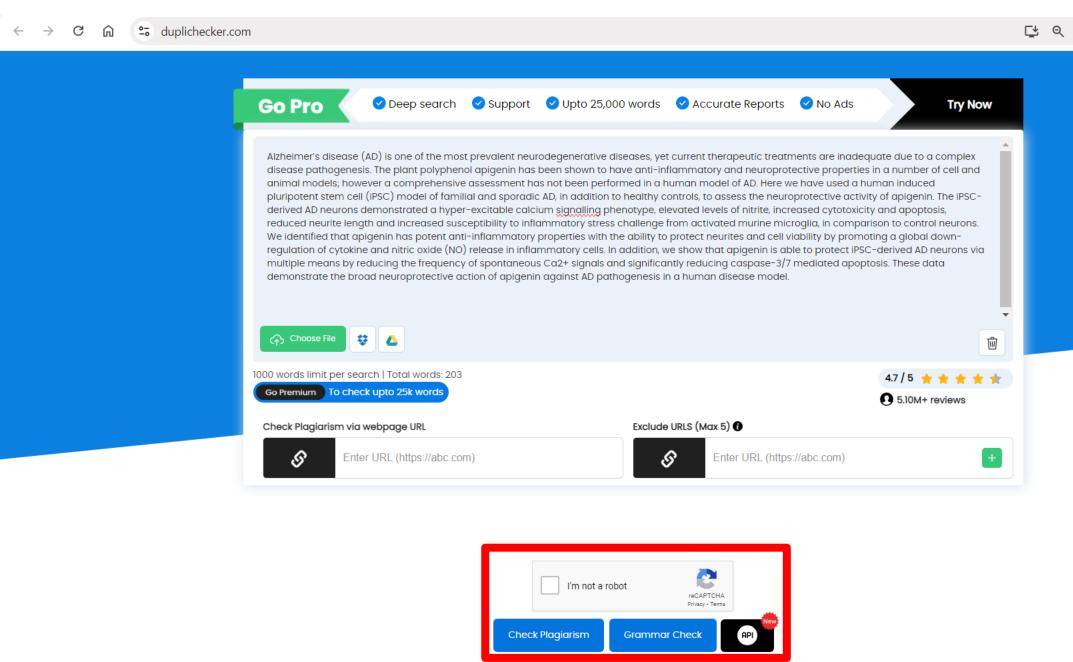




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Alzheimer's disease (AD) is one of the most prevalent neurodegenerative diseases, yet current therapeutic treatments are inadequate due to a complex disease pathogenesis. The plant polyphenol apigenin has been shown to have anti-inflammatory and neuroprotective properties in a number of cell and animal models; however a comprehensive assessment has not been performed in a human model of AD. Here we have used a human induced pluripotent stem cell (iPSC) model of familial and sporadic AD, in addition to healthy controls, to assess the neuroprotective activity of apigenin. The iPSC-derived AD neurons demonstrated a hyper-excitable calcium signalling phenotype, elevated levels of nitrite, increased cytotoxicity and apoptosis, reduced neurite length and increased susceptibility to inflammatory stress challenge from activated murine microglia, in comparison to control neurons. We identified that apigenin has potent anti-inflammatory properties with the

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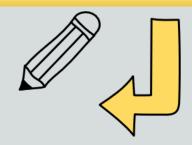
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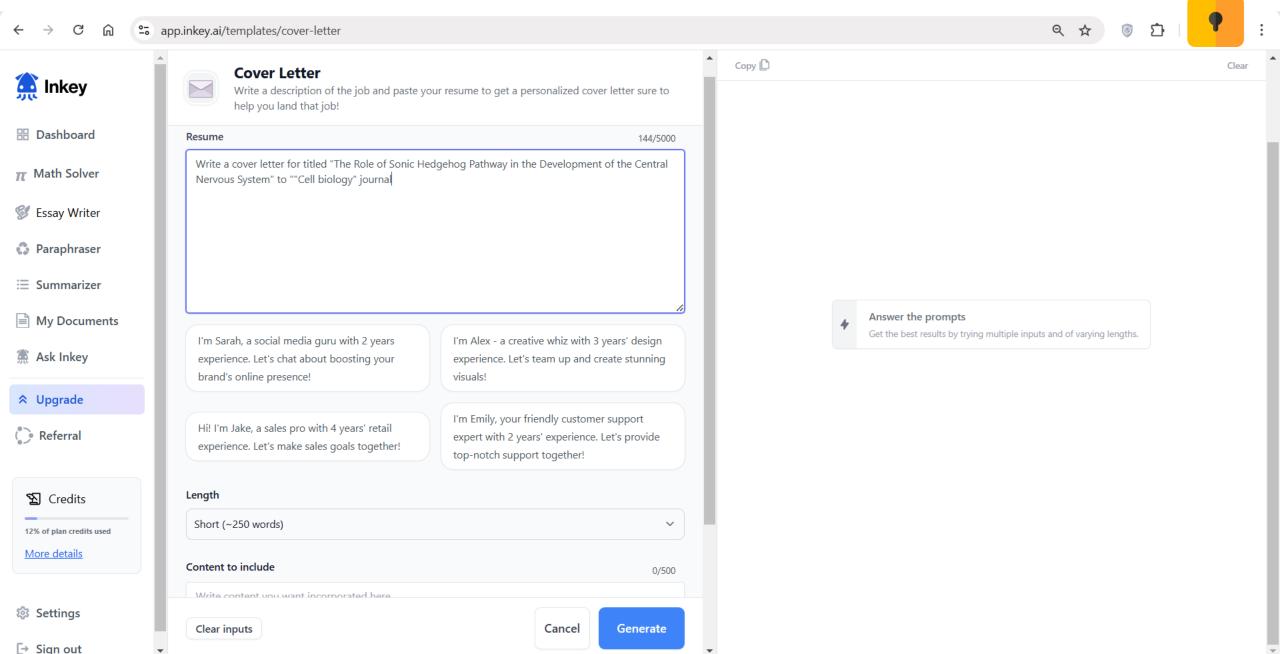


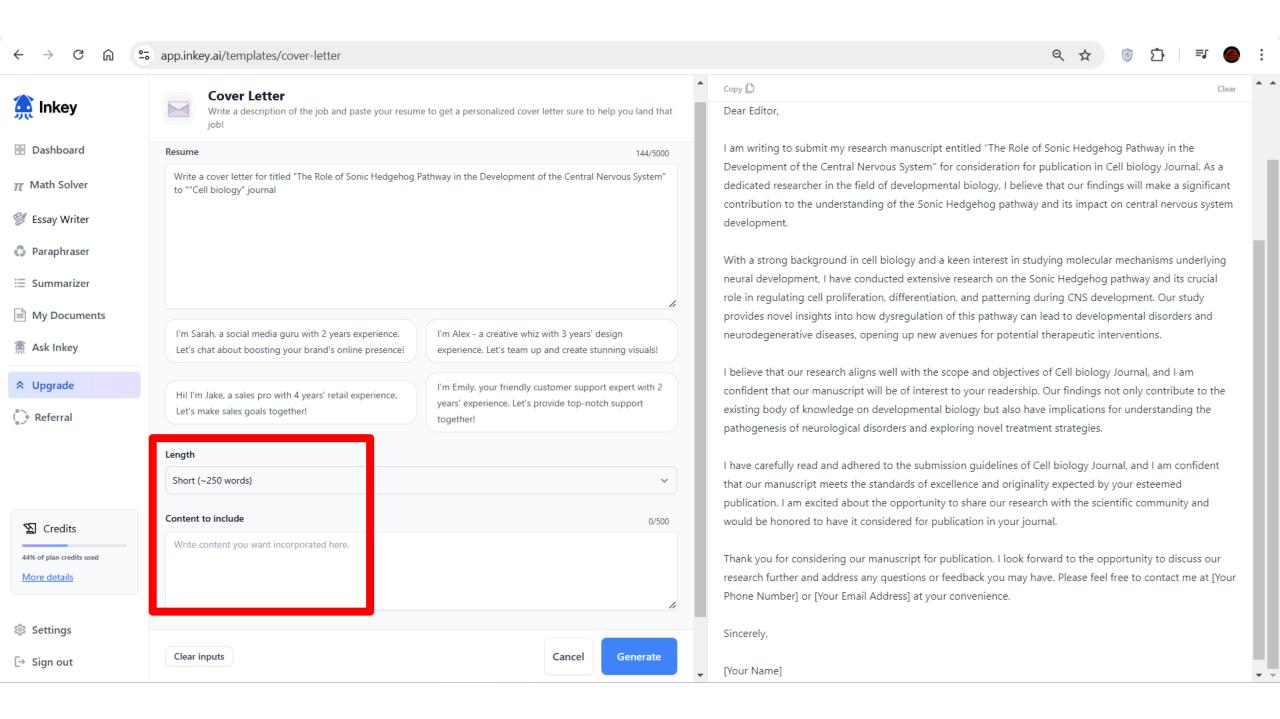


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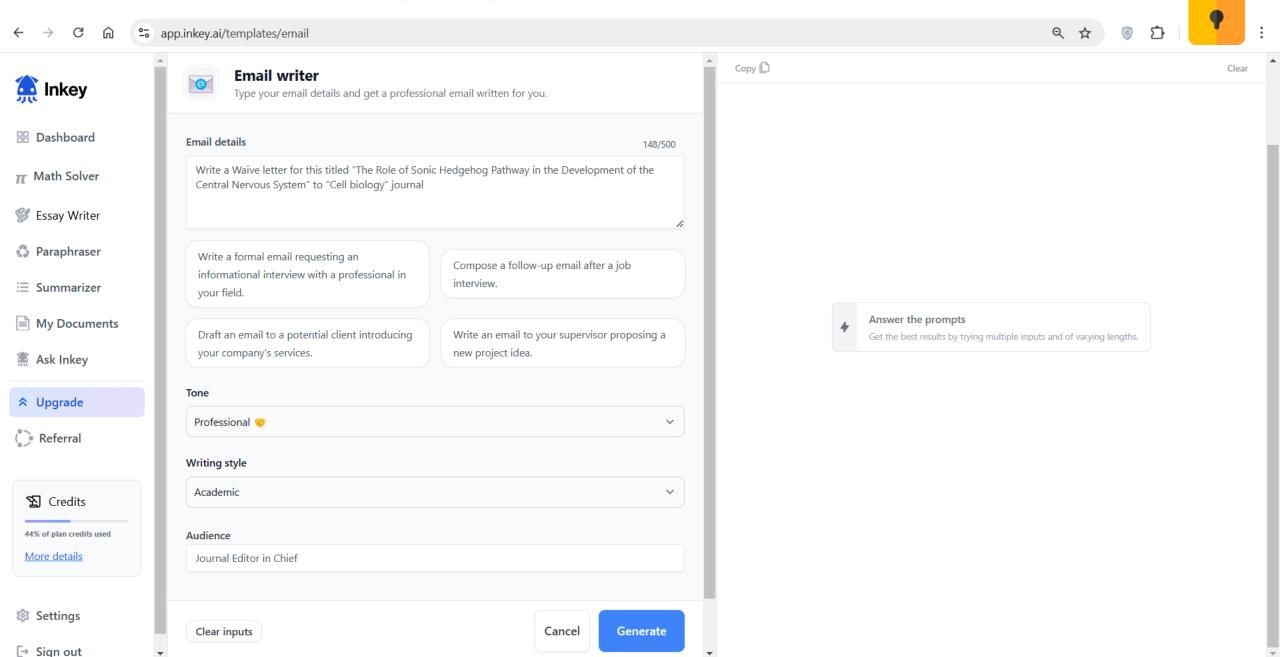


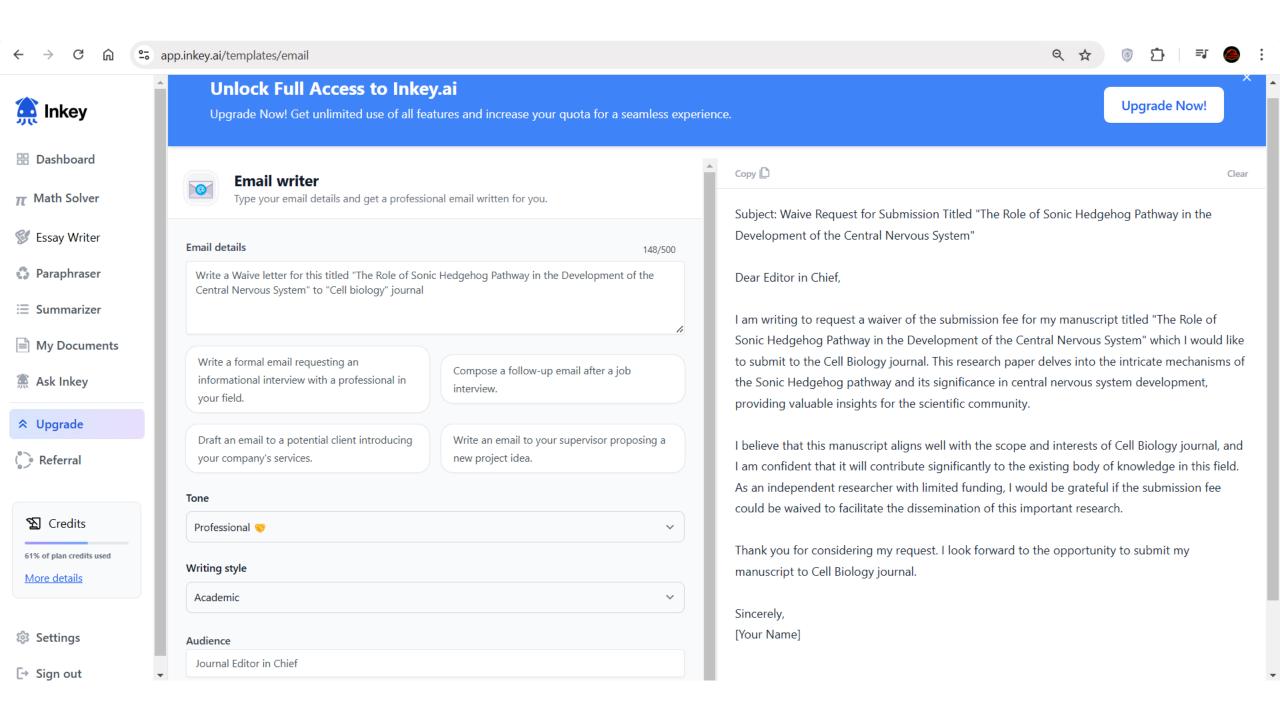
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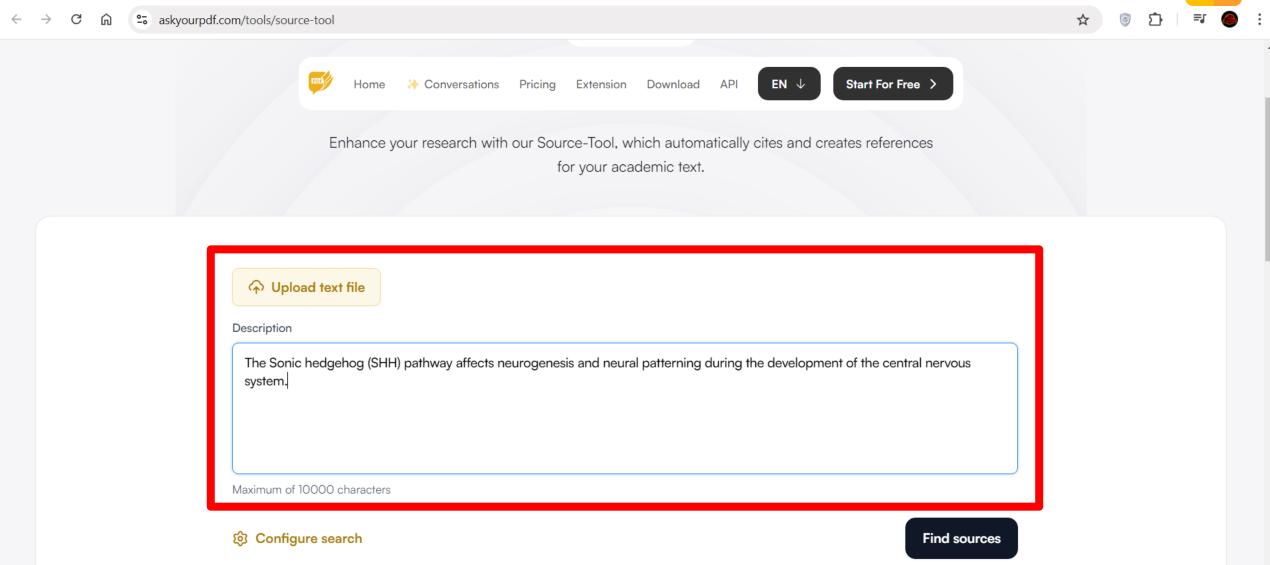


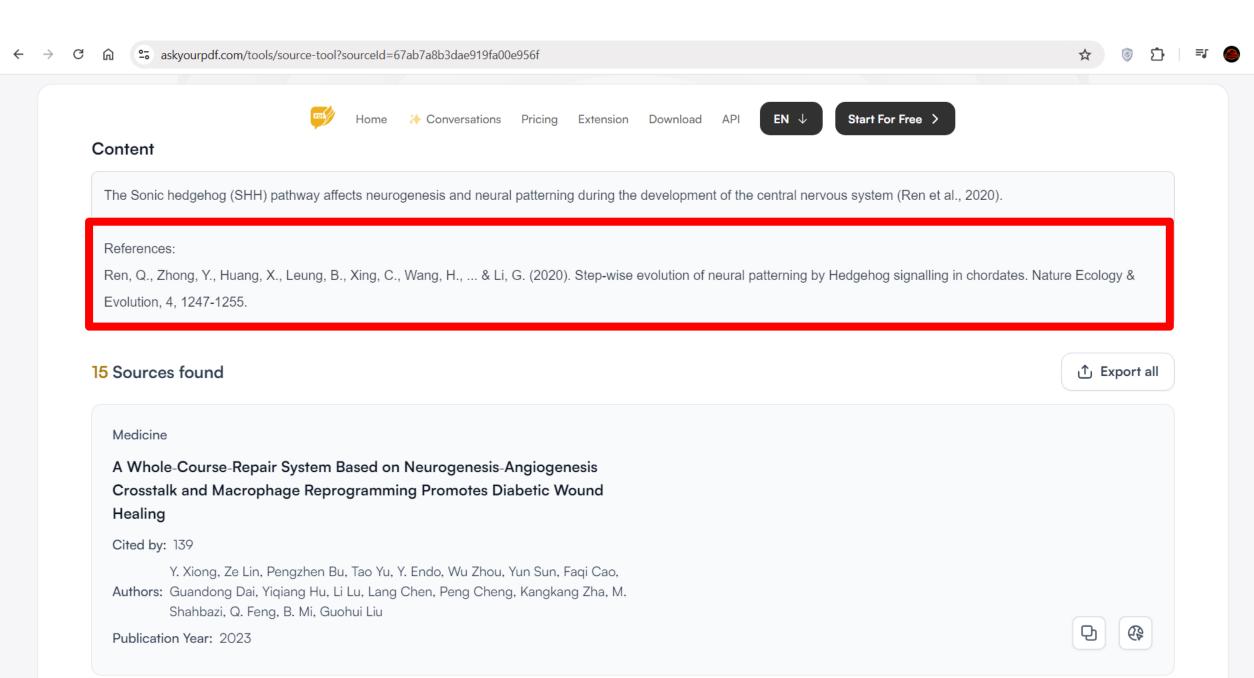




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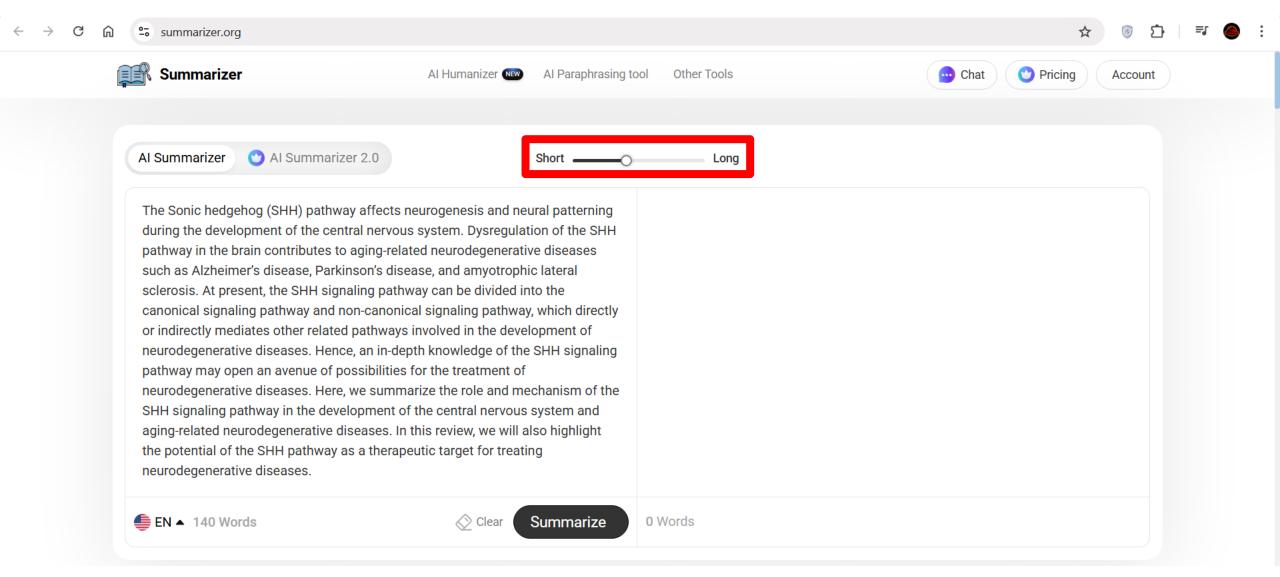


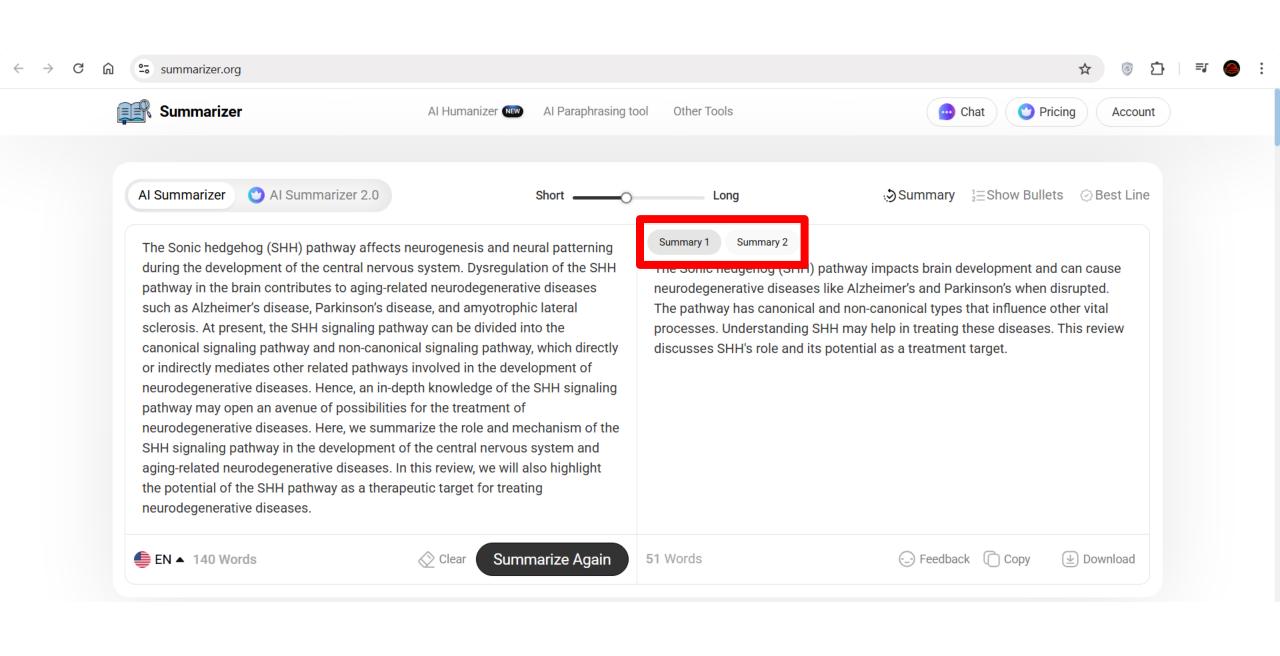




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Apigenin is a common dietary flavonoid that is abundantly present in many fruits, vegetables and Chinese medicinal herbs and serves multiple physiological functions, such as strong antiinflammatory, antioxidant, antibacterial and antiviral activities and blood pressure reduction. Therefore, apigenin has been used as a traditional medicine for centuries. Recently, apigenin has been widely investigated for its anti-cancer activities and low toxicity. Apigenin was reported to suppress various human cancers in vitro and in vivo by multiple biological effects, such as triggering cell apoptosis and autophagy, inducing cell cycle arrest, suppressing cell migration and invasion, and stimulating an immune response. In this review, we focus on the most recent advances in the anti-cancer effects of apigenin and their underlying mechanisms, and we summarize the signaling pathways modulated by apigenin, including the PI3K/AKT, MAPK/ERK, JAK/STAT, NF-κB and Wnt/β-catenin pathways.





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Characters

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Apigenin is a common dietary flavonoid that is abundantly present in many fruits, vegetables and Chinese medicinal herbs and serves multiple physiological functions, such as strong antiinflammatory, antioxidant, antibacterial and antiviral activities and blood pressure reduction. Therefore, apigenin has been used as a traditional medicine for centuries. Recently, apigenin has been widely investigated for its anti-cancer activities and low toxicity. Apigenin was reported to suppress various human cancers in vitro and in vivo by multiple biological effects, such as triggering cell apoptosis and autophagy, inducing cell cycle arrest, suppressing cell migration and invasion, and stimulating an immune response. In this review, we focus on the most recent advances in the anti-cancer effects of apigenin and their underlying mechanisms, and we summarize the signaling pathways modulated by apigenin, including the PI3K/AKT, MAPK/ERK, JAK/STAT, NF-κB and Wnt/β-catenin pathways.

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#### Summary

Apigenin is a flavonoid found in fruits, vegetables, and Chinese herbs with various health benefits, including anti-inflammatory, antioxidant, antibacterial, and antiviral properties. It has been used in traditional medicine for centuries and is now being studied for its potential anticancer effects. Apigenin has shown promise in suppressing various human cancers by inducing cell death, cell cycle arrest, and immune responses. It modulates several signaling pathways involved in cancer development, such as PI3K/AKT, MAPK/ERK, JAK/STAT, NF-κB, and Wnt/βcatenin.

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l	163 Syllables
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	Top keywords
ı	apigenin

77

Words

Top keywords	1-word	2-word	3-word	
apigenin			2	2.5%
cell			2	2.5%
various			2	2.5%
anti-cancer			1	1.2%
anti-inflammatory			1	1.2%
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The Sonic Hedgehog (SHH) signaling pathway plays a crucial role in influencing neurogenesis and neural patterning throughout the intricate process of central nervous system development. This pathway is essential for the proper formation and organization of neural structures, guiding the differentiation of neural progenitor cells and ensuring that they develop into the appropriate types of neurons and glial cells. By regulating various genetic and molecular mechanisms, the SHH pathway helps to establish the precise spatial and temporal patterns necessary for the formation of a functional and well-structured central nervous system.









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The Sonic hedgehog (SHH) pathway affects neurogenesis and neural patterning during the development of the central nervous system.

The Sonic hedgehog (SHH) pathway plays a crucial role in guiding the intricate processes of neurogenesis and neural patterning that take place throughout the development of the central nervous system. During this intricate developmental phase, SHH signaling acts as a master regulator, orchestrating the formation of diverse neuronal cell types and ensuring the precise arrangement and connectivity of these cells within the complex neural networks. By exerting its influence at various stages of CNS development, the SHH pathway has a profound impact on shaping the architecture and function of the brain and spinal cord. Through a series of intricate molecular interactions and signaling cascades, SHH helps to coordinate the proliferation, differentiation, and migration of neural progenitor cells, ultimately contributing to the establishment of

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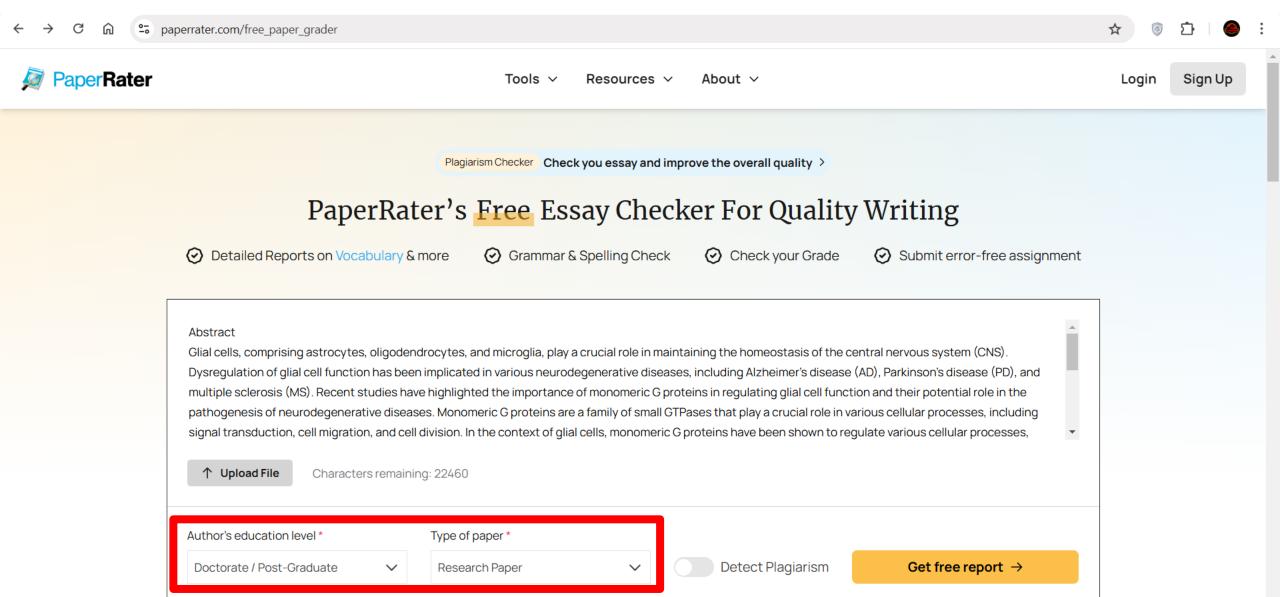
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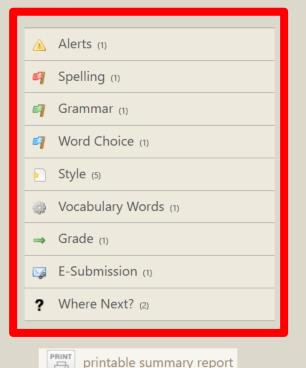




Glial cells, comprising astrocytes, oligodendrocytes, and microglia, play a crucial role in maintaining the homeostasis of the central nervous system (CNS). Dysregulation of glial cell function has been implicated in various neurodegenerative diseases, including Alzheimer's disease (AD), Parkinson's disease (PD), and multiple sclerosis (MS). Recent studies have highlighted the importance of monomeric G proteins in regulating glial cell function and their potential role in the pathogenesis of neurodegenerative diseases. Monomeric G proteins are a family of small GTPases that play a crucial role in various cellular processes, including signal transduction, cell migration, and cell division. In the context of glial cells, monomeric G proteins have been shown to regulate various cellular processes, including proliferation, differentiation, and survival. For instance, the Ras family of monomeric G proteins has been implicated in the regulation of astrocyte proliferation and differentiation, while the Rho family has been shown to regulate the migration and survival of oligodendrocytes. One of the key mechanisms by which monomeric G proteins regulate glial cell function is through their ability to interact with various downstream effectors, including protein kinases, phosphatases, and cytoskeletal proteins. The Ras protein has been shown to interact with the Raf kinase, leading to the activation of the MAPK pathway, which regulates astrocyte proliferation and differentiation. Also, inhibition of Ras activity has been shown to reduce the formation of amyloid plaques in AD. However, the available evidence suggests that modulation of monomeric G protein activity may be a promising therapeutic strategy for the treatment of neurodegenerative diseases.



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#### **Word Choice**

1 of 1 < Prev Next >

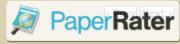
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#### Introduction

Apigenin, a natural flavonoid found in various fruits, vegetables, and herbs, has gained significant attention for its potential cancer-fighting properties. As researchers continue to explore its cellular and molecular effects, apigenin emerges as a compelling compound worthy of investigation in oncology. This review delves into the characteristics of apigenin, its mechanisms of action against cancer cells, and the current understanding of its efficacy and safety.

#### Understanding Apigenin

Apigenin is predominantly found in parsley, celery, chamomile, and various other plants. It is known for its anti-inflammatory, antioxidant, and anticancer properties. Targeting multiple pathways involved in cancer progression, apigenin could be particularly beneficial in inhibiting tumor growth, promoting apoptosis (programmed cell death), and preventing metastasis. It appears to be effective in various cancer types, including breast, colon, prostate, and leukemia, making it a versatile candidate in cancer research.

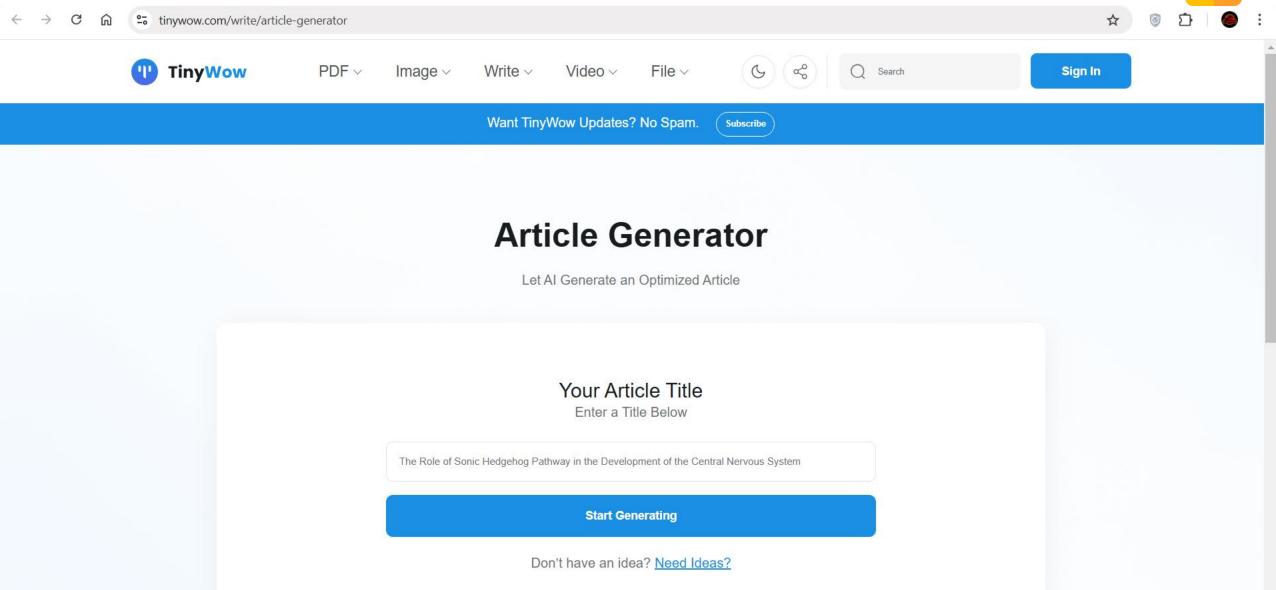
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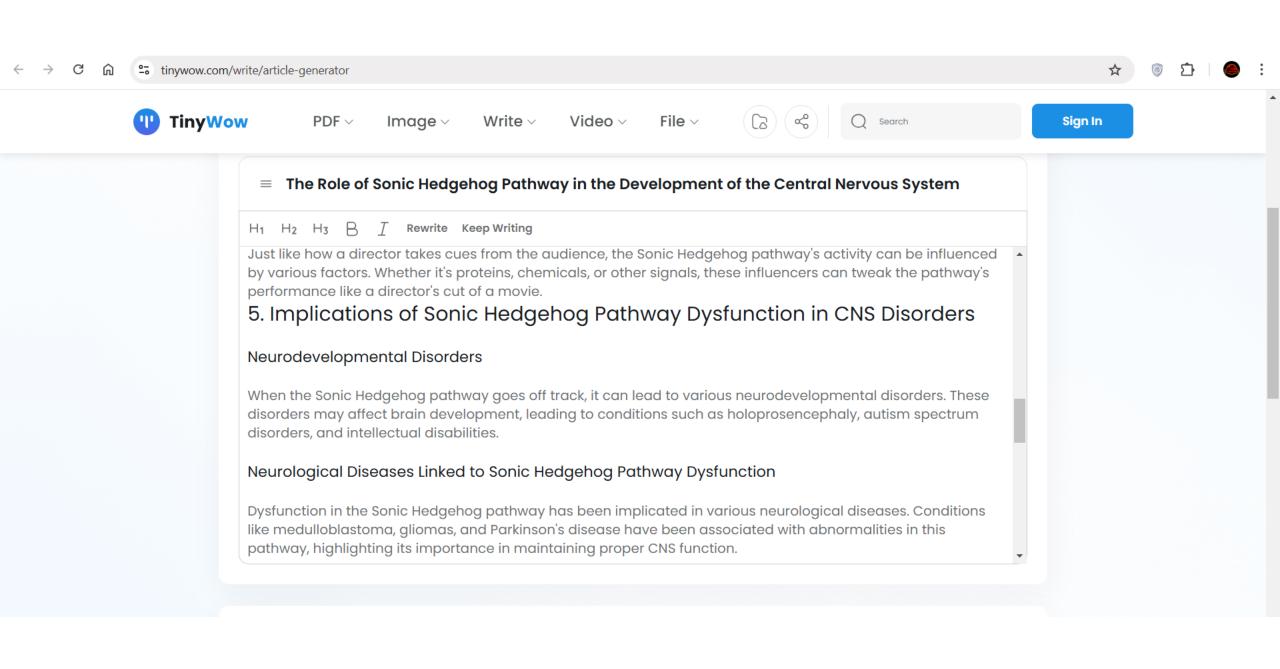




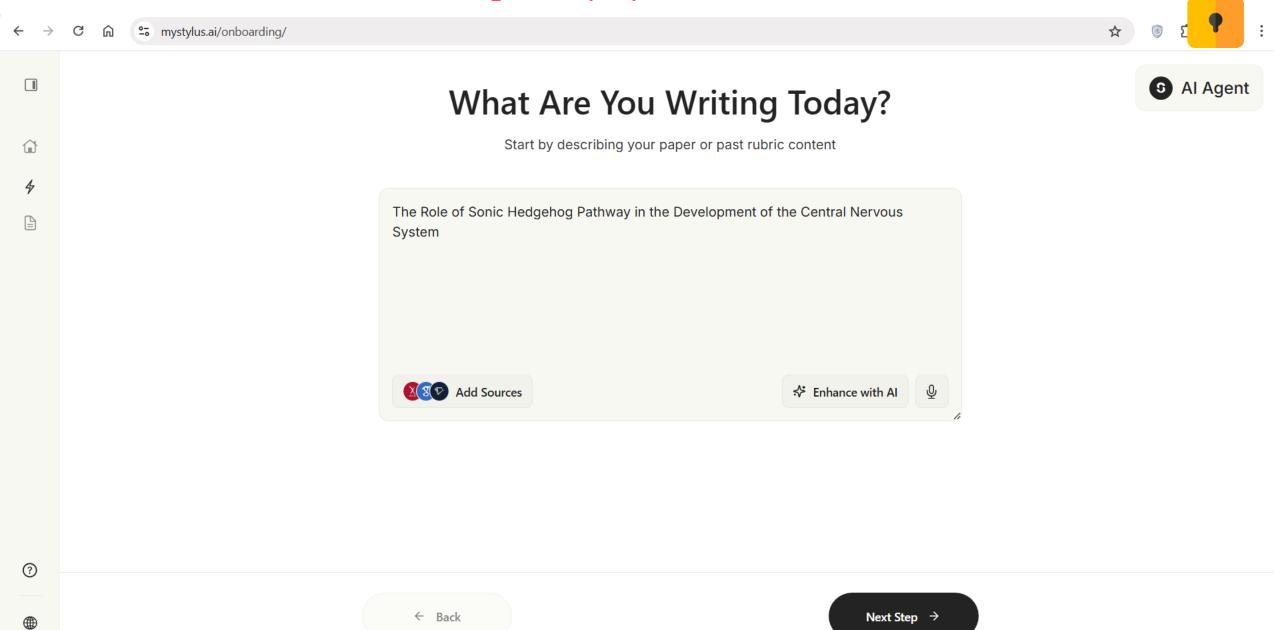
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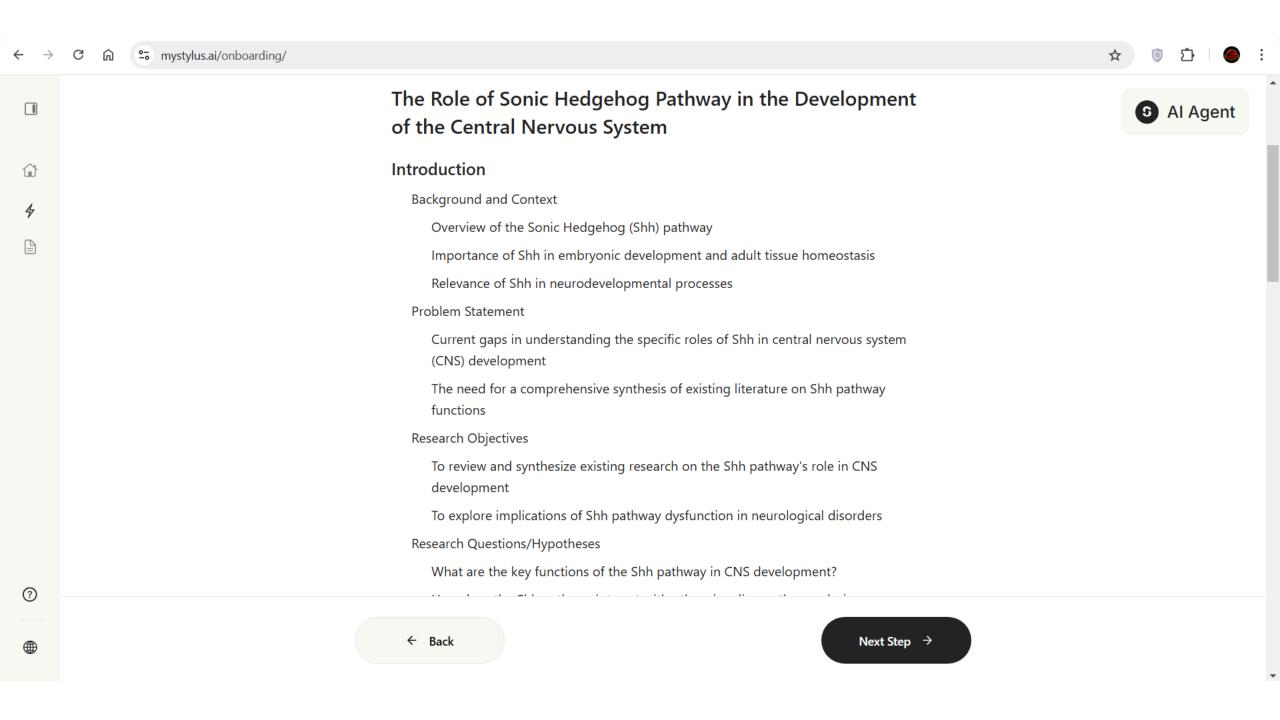


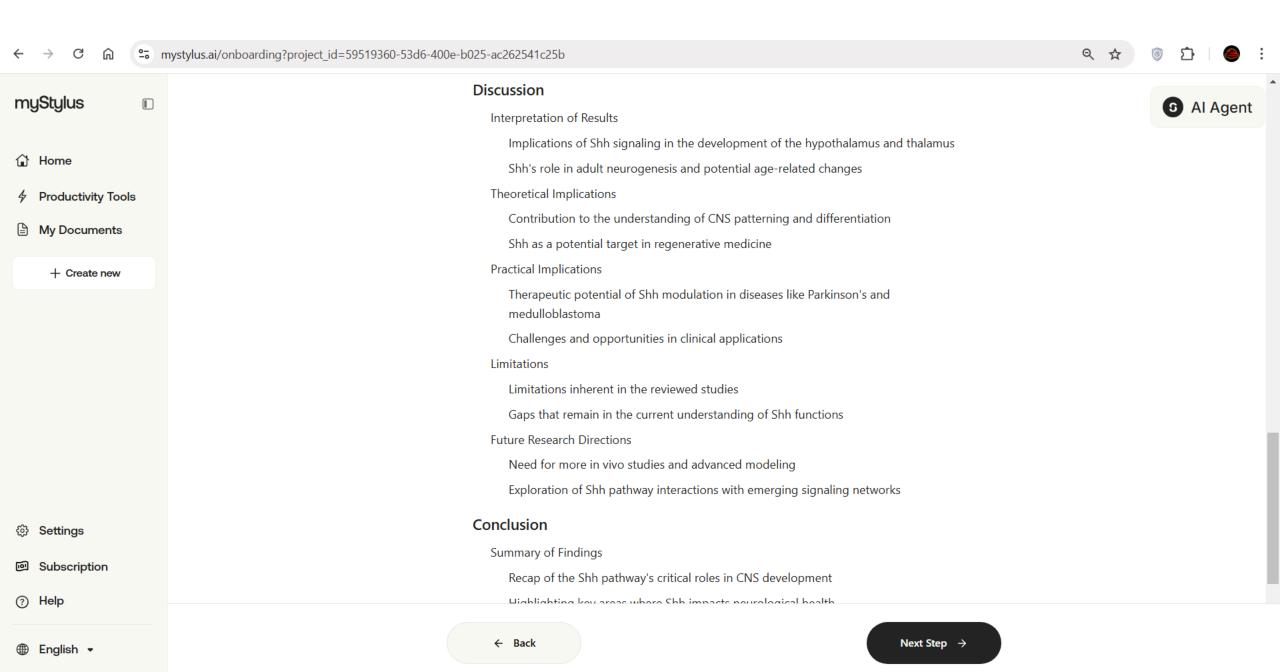


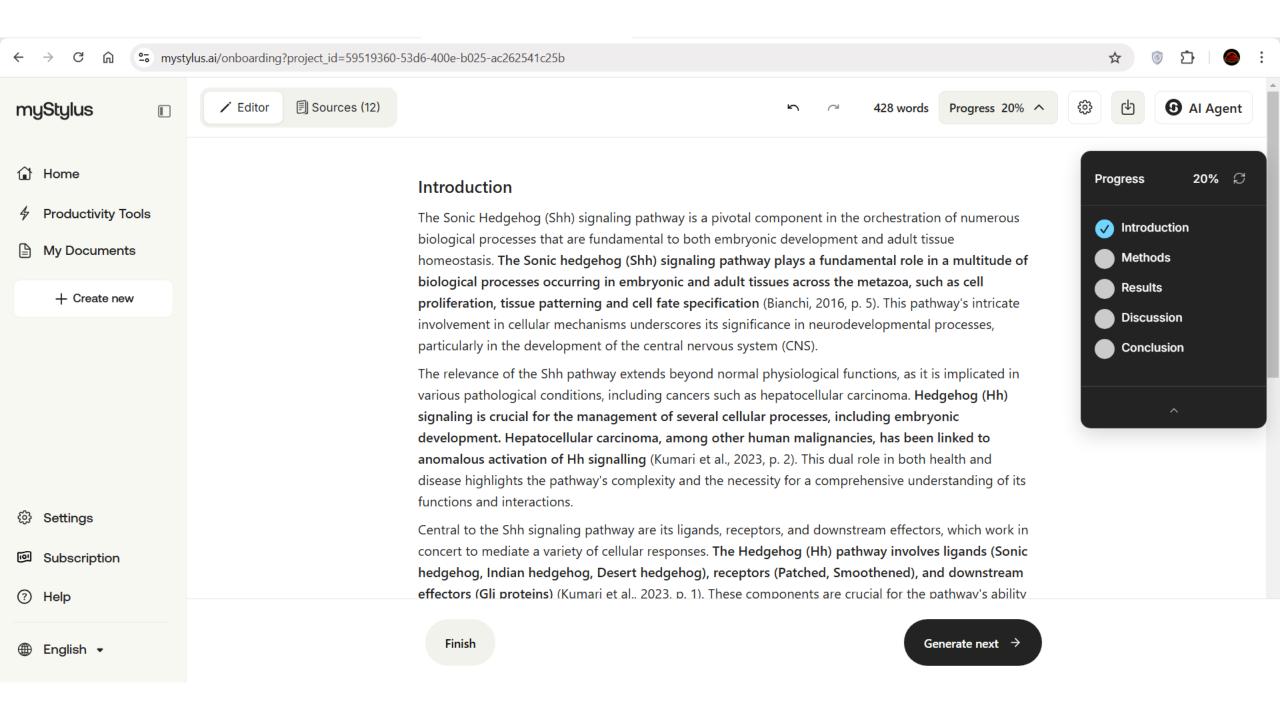


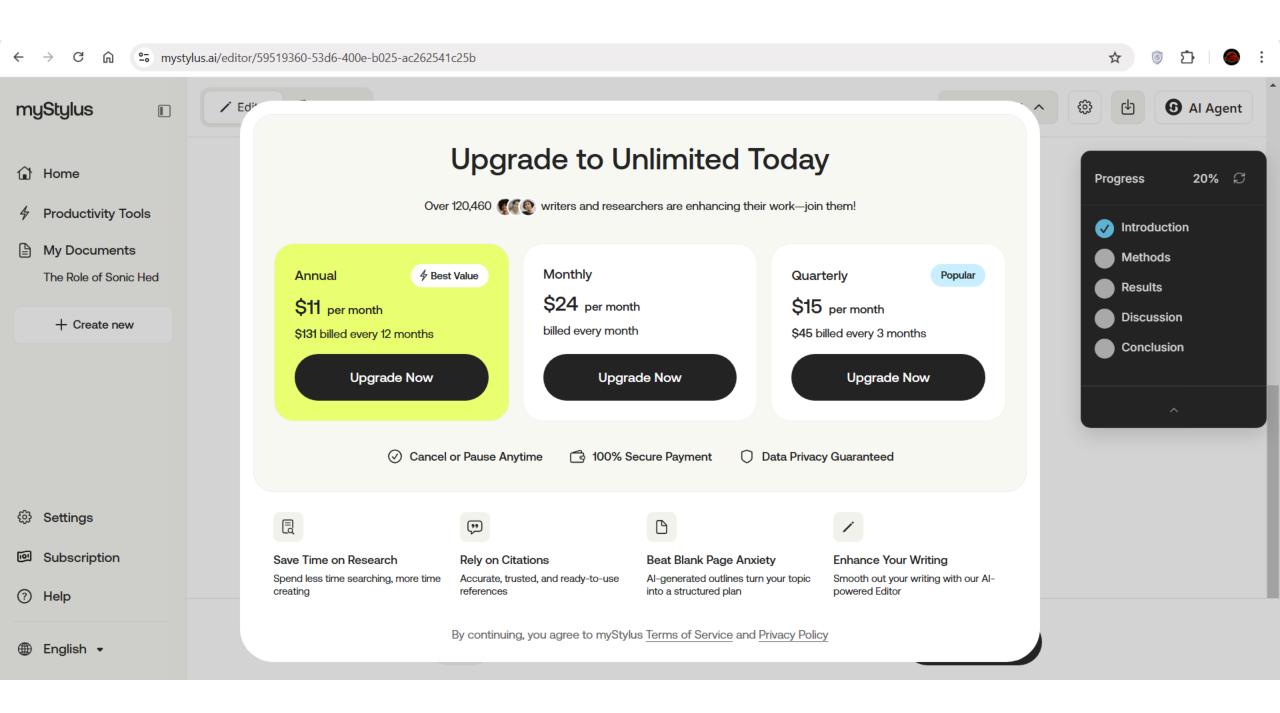
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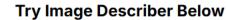
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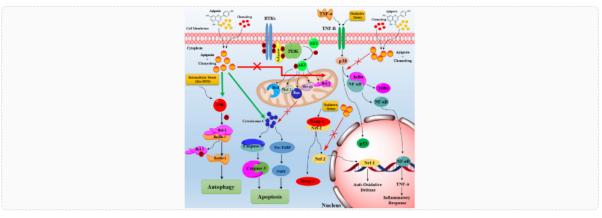




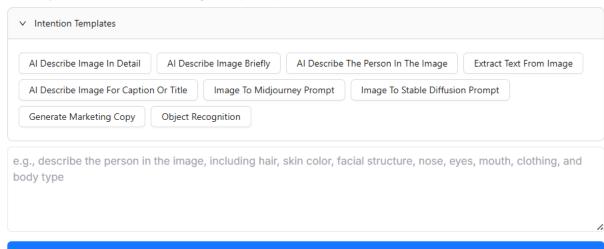




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#### Image describe result

Here is a description of the image:

The image is a detailed diagram illustrating the molecular mechanisms of apigenin and chemodrug in cancer therapy, showing how they impact various cellular pathways and processes.

The diagram is complex, filled with various shapes and arrows representing proteins, genes, and cellular processes. **Key elements include:** 

**Cell Membrane:** Depicted at the top, illustrating the entry of apigenin and chemodrugs into the cell.

**Cytoplasm:** The region between the cell membrane and nucleus, where many signaling pathways occur. Apigenin and chemodrug are shown interacting with various proteins in the cytoplasm.

**Mitochondria:** A large, bean-shaped organelle centrally located, showcasing the release of cytochrome C (a key step in apoptosis)

**Nucleus:** A large, round structure at the bottom, showing how the signaling cascade affects gene expression (Nrf2, NF-κB binding to DNA).

**Proteins & Genes:** Numerous proteins (AKT, PI3K, JNK, p38, NF-κB, Nrf2, Bax, BcI-2, Beclin-1, Caspases, PARP) and genes are illustrated, connected by arrows indicating their interactions and downstream effects. Many of these are labeled with names and phosphorylation symbols (p).

**Pathways:** The diagram displays multiple pathways, including the apoptotic pathway (leading to programmed cell death), autophagy (a cellular self-cleaning process), and inflammatory/anti-oxidative pathways, along with their regulation by apigenin and chemodrug.

**Oxidative Stress:** Represented by orange stars, this is highlighted as a key element involved in the cellular response. The diagram shows that apigenin and the chemodrug can both mitigate oxidative stress.

**Color-Coding:** Different colors are used to represent different proteins, pathways, and processes, making it easier to follow the complex interactions. Green arrows highlight the main effects of apigenin and chemodrug combinations, red X's primarily denote blocked pathways.

**Overall Atmosphere and Mood:** The diagram is clinical and scientific, aimed at conveying complex biological information rather than evoking an emotional response. It communicates a sense of order and complexity within cellular processes.

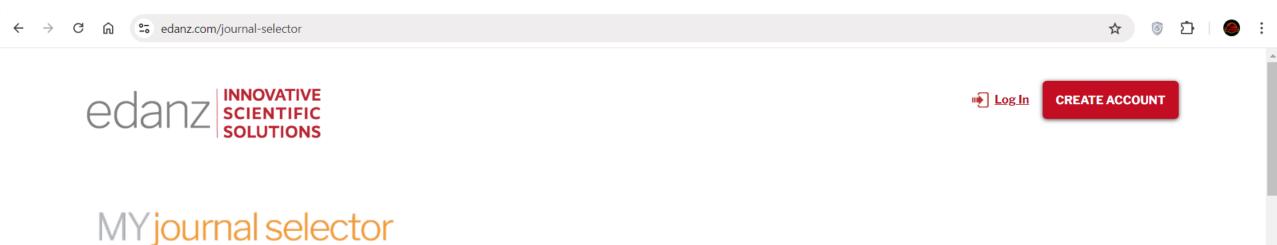




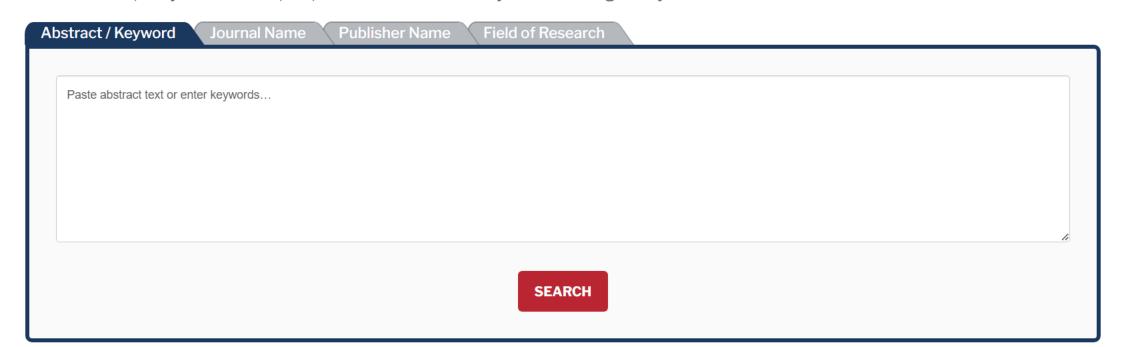


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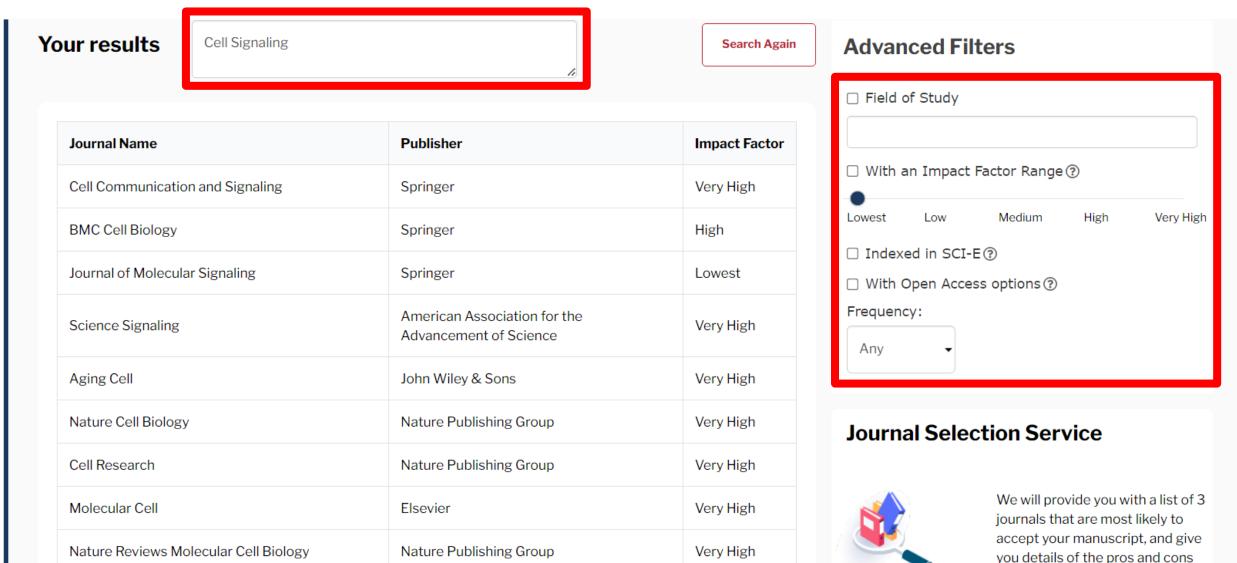
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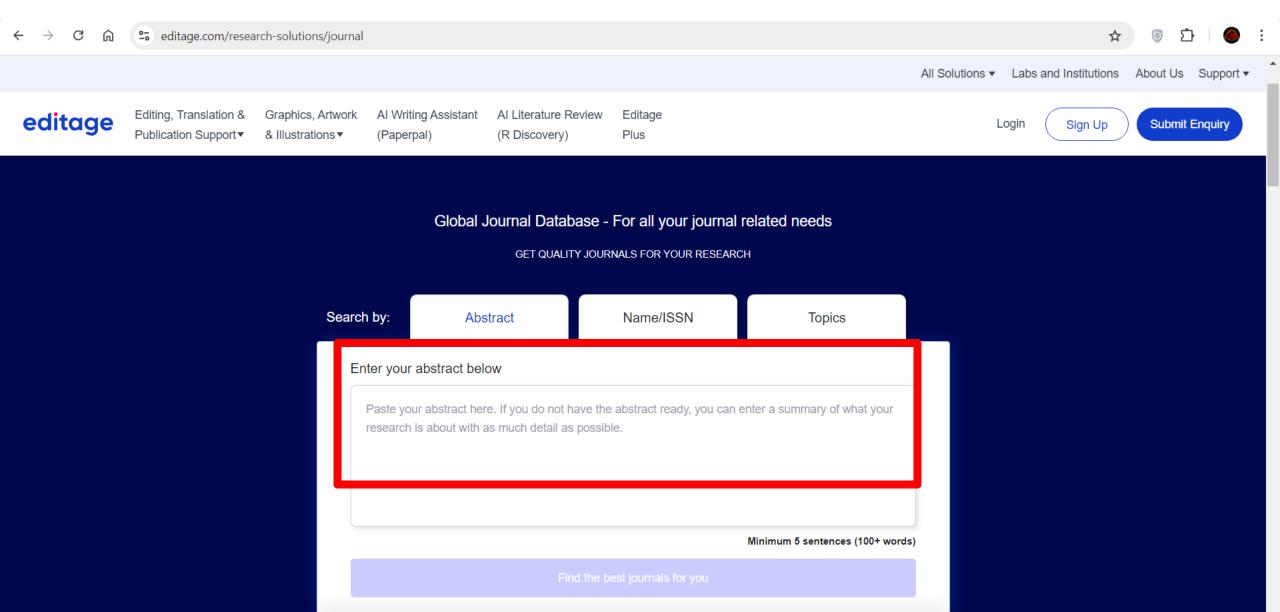






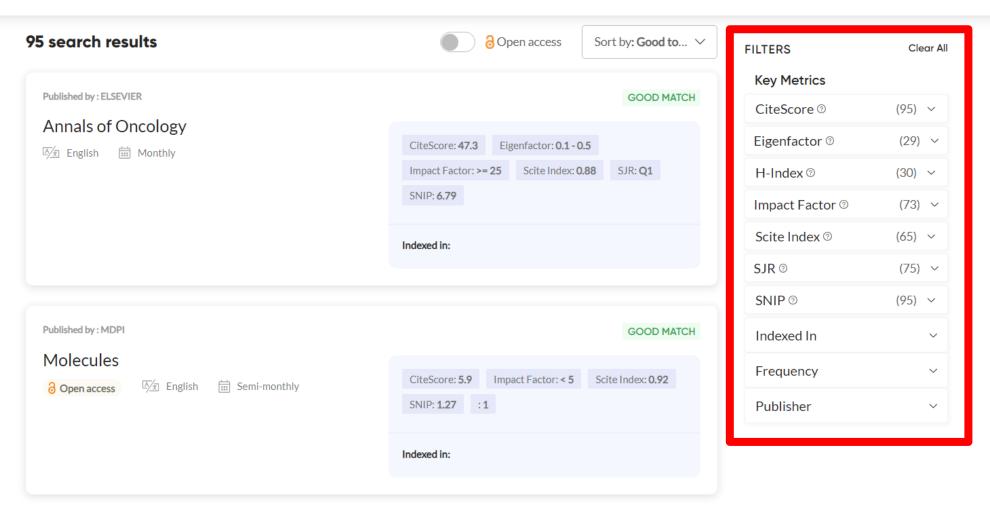
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