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**“WOMEN IN INNOVATION AND SCIENCE”
XALQARO ILMIY-AMALIY KONFERENSIYA VA FORUMI
ILMIY ISHLARI TO‘PLAMI**

Toshkent davlat transport universitetida 2026-yil 27-mart kuni “Women in Innovation and Science” xalqaro forumi ilmiy ishlari to‘plami chop etildi.

Mazkur nufuzli tadbirlar O‘zbekiston Respublikasi Oliy ta’lim, fan va innovatsiyalar vazirligi hamda Transport vazirligi hamkorligida tashkil etilib, mamlakatimizda ilm-fan, innovatsiyalar va zamonaviy texnologiyalarni rivojlantirish, shuningdek xalqaro ilmiy hamkorlikni yanada kengaytirishga qaratilgan muhim platforma hisoblanadi.

Konferensiya ochilish marosimida davlat organlari, xalqaro tashkilotlar va yetakchi oliy ta’lim muassasalari vakillari ishtirok etdi. Jumladan, Toshkent davlat transport universiteti rektori G‘ulomov Abdulaziz Abdullayevich, O‘zbekiston Respublikasi Transport vazirligi vakili Ulmasova Lobarkhon Adilovna, Osiyo taraqqiyot banki eksperti Claire Charnac, Istanbul Aydin universiteti vasiylik kengashi raisi Mustafa Aydin, Belarus davlat transport universiteti rektori Kazakov Nikolay Nikolayevich hamda Qirg‘iziston Respublikasi Transport vazirligi vakili Dinara Moldokalieva ishtirok etdilar.

Konferensiya ilmiy dasturi doirasida plenar va panel sessiyalar, shuningdek ilmiy ma’ruzalar taqdimoti o‘tkazilib, ularda sun’iy intellekt va raqamli texnologiyalarni iqtisodiyot va ta’limga joriy etish, transport va aviatsiya sohasida innovatsion yechimlar, sanoat va IT sohasida texnologiya transferi, shuningdek ta’limda raqamli platformalar va intellektual tizimlar kabi dolzarb masalalar yoritildi.

2026-yil 27-mart kuni o‘tkazilgan “Women in Innovation and Science” xalqaro forumi ayollar yetakchiligi, innovatsiyalar va STEM sohalariga bag‘ishlangan bo‘lib, mamlakatimizda ayollarning ilm-fan va texnologiya sohalaridagi ishtirokini kengaytirish, ularning yetakchilik salohiyatini rivojlantirish hamda xalqaro hamkorlikni mustahkamlashga qaratilgan strategik maydon sifatida tashkil etildi.

Forum doirasida ayollar yetakchiligini rivojlantirish va rag‘batlantirish, xalqaro tajriba almashinuvi va ilmiy tarmoqlarni kengaytirish, yosh tadqiqotchi ayollar va PhD talabalarni qo‘llab-quvvatlash hamda innovatsion ekotizimlarda gender tenglikni ta’minlash kabi ustuvor vazifalar amalga oshirildi. Shuningdek, forumda ayollar yetakchiligi va STEM, gender tenglik va ilm-fan institutsional rivoji, ayollar tadbirkorligi va innovatsion startaplar, innovatsiya ekotizimlari va xalqaro hamkorlik, yosh ayol tadqiqotchilarni rivojlantirish, mentorlik va akademik tarmoqlar yo‘nalishlarida tematik sessiyalar tashkil etildi.

Forumda davlat organlari, xalqaro tashkilotlar, yetakchi oliy ta’lim muassasalari va ilmiy markazlar vakillari, jumladan “OLIMA” uyushmasi raisi Murtazayeva Raxbar Hamidovna, “Sharq ayoli” xalqaro ayollar jamoat fondi raisi Tursunbayeva Saodat Gafurovna, Osiyo taraqqiyot banki eksperti Claire Charnac, O‘zbekiston Respublikasi Transport vazirligi vakili Ulmasova Lobarkhon Adilovna, Toshkent davlat transport universiteti rektori G‘ulomov Abdulaziz Abdullayevich, Qirg‘iziston Respublikasi Transport vazirligi vakili Dinara Moldokalieva hamda Indoneziyadan universitet dekani Yoga Prihatin ishtirok etdilar.



Shuningdek, forumda Yevropa va Osiyo davlatlaridan yetakchi olimlar, ekspertlar va yosh tadqiqotchilar keng qatnashdi.

Forum dasturiga ochilish marosimi, panel muhokamalar, ilmiy chiqishlar hamda professional rivojlanish bo'yicha workshoplar kiritilgan bo'lib, ular doirasida gender tenglik, ayollar karyerasini rivojlantirish, ilmiy tadqiqotlar va innovatsion boshqaruv bo'yicha amaliy seminarlar tashkil etildi.

Mazkur tadbirlar Osiyo taraqqiyot banki (ADB), "OLIMA" — O'zbekiston xotin-qizlar olimlari uyushmasi, "Sharq ayoli" xalqaro ayollar jamoat fondi, Germaniya xalqaro hamkorlik jamiyati (GIZ), shuningdek Rossiya, Turkiya, Indoneziya va Qirg'iziston oliy ta'lim muassasalari hamda Belarus Milliy fanlar akademiyasi ilmiy institutlari hamkorligida o'tkazildi.

Mazkur xalqaro ilmiy-amaliy konferensiya va forumning ahamiyati shundaki, ular xalqaro ilmiy hamkorlikni mustahkamlash, innovatsion ishlanmalarni amaliyotga joriy etish, raqamli va barqaror rivojlanishni ta'minlash, shuningdek ayollarning ilm-fan va innovatsiya sohalaridagi rolini kuchaytirishga xizmat qiladi. Shu bilan birga, mazkur tadbirlar yosh olimlar va tadqiqotchilarni qo'llab-quvvatlash, ilmiy tajriba almashish hamda global ilmiy makonga integratsiyalashuvni jadallashtirishda muhim ahamiyat kasb etadi.

Mazkur xalqaro ilmiy-amaliy konferensiya va forumning maqsadi zamonaviy ilmiy tadqiqot yo'nalishlarini muhokama qilish, innovatsion yondashuvlar va ilg'or texnologiyalar bo'yicha ilmiy natijalar almashuvini ta'minlash, shuningdek xalqaro ilmiy hamkorlikni rivojlantirishdan iboratdir.

Tadbirda O'zbekiston Respublikasi hamda xorijiy mamlakatlarning oliy ta'lim muassasalari va ilmiy-tadqiqot institutlari olimlari, shuningdek amaliyotda muhim natijalarga ega bo'lgan mutaxassislar o'z ilmiy ishlari bilan ishtirok etdilar.

Ushbu ilmiy to'plamda muhandislik, transport, raqamlashtirish, sun'iy intellekt, innovatsion boshqaruv, gender tenglik va barqaror rivojlanish sohalarida faoliyat yuritayotgan yetakchi olimlar, professor-o'qituvchilar va tadqiqotchilarning ilmiy ishlari jamlangan. To'plamda dolzarb ilmiy muammolar, zamonaviy yechimlar, nazariy va amaliy tadqiqot natijalari yoritilib, ushbu yo'nalishlarning bugungi holati va istiqboldagi rivojlanish tendensiyalari aks ettirilgan.

Mazkur nashr mamlakatimizda ilm-fan va innovatsion rivojlanishni ta'minlash, xalqaro ilmiy aloqalarni mustahkamlash hamda yosh olimlar va tadqiqotchilarni qo'llab-quvvatlash yo'lidagi muhim qadamlardan biri sifatida e'tirof etiladi.

Shuningdek, mazkur ilmiy to'plamda keltirilgan natijalar kelgusida ilmiy tadqiqotlar samaradorligini oshirish, amaliyotga yo'naltirilgan innovatsion yechimlarni keng joriy etish hamda sohalararo integratsiyani chuqurlashtirish uchun muhim ilmiy asos bo'lib xizmat qiladi. To'plam materiallari nafaqat ilmiy jamoatchilik, balki ishlab chiqarish sohasi mutaxassislari, doktorantlar va talabalar uchun ham foydali manba hisoblanadi.

Mazkur nashrda yoritilgan ilmiy g'oyalar va ilg'or yondashuvlar asosida yangi ilmiy izlanishlar olib borilishi, xalqaro ilmiy aloqalarning yanada rivojlanishi hamda innovatsion rivojlanish strategiyalarining takomillashishiga xizmat qilishi kutilmoqda.



Stress-dependent senomorphic-like activity of buckwheat-derived rutin mediated via SIRT1

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Abstract: Sirtuin 1 (SIRT1) is a key NAD⁺-dependent deacetylase regulating cellular metabolism, oxidative stress resistance, and longevity. This study investigates the senomorphic-like effects of buckwheat-derived rutin on SIRT1 under basal and toxic stress conditions. Using in vivo experiments in mice (liver and kidney tissues, n=8/group) and in silico molecular docking, we assessed SIRT1 protein levels and rutin interactions. BPA-induced stress decreased SIRT1 expression ($p < 0.05$), whereas co-treatment with buckwheat extract restored and elevated SIRT1 above control levels, indicating stress-responsive modulation. Docking analysis revealed strong binding of rutin to the SIRT1 catalytic site (up to -10.3 kcal/mol) with multiple hydrogen bonds, supporting direct modulatory potential. These findings suggest that buckwheat bioactives maintain homeostatic SIRT1 under normal conditions while enhancing its activity under stress, providing a potential dietary strategy to mitigate senescence-associated secretory phenotype (SASP)-related damage.

Keywords: SIRT1, buckwheat, senomorphic, rutin, molecular docking, oxidative stress, SASP

1. Introduction

Rapid Cellular senescence is a physiological process characterized by irreversible cell cycle arrest, contributing to age-related functional decline, chronic inflammation, tissue dysfunction, and the progression of degenerative diseases. Sirtuin 1 (SIRT1), an NAD⁺-dependent deacetylase, serves as a central regulator of this process by modulating cellular metabolism, mitochondrial function, autophagy, oxidative stress responses, and longevity pathways [1,2].

Beyond its canonical roles, SIRT1 exerts senomorphic effects: it modulates the senescence-associated secretory phenotype (SASP) in senescent cells by suppressing pro-inflammatory cytokines (e.g., IL-6, IL-8), proteases, and other factors without inducing cell death. This attenuates senescence-related tissue damage while preserving basal cellular function under normal conditions [3,4,5]. Reduced SIRT1 activity is associated with heightened SASP activation and accelerated cell cycle arrest, underscoring its protective role against senescence-driven pathology [6].

Natural polyphenols, particularly flavonoids such as rutin, have been shown to activate SIRT1 and exhibit context-dependent senomorphic activity. These compounds enhance cellular resilience to oxidative and metabolic stress by selectively upregulating SIRT1 under stress conditions while maintaining homeostatic expression in unstressed cells [7,8]. Rutin, a major flavonoid abundant in buckwheat (*Fagopyrum esculentum*), has demonstrated protective effects against oxidative stress and inflammation, partly through SIRT1-dependent pathways, including modulation of NF- κ B signaling and improvement of mitochondrial function [9,10]. Rutin also reduces markers of cellular senescence in various models and suppresses aspects of SASP, supporting its potential as a senomorphic agent [11,12].

Buckwheat (*Fagopyrum esculentum*) is a rich dietary source of rutin and related bioactives, positioning it as a promising modulator of SIRT1-mediated senomorphic

pathways [13,14,15]. However, the specific mechanisms by which buckwheat bioactives influence SIRT1 expression and activity particularly under stress versus basal conditions remain incompletely understood. Integrating in vivo expression analysis with in silico molecular docking provides a robust approach to characterize these interactions and the potential senomorphic effects of buckwheat flavonoids.

The aim of this study was to investigate the modulatory effects of buckwheat bioactives on SIRT1, quantify SIRT1 protein levels in liver and kidney tissues under basal and toxic stress conditions, and predict the molecular interactions of rutin with SIRT1 through docking analysis. By doing so, we sought to demonstrate the senomorphic potential of buckwheat bioactives and their role in protecting cells from stress-induced senescence.

2. Research methodology

1. Animals and Treatment Protocol

The experiment was conducted on laboratory mice maintained under standard conditions (12 h light/12 h dark cycle, $22 \pm 2^\circ\text{C}$, ad libitum access to food and water). Animals were randomly assigned to four experimental groups (n = 8 per group):

1. **Control** — untreated, unstressed rats.
2. **BKW-STW** — mice treated with buckwheat extract alone.
3. **BPA-STP** — mice exposed to bisphenol A (BPA) to induce toxic stress.
4. **BKW-BPA** — mice treated with both buckwheat extract and BPA.

Treatments were administered orally at predetermined doses for 25 consecutive days. All procedures followed national and international ethical guidelines for animal experimentation.

2. In Vivo SIRT1 Expression Analysis

At the end of the treatment period, animals were

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anesthetized, and liver and kidney tissues were collected. Tissue samples were homogenized, and SIRT1 protein levels were quantified using an ELISA kit according to the manufacturer's instructions. Protein concentrations were expressed as ng/mg of total protein.

Statistical Analysis: Data were presented as Mean $\pm$ SEM. One-way ANOVA followed by Tukey's post-hoc test was used to assess differences among groups. Statistical significance was defined as $p < 0.05$.

3. *In Silico* Molecular Docking

- **Ligands:** Major buckwheat flavonoid -rutin.
- **Receptor:** 3D structure of SIRT1 obtained from the Protein Data Bank (PDB).
- **Docking Tool:** CB-Dock2 was used to predict ligand binding to the SIRT1 catalytic site.
- **Evaluation Criteria:**
 - Binding affinity (kcal/mol)
 - Formation of hydrogen bonds and interactions with key catalytic residues

Formation of hydrogen bonds and interactions with key catalytic residues

Docking results were visualized to confirm the stability of ligand-SIRT1 interactions, providing insight into the direct modulatory potential of buckwheat flavonoids.

4. Ethical Considerations

All animal experiments were conducted in accordance with the guidelines of the National and International Committees for Laboratory Animal Care. ELISA and in silico procedures adhered to standard biosafety protocols.

4. Results and discussion

1. SIRT1 Expression in Liver and Kidney
SIRT1 protein levels in liver and kidney tissues varied significantly among the experimental groups (Table 1).

Table 1

SIRT1 protein levels in liver and kidney tissues (Mean $\pm$ SEM, ng/mg protein)

Group	Liver	Kidney
Control	4.34 $\pm$ 0.21	5.40 $\pm$ 0.04
BKW-STW	3.94 $\pm$ 0.08	4.73 $\pm$ 0.16
BPA-STP	3.73 $\pm$ 0.10	4.65 $\pm$ 0.14
BKW-BPA	4.67 $\pm$ 0.19*	5.91 $\pm$ 0.10*

BPA treatment significantly reduced SIRT1 expression in both liver and kidney ($p < 0.05$), reflecting toxic stress-induced downregulation. Buckwheat treatment alone (BKW-STW) slightly decreased SIRT1 in unstressed tissues, consistent with context-dependent negative

feedback. Co-treatment with buckwheat and BPA (BKW-BPA) restored and enhanced SIRT1 expression, exceeding control levels ($p < 0.05$), indicating stress-responsive senomorphic activity.

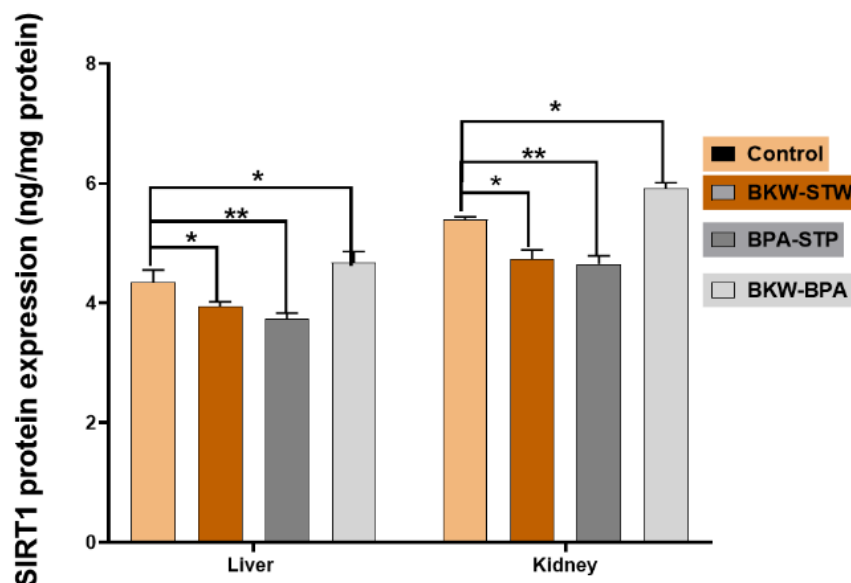


Fig. 1. SIRT1 protein levels in liver and kidney tissues. Statistical significance between groups is indicated (* $p < 0.05$, ** $p < 0.01$)

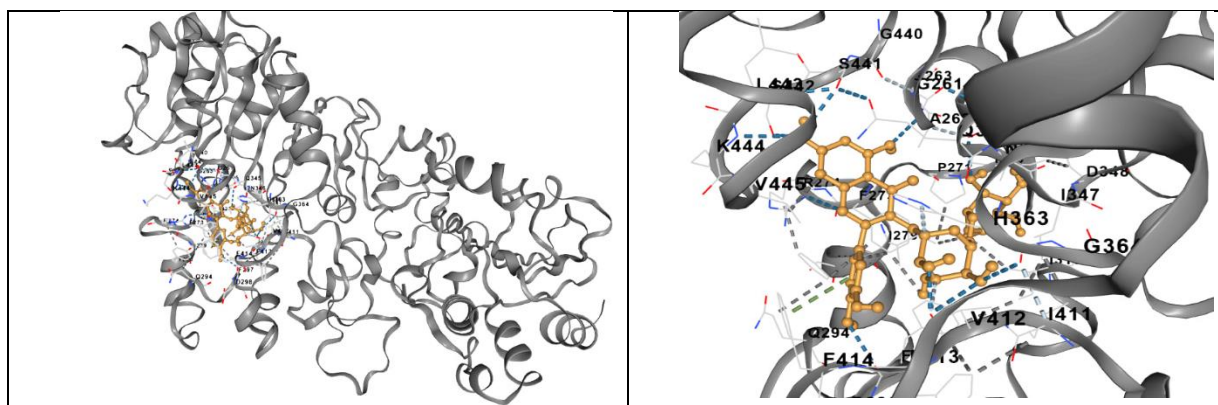


Fig. 2. 3D molecular docking of Rutin with SIRT1

2. In Silico Docking Analysis
Molecular docking of buckwheat flavonoids revealed stable interactions with the SIRT1 binding site.

The docking analysis identified several potential binding pockets of SIRT1 interacting with Rutin (Table 1). Among them, pocket C2 exhibited the highest binding affinity at -10.3 kcal/mol, with a large volume of $4052 \text{ \AA}^3$ and centered

at coordinates $(10, -2, 19)$, indicating a spacious and energetically favorable site for ligand accommodation. Pockets C3, C5, C4, and C1 showed slightly lower affinities (-8.7 to -8.0 kcal/mol), with variable volumes and positions, suggesting multiple potential binding sites that may contribute to the modulatory effect of Rutin.

Table 2

Binding affinities, pocket volumes, and docking coordinates of Rutin with SIRT1

Pocket ID	Affinity (kcal/mol)	Volume ($\text{\AA}^3$)	Center (x,y,z)	Docking Size(x,y,z)
C2	-10.3	4052	$(10, -2, 19)$	$(25, 33, 31)$
C3	-8.7	2827	$(19, -17, 27)$	$(25, 33, 25)$
C5	-8.7	485	$(40, -8, 26)$	$(25, 25, 25)$
C4	-8.4	646	$(30, -21, 27)$	$(25, 25, 25)$
C1	-8.0	4407	$(43, -27, 18)$	$(25, 35, 32)$

Rutin formed multiple hydrogen bonds with key catalytic residues of SIRT1, supporting a direct modulatory effect on SIRT1 activity. These interactions provide a structural basis for the observed *in vivo* senomorphic effects, as flavonoids may enhance SIRT1 activity under stress conditions without affecting basal levels.

3. Integrated Analysis
The combination of *in vivo* and *in silico* data demonstrates that buckwheat bioactives exhibit senomorphic properties:

1. Stress-free conditions (BKW-STW): SIRT1 expression is slightly decreased, maintaining homeostasis.
2. Toxic stress (BPA-STP): SIRT1 expression decreases due to oxidative stress.
3. Stress + Buckwheat (BKW-BPA): SIRT1 is restored and enhanced, indicating stress-responsive senomorphic activity.
4. Docking evidence: Rutin binds stably to SIRT1, supporting direct modulation.

These results suggest that buckwheat bioactives can selectively modulate SIRT1, protecting cells from SASP-related damage under stress while maintaining basal function under normal conditions.

Cellular senescence is a hallmark of aging and contributes to tissue dysfunction, chronic inflammation, and increased susceptibility to degenerative diseases. SIRT1, a NAD^+ -dependent deacetylase, has emerged as a key regulator of senescence, oxidative stress response, and metabolic homeostasis [1,2]. Beyond its canonical roles, SIRT1 has been implicated in senomorphic activity, whereby it modulates the senescence-associated secretory phenotype (SASP) without inducing cell death, thereby

attenuating pro-inflammatory signaling and tissue damage [3,4,5].

In the present study, we demonstrate that buckwheat bioactives exert context-dependent, senomorphic effects on SIRT1. In unstressed tissues, buckwheat treatment (BKW-STW) slightly decreased SIRT1 expression relative to control, consistent with homeostatic regulation and negative feedback mechanisms. This suggests that buckwheat flavonoids do not overactivate SIRT1 in basal conditions, preventing potential metabolic imbalance. Conversely, under BPA-induced toxic stress, SIRT1 expression was significantly reduced (BPA-STP), reflecting oxidative and metabolic perturbations. Co-treatment with buckwheat (BKW-BPA) restored and further enhanced SIRT1 levels, indicating stress-responsive modulation and supporting a senomorphic mechanism [13].

In silico docking analysis provides structural insights into this modulatory activity. Rutin exhibited favorable binding energy (-10.2 kcal/mol,) and formed stable hydrogen bonds with key catalytic residues of SIRT1. These findings suggest that the flavonoid act as direct modulators, enhancing SIRT1 activity under stress conditions, which aligns with the observed *in vivo* restoration of protein levels [15]. Importantly, this direct interaction likely underpins the selective senomorphic activity, whereby SIRT1-mediated suppression of SASP reduces inflammatory signaling in stressed cells without affecting normal cellular function.

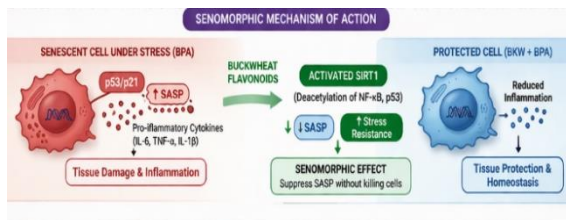


Fig. 3. Role of SIRT1 in the senomorphic mechanism mediated by buckwheat flavonoids

Previous studies have reported similar senomorphic effects for polyphenols such as resveratrol and quercetin, which enhance SIRT1 activity, mitigate SASP factors, and protect tissues from age- or toxin-induced damage [16]. Our results extend these findings by demonstrating that buckwheat-derived flavonoids can modulate SIRT1 in a tissue-specific and stress-dependent manner, highlighting their potential as natural senomorphic agents [17].

The integrated *in vivo* and *in silico* approach strengthens the mechanistic understanding of buckwheat flavonoids. Restoration of SIRT1 in liver and kidney under toxic stress, coupled with stable ligand–receptor interactions *in silico*, provides compelling evidence that the senomorphic effects are mediated by direct SIRT1 modulation, rather than indirect antioxidant effects alone. This distinction is critical for designing senomorphic interventions, as it emphasizes selective modulation of senescence-associated pathways without cytotoxicity.

Overall, these findings support the concept that buckwheat bioactives confer protective senomorphic effects through SIRT1, preserving tissue homeostasis under normal conditions and enhancing stress resilience. Such compounds may have translational potential in preventing or mitigating senescence-associated tissue dysfunction and toxicity-induced cellular damage.

5. Conclusion

This study demonstrates that buckwheat (*Fagopyrum esculentum*) bioactives, particularly rutin and quercetin, exert senomorphic effects through the selective modulation of SIRT1. Key findings include:

1. Under normal, unstressed conditions, buckwheat maintains basal SIRT1 expression, preventing unnecessary overactivation and supporting cellular homeostasis.
2. Under BPA-induced toxic stress, SIRT1 expression is downregulated, but co-treatment with buckwheat restores and enhances SIRT1 levels, demonstrating stress-responsive senomorphic activity.
3. *In silico* docking confirms that rutin binds stably to SIRT1's catalytic site, providing mechanistic evidence for direct modulation of SIRT1 activity.
4. Overall, buckwheat bioactives suppress stress-induced SASP and inflammatory signaling without inducing cell death, highlighting their potential as natural senomorphic agents.

These results support the concept that buckwheat-derived flavonoids can enhance cellular resilience and protect tissues from senescence-associated dysfunction, offering a promising dietary or therapeutic approach for age- and toxin-related cellular stress. Future studies should explore long-term effects and translational applications in models of chronic senescence and metabolic dysfunction.

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