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Original Article

Protective effect of taurine on skin aging caused by deficiency of B cell-specific Moloney MLV insertion site-1

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Abstract: Bmi-1 deficiency leads to elevated oxidative damage and premature aging, making it a suitable model for studying the mechanisms of intrinsic skin aging. Taurine is abundant in the skin and has been shown to have various health benefits. However, it is unclear whether taurine can inhibit intrinsic aging by protecting skin from oxidative damage and suppressing cellular senescence. In the current study, Bmi-1 homozygous mice were fed with or without taurine. We constructed siRNA plasmids specifically targeting Bmi-1 and transfected them into human dermal fibroblasts (HDFs). HDFs were then treated with or without taurine. The changes in dermal morphology of mice, HDF function, proliferation, oxidative stress, DNA damage, and cell senescence were compared to observe whether taurine can inhibit skin aging caused by Bmi-1 deficiency. Results showed that, in Bmi-1 homozygous mice drinking normal water, the vertical thickness of skin, the positive area of collagen bundles, type I collagen and elastic fiber, the percentage of PCNA-positive cells, and SOD1 and SOD2 proteins levels were significantly reduced. ROS levels, the proportion of 8-OHdG positive cells, p53, Chk2, p-Chk2, γ-H2AX and MMP3 proteins levels, as well as the percentage of SA-β-gal, MMP1 and MMP3-positive areas were upregulated dramatically. However, taurine supplementation reversed these changes. *In vitro*, compared to the control group, dramatic increases of γ-H2AX-positive HDFs, p16 mRNA levels, and SA-β-gal-positive areas were observed in the si-Bmi-1 group, while taurine treatment significantly decreased these expression levels. These results demonstrated that taurine could prevent Bmi-1 deficiency-induced skin aging.

Keywords: Taurine, Bmi-1, skin aging, redox balance

Introduction

Skin aging is a complicated process caused by both internal and external environmental factors [1]. Intrinsic skin aging, which is associated with age, genetics, or endocrine factors, is characterized by skin thinning, laxity, elasticity loss, and skin dryness. External factors, such as ultraviolet exposure, air pollutants, smoking, and alcohol consumption, have greater effects on the development of aging phenotypes, leading to wrinkles, hyperpigmentation, and dry skin [2].

Numerous studies have shown that the excessive accumulation of reactive oxygen species (ROS) can attack nucleic acids, proteins, and other cellular components, and lead to oxidative damage and cellular senescence [3]. There are multiple secretory cytokines that can be increased by senescent cells, and they are referred to as the senescence-associated secretory phenotype (SASP), such as proinflammatory factors, chemokines, and matrix metalloproteinases (MMPs) that accelerate chronic inflammation and tissue damage [4]. MMPs are important proteolytic enzymes that specifically degrade and destroy extracellular matrix (ECM) components in the dermis, such as collagen and elastin, ultimately leading to skin aging [5]. Therefore, removing oxidative damage to skin cells is one of the most common methods to protect from skin aging [6]. Taurine (2-aminoethanesulfonic acid) is an amino acid that is found widely in all the tissues of mammals. Taurine has been proven to be an antioxidant

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[7]. Taurine can significantly inhibit mitochondrial oxidative stress, enhance mitochondrial membrane potential, and improve sperm motility and viability [8]. Taurine had the potential to suppress oxidative stress in the spinal cord of diabetic rats by modulating the Keap1-Nrf2 signaling pathway [9]. Taurine is also reported to be present in high concentrations in the skin and contributes to skin homeostasis [10]. Taurine contributes to skin anti-aging effects through immunological pathways [11]. However, it remains unclear whether taurine can inhibit intrinsic skin aging by suppressing redox imbalance and the DNA damage response (DDR).

Studies have shown that B cell-specific Moloney MLV insertion site-1 (Bmi-1) deficiency causes severe mitochondrial dysfunction with a continuously raised level of ROS, which is sufficient to trigger organismal aging by the DDR pathway [12, 13]. Our previous study has shown that Bmi-1 is extremely active in the skin and plays a pivotal role in the fight against skin aging through maintenance of redox balance [14]. However, whether taurine can inhibit Bmi-1 deletion-induced skin aging by inhibiting oxidative imbalance and cell senescence is also unknown. To address this question, Bmi-1 deficiency model mice fed with or without taurine in the drinking water were used in the present study. We constructed siRNA plasmids specifically targeting Bmi-1 and transfected them into human dermal fibroblasts (HDFs). HDFs were then treated with or without taurine. We analyzed the changes in the morphology of skin, cell proliferation, oxidative damage, cell senescence, and DDR to establish whether taurine has the ability to counteract skin aging related to the Bmi-1 deletion.

Materials and methods

Mice

Professor Dengshun Miao kindly provided Bmi-1 heterozygous mice. The mice were separated into three groups: (1) WT littermates; (2) Bmi-1 homozygous mice; (3) Bmi-1 homozygous mice supplemented with 3% (w/v) taurine. Taurine (Sigma-Aldrich, USA) was added to drinking water and provided to mice on ad libitum during 5 weeks. The dose and duration of oral taurine supplementation were determined by referring to previous studies [10, 15, 16]. All the mice were anesthetized with 1.25% tribromoethanol (30 μl/g, Tuoran Biotechnology, Shanghai, China) by intraperitoneal injection. When the mice showed no response to stimuli, the anesthesia was considered successful. Subsequently, we euthanized mice by cervical dislocation. The animal studies were performed under the supervision of the Institutional Animal Care and Use Committee of Nanjing Medical University (No. IACUC-2508045).

Cell culture and treatment

The human dermal fibroblasts (HDFs; NB1RGB, from ATCC) were grown in DMEM medium at 37°C with 5% CO_2. si-Bmi-1 (Small interfering RNA, siRNA) was obtained from GenePharma (Shanghai, China). We used Lipofectamine 3000 (Invitrogen, USA) to carry out cell transfection according to the protocol of the manufacturer. Cells were grown in suitable culture dishes until they were 70 percent confluent before transfection. siRNAs (50 nM final concentration) were transfected. The HDFs were then incubated with or without 5-50 mM taurine.

Cell viability analysis

We used the Methyl Thiazolyl Tetrazolium (MTT) assay to detect cell viability. The protocol was as follows: cells were cultured for 1, 2, 3, and 4 days after treatment. Each well was added with MTT solution (Sigma-Aldrich, USA) and treated for 4 hours. The formazan crystals were dissolved in Dimethyl sulfoxide (DMSO). The absorbance was then determined at 490 nm with a microplate reader (BioTek).

Senescence-associated β-galactosidase (SA-β-gal) staining

Cellular senescence was determined using the Senescence β-Galactosidase Staining Kit (Beyotime Biotechnology, China). The procedure was as follows: cells were fixed at room temperature for 15 minutes with 4% paraformaldehyde, washed with PBS, and then stained with β-galactosidase staining solution at 37°C overnight. The blue-stained cells were senescent, and the percentage of SA-β-gal-positive cells was calculated using random fields.

Immunofluorescence staining

Cells cultured on coverslips were fixed for 15 minutes with fixative solution, incubated for 10

minutes with 0.5% Triton X-100, and blocked for 1 hour at room temperature with 5% bovine serum albumin (BSA). The primary antibody against γH2AX (Cell Signaling Technology, USA) was added to the cells overnight at 4°C. Then, cells were incubated with Alexa Fluor 488-conjugated secondary antibody (Invitrogen, USA) for 1 hour at room temperature. DAPI (Sigma-Aldrich, USA) was used to counterstain the cell nuclei. A confocal laser scanning microscope was used to obtain images, and the percentage of positive cells was calculated by randomly selecting fields.

Quantitative real-time RT-PCR

The relative p16 mRNA level was quantified by qRT-PCR as described previously [17]. The following sequence-specific primers of human p16 were used: forward primer 5'-ATGGAGCCTTCGGCTGACT-3' and reverse primer 5'-GTAACTATTCGGTGCGTTGGG-3'. Each reaction was normalized using GAPDH as the internal control.

Western blot analysis

Western blot was performed as described previously [17]. Primary antibodies against superoxide dismutase 1 (SOD1, Cell Signaling Technology, USA), superoxide dismutase 2 (SOD2, Cell Signaling Technology, USA), Chk2 (Abcam, USA), p-Chk2 (Abcam, USA), Histone H2A.X (γH2AX, Cell Signaling Technology, USA), matrix metallopeptidase 3 (MMP3, Abcam, USA), p53 (Cell Signaling Technology, USA) and β-actin (Abcam, USA) were used.

Histology

The dorsal skin flaps below the scapulae of the mice were removed and fixed at 4°C for 24 hours. Afterward, the specimens were dehydrated, embedded in paraffin, and cut into 5 μm sections. Then, the sections were stained with hematoxylin and eosin or histochemically for Masson's trichrome staining, Elastic Van Gieson staining, and SA-β-gal staining and immunohistochemically as described previously [14].

Immunohistochemistry

Immunohistochemical staining was performed in skin paraffin-embedded sections for type I collagen (SouthernBiotech, USA), PCNA (Santa Cruz, China), 8-OHdG (Abcam, USA), MMP1 (Proteintech Biotechnology, China), and MMP3 (Abcam, USA) as described previously [18].

ROS detection

The dorsal skin flaps were trypsinized to prepare single-cell suspensions. Intracellular ROS analysis was carried out as described previously [18].

Computer-assisted image analysis

After performing immunohistochemical or histochemical staining, rectangular templates were used to digitally record the micrographs from single sections. The Northern Eclipse image analysis software was used to process and analyze the recorded data.

Statistical analysis

All data were performed as mean ± SEM. GraphPad Software was used to perform statistical analysis. The data were calculated by Student's t-test. One-way ANOVA was used to describe the comparison between the groups. Qualitative values were presented as percentages, and they were analyzed through the chi-square test. *P*-values < 0.05 were considered significant.

Results

Taurine prevents skin aging induced by Bmi-1 deletion

To clarify a potential role of taurine supplementation in preventing skin aging due to Bmi-1 deletion, 3-week-old Bmi-1 homozygous mice were fed drinking water containing taurine for 5 weeks, while the control groups received normal drinking water. Following sacrifice, we performed H&E staining, Masson's trichrome staining, elastic Van Gieson staining, and type I collagen (col-1) immunohistochemical staining to compare the morphology of skin. Compared to Bmi-1 homozygous littermates drinking normal water, taurine supplementation significantly alleviated skin aging in Bmi-1 homozygous mice, as evidenced by decreased skin vertical thickness (**Figure 1A**, **1B**), decreased positive density and area of collagen fibers (**Figure 1C**, **1D**), type I collagen (**Figure 1E**, **1F**) and elastic

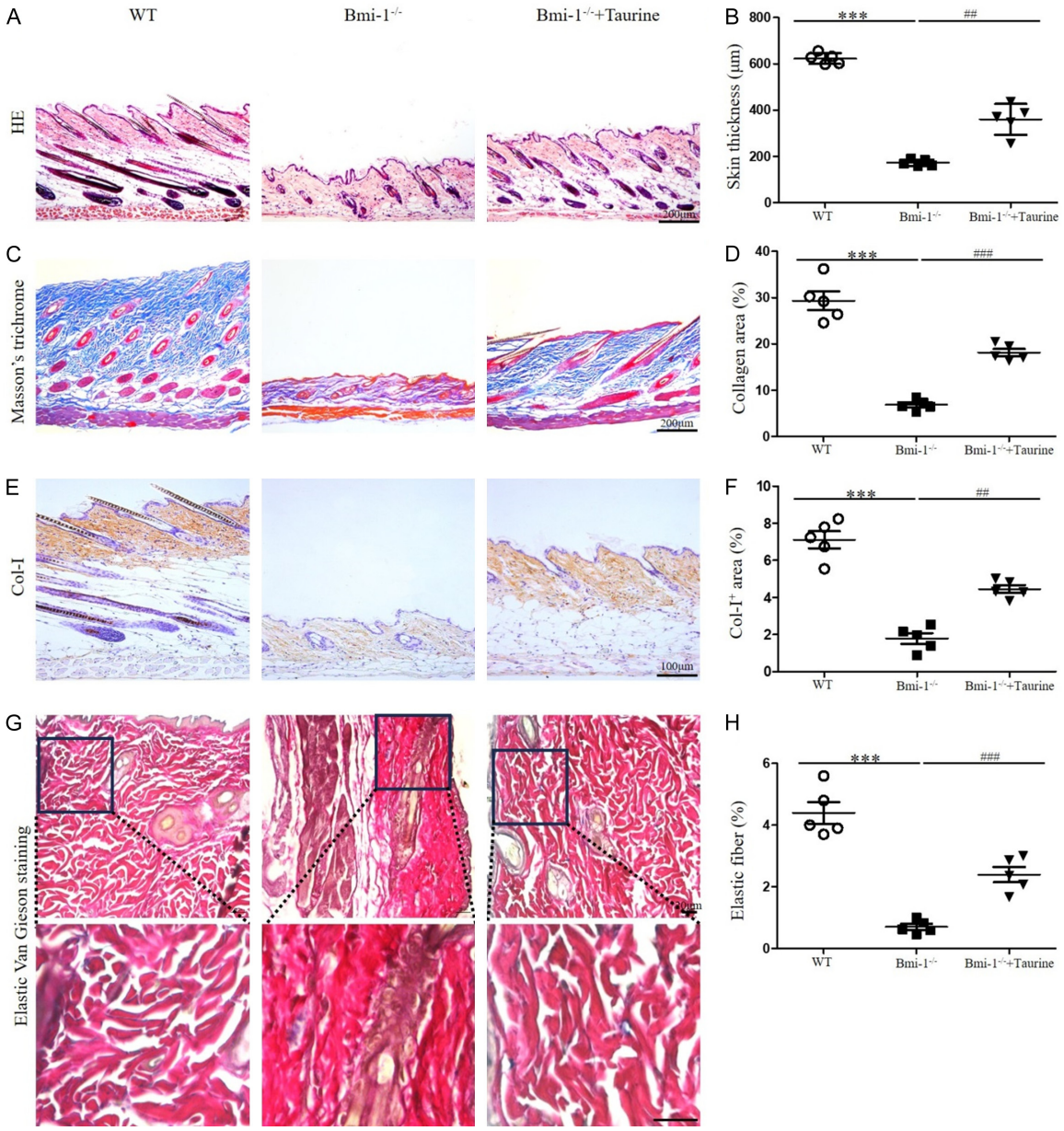

Figure 1. Taurine prevents skin aging induced by Bmi-1 deletion. A. H&E staining in skin sections. B. Quantification of skin thickness. C. Masson's trichrome staining in skin sections. D. Quantification of collagen bundles (%). E. Immunohistochemical staining for type I collagen in skin sections. F. Quantification of type I collagen-positive area (%). G. Van Gieson staining in skin sections. H. Quantification of elastic fibers (%). Data were presented as mean ± SEM (n = 5). ***P < 0.001 vs. WT mice; ##P < 0.01, ###P < 0.001 vs. Bmi-1 homozygous mice drinking normal water.

fibers (**Figure 1G**, **1H**). These findings suggest that taurine supplementation can effectively prevent skin aging caused by Bmi-1 deletion.

Taurine promotes skin cell proliferation and alleviates skin cell senescence induced by Bmi-1 deletion

To determine whether the skin aging phenotypes due to Bmi-1 deletion were related to altered skin cell proliferation and cellular senescence, we examined the cell proliferation marker PCNA, the cellular senescence marker SA-β-gal, and p53. Results showed that, compared to WT mice, the proportion of PCNA-positive cells in the skin of Bmi-1 homozygous mice drinking normal water was decreased (**Figure 2A**, **2B**), while the SA-β-gal-positive area and p53 protein expression levels were elevated (**Figure 2C-F**). However,

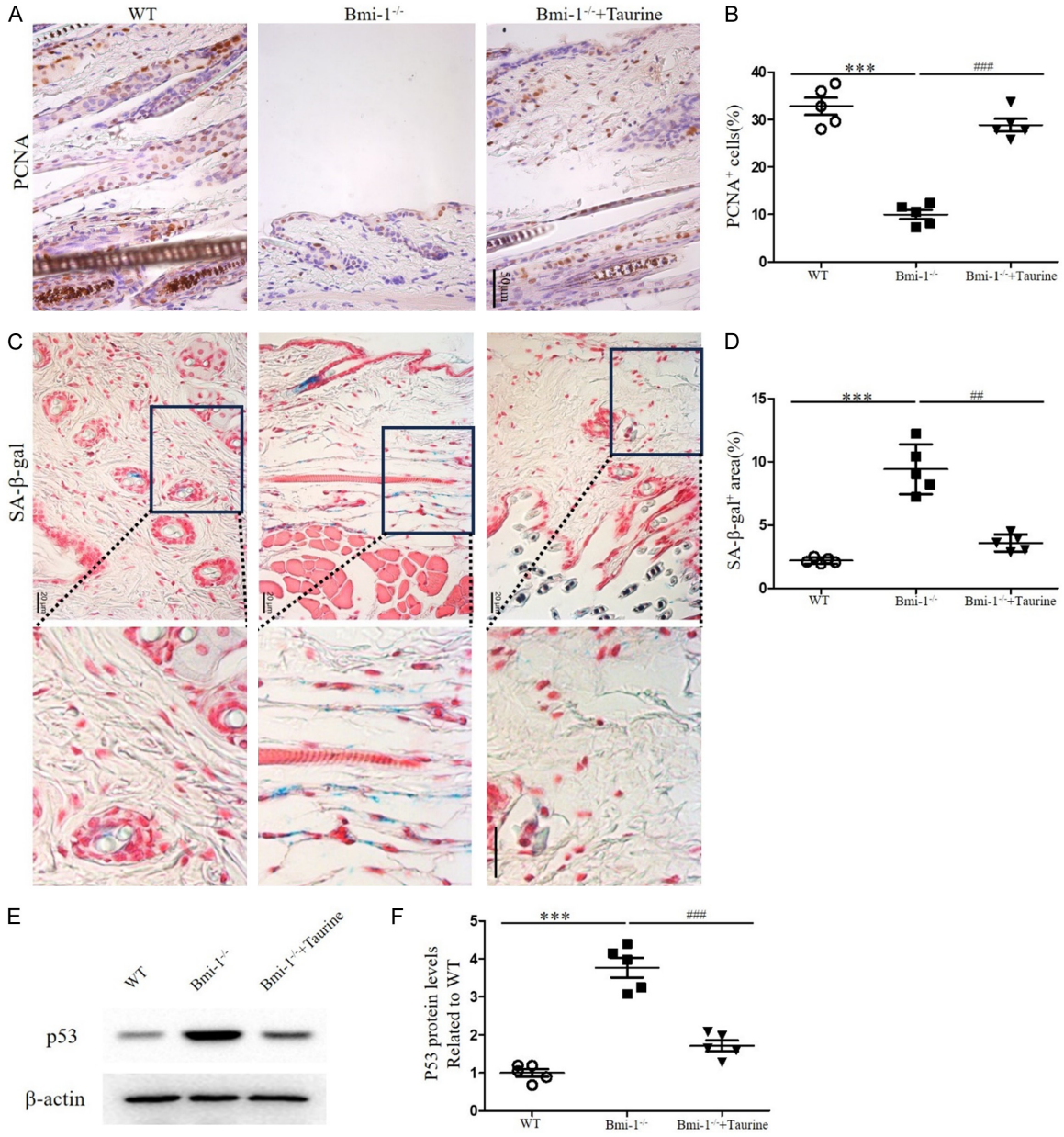

Figure 2. Taurine promotes skin cell proliferation and alleviates skin cell senescence induced by Bmi-1 deletion. A. Immunohistochemical staining for PCNA in skin sections. B. Quantification of PCNA-positive cells (%). C. SA-β-gal staining in skin sections. D. Quantification of SA-β-gal-positive area (%). E. Western blot of p53 protein in skin. F. Quantification of p53 protein level normalized to WT mice. Data were presented as mean ± SEM (n = 5). ***P < 0.001 vs. WT mice; ##P < 0.01, ###P < 0.001 vs. Bmi-1 homozygous mice drinking normal water.

these changes were significantly reversed after taurine supplementation. These results suggested that taurine supplementation alleviated skin aging caused by Bmi-1 deletion, which may be related to the upregulation of skin cell proliferation and downregulation of cellular senescence.

Taurine partly rescues skin redox imbalance induced by Bmi-1 deletion

Research on the skin aging mechanisms has shown that elevated oxidative stress is one of the key mechanisms in the skin. We further confirmed whether the decrease in cell prolif-

eration and the increase in cellular senescence in the skin of Bmi-1 homozygous mice were associated with changes in oxidative stress. Flow cytometry analysis revealed that the levels of intracellular ROS were obviously elevated in the skin of Bmi-1 homozygous mice drinking normal water, while taurine treatment significantly corrected these changes (**Figure 3A**, **3B**). To further confirm whether the increased oxidative stress in skin caused by Bmi-1 deletion was related to decreased antioxidant capacity, SOD1 and SOD2 protein levels in the skin were detected. We found a significant decrease in the levels of these two proteins in Bmi-1 homozygous mice drinking normal water (**Figure 3C-E**). However, after taurine supplementation, the antioxidant capacity of Bmi-1 homozygous mice was significantly improved. These findings suggested that taurine may promote skin cell proliferation and suppress cellular senescence induced by Bmi-1 deletion by suppressing oxidative damage.

Taurine suppresses skin DDR activation induced by Bmi-1 deletion

Oxidative stress can trigger the DDR activation. To confirm whether the increased oxidative stress induced by Bmi-1 deletion can cause DDR activation, the DNA damage markers were detected in the skin. Results showed that, compared to WT mice, the proportion of 8-OHdG-positive cells (**Figure 4A**, **4B**) and the protein levels of Chk2, p-Chk2, and γ-H2AX in the skin of Bmi-1 homozygous mice drinking normal water were upregulated dramatically (**Figure 4C-F**). However, the elevated levels of these oxidative damage markers observed in Bmi-1 homozygous mice drinking normal water were also alleviated by taurine supplementation. Our findings indicated that taurine supplementation could suppress oxidative stress-induced DNA damage caused by Bmi-1 deletion.

Taurine suppresses MMP activation induced by Bmi-1 deletion

The DNA damage response activation may lead to cellular senescence, which in turn triggers the secretion of inflammatory cytokines, such as MMPs. Therefore, the MMP-1 and MMP-3 expression levels were detected. Results revealed that the elevated MMPs (the percentage of MMP1 and MMP3-positive area (**Figure 5A-D**) and the protein levels of MMP3 (**Figure 5C-F**) observed in the skin of Bmi-1 homozygous mice drinking normal water were ameliorated by taurine. These findings suggested that the effect of taurine supplementation on the skin aging phenotypes due to Bmi-1 deletion may be related to the inhibition of cellular senescence and MMPs secretion.

Taurine inhibits HDF senescence induced by Bmi-1 knockdown

To clarify the effects of Bmi-1 on HDF function, we constructed siRNA plasmids specifically targeting Bmi-1. After transfection into HDFs, HDFs were treated with or without taurine. Our results revealed that taurine exhibited no significant cytotoxicity at concentrations ranging from 5 to 50 mM (**Figure 6A**). In contrast to the control group, the cell viability of the si-Bmi-1 group was dramatically dampened, while taurine treatment improved the survival rate of HDFs (**Figure 6B**). Then, we evaluated alterations in DNA damage and cell senescence using immunofluorescence staining for γ-H2AX, Real-Time RT-PCR analysis for p16, and cytochemical SA-β-gal staining. Results showed that dramatic increases of γ-H2AX-positive HDFs (**Figure 6C**, **6D**), p16 mRNA levels (**Figure 6E**), and SA-β-gal-positive HDFs (**Figure 6F**, **6G**) were observed in the si-Bmi-1 group compared to the control group while significant decreases were observed in si-Bmi-1 HDFs supplemented with taurine compared to the si-Bmi-1 group. These results demonstrated that taurine supplementation may stimulate proliferation and inhibit cell senescence in si-Bmi-1 HDFs by inhibiting DNA damage.

Discussion

Our findings presented in this study suggested that taurine supplementation may serve as an effective agent to alleviate skin aging caused by Bmi-1 deletion. Bmi-1 plays a critical role in skin homeostasis [19]. Bmi-1 deletion causes an early aging process and cellular dysfunctions, which makes it a good model to study skin aging. Taurine is a sulfur-containing amino acid that is present in the skin. The studies have shown that taurine concentrations in the mice and rat skin diminished gradually with age [10]. Exogenous taurine chloramine supplementation to the skin may directly increase cellular resistance to UVB-induced inflammation and oxidative damage [20]. Topical taurine

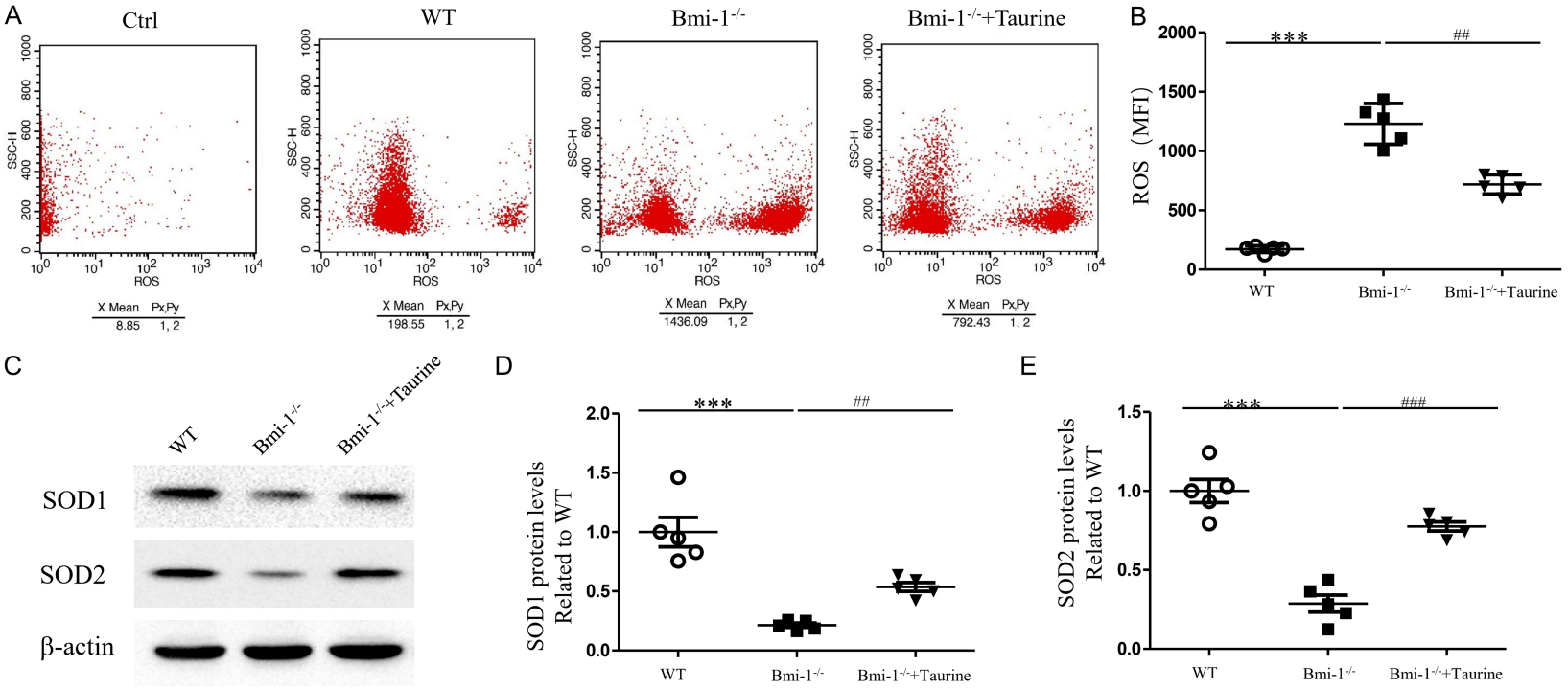

Figure 3. Taurine partly rescues skin redox imbalance induced by Bmi-1 deletion. (A) Flow cytometric analysis of cellular ROS levels in skin. (B) The relative fluorescence intensity (RFI) of ROS was measured. (C) Western blots of SOD1 and SOD2 proteins in skin. Quantification of (D) SOD1 and (E) SOD2 protein levels normalized to WT mice. Data were presented as mean ± SEM (n = 5). ***P < 0.001 vs. WT mice; ##P < 0.01, ###P < 0.001 vs. Bmi-1 homozygous mice drinking normal water.

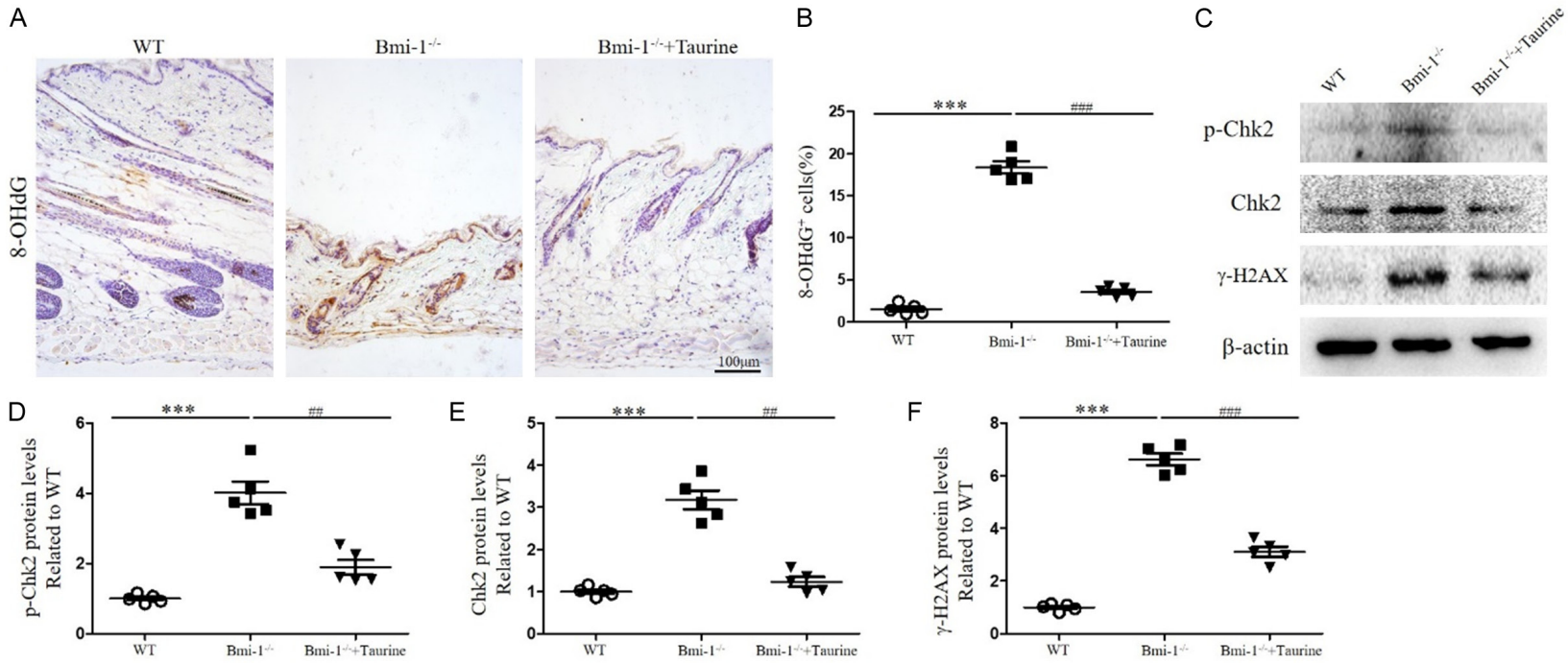

Figure 4. Taurine suppresses skin DDR activation induced by Bmi-1 deletion. (A) Immunohistochemical staining for 8-OHdG in skin sections. (B) Quantification of 8-OHdG-positive cells (%). (C) Western blots of p-Chk2, Chk2, and γ-H2AX proteins in skin. Quantification of (D) p-Chk2, (E) Chk2, and (F) γ-H2AX protein levels normalized to WT mice. Data were presented as mean ± SEM (n = 5). ***P < 0.001 vs. WT mice; ##P < 0.01, ###P < 0.001 vs. Bmi-1 homozygous mice drinking normal water.

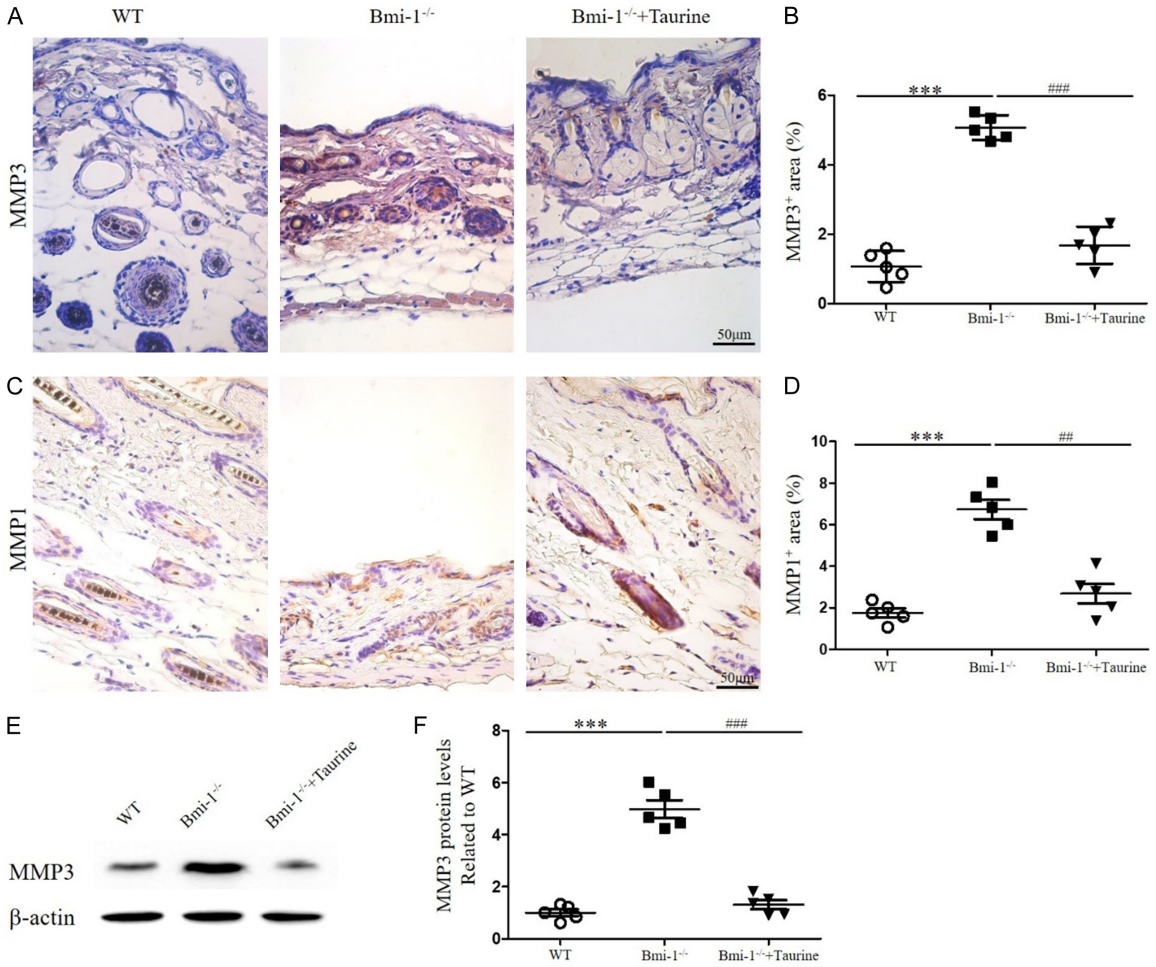

Figure 5. Taurine suppresses MMP activation induced by Bmi-1 deletion. A. Immunohistochemical staining for MMP-3 in skin sections. B. Quantification of MMP-3-positive cells (%). C. Immunohistochemical staining for MMP-1 in skin sections. D. Quantification of MMP-1-positive cells (%). E. Western blot of MMP-3 protein in skin. F. Quantification of MMP-3 protein levels normalized to WT mice. Data were presented as mean ± SEM (n = 5). ***P < 0.001 vs. WT mice; ##P < 0.01, ###P < 0.001 vs. Bmi-1 homozygous mice drinking normal water.

application promoted the expression of tight junction-related proteins and effectively restored the skin homeostasis in the sleep-deprived female mice [21]. Although taurine has several health benefits, there is very little information regarding how it may inhibit skin aging by preventing oxidative damage and cellular senescence.

The skin morphology analysis indicated that taurine could act as a protective agent against skin structure degradation caused by Bmi-1 deletion, including a great increase in the skin thickness and a high proportion of elastic fibers or collagen fibers. Reduced cell proliferation and accelerated aging mark a decline in tissue regeneration and functioning. The reduced levels of the cell proliferation marker PCNA in the skin caused by Bmi-1 deletion were significantly restored in the skin of Bmi-1 homozygous mice that drank taurine-supplemented water. This indicated that taurine would be helpful in the recovery of the skin cell proliferative ability, which is very important in skin repair and regeneration. However, the enhanced skin cell senescence caused by Bmi-1 deletion, characterized by high levels of SA-β-gal positive cells and p53 protein expression, was greatly alleviated by taurine. *In vitro* experiments of this study also showed that taurine supplementation could prevent cellular senescence in si-Bmi-1 HDFs. It is reported that the levels of circulating taurine decrease with age in mice, monkeys and humans, and taurine supplementation might

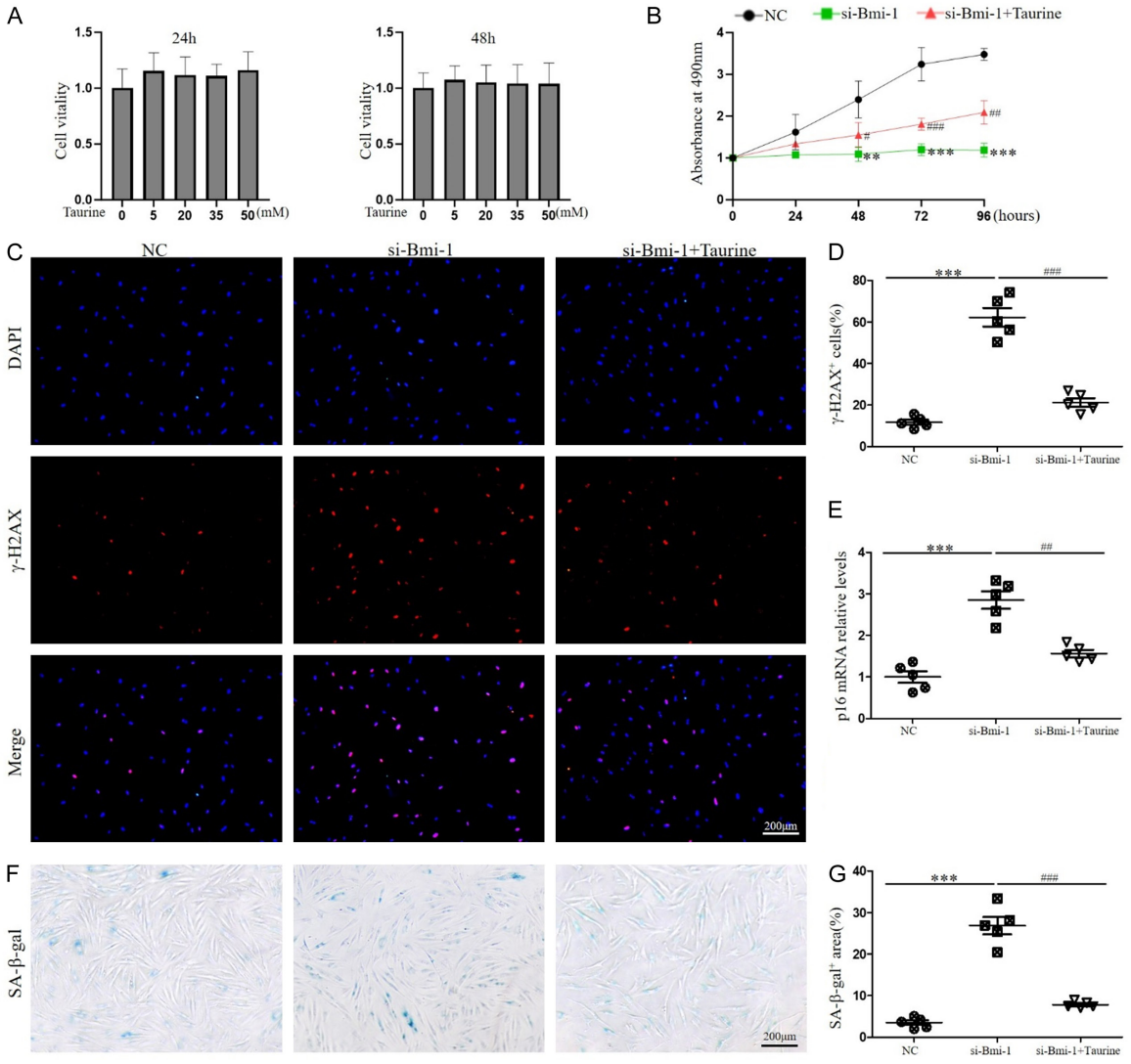

Figure 6. Taurine inhibits fibroblast senescence induced by Bmi-1 knockdown. A. Human dermal fibroblast (HDF) viability was detected using MTT assay following taurine (0, 5, 20, 35, 50 mM) treatment for 24 and 48 h. B. MTT assay examining the effects of taurine supplementation on HDF viability from the normal control (NC) group, the si-Bmi-1 group, and the si-Bmi-1 supplemented with taurine group. C. Immunofluorescence staining for γ-H2AX. D. Quantification of γ-H2AX-positive cells. E. p16 mRNA relative levels. F. Cytochemical staining for SA-β-gal. G. Quantification of SA-β-gal-positive area. Data were presented as mean ± SEM (n = 5). $^{**}P < 0.01$, $^{***}P < 0.001$ vs. NC; $^{\#}P < 0.05$, $^{\#\#}P < 0.01$, $^{\#\#\#}P < 0.001$ vs. si-Bmi-1 group.

extend the lifespan of the mice and the health of the monkeys [22]. Studies on humans have also revealed that increasing taurine could prevent deterioration of the biological functions of the aging process [23]. Consistent with these reports, our study also determined that taurine may be beneficial to extend the functional lifespan of skin cells. Although our results showed that taurine supplementation effectively delayed skin aging in Bmi-1 deficient mice, it remains unclear whether the effects of taurine were specific to the Bmi-1 deficiency or if they represent a general response in WT mice. Further studies including taurine administration to WT mice would be valuable to clarify the role of taurine in skin aging.

Oxidative stress has been a major contributor to skin aging [24]. Antioxidant supplements are also known to play a significant role in alleviating oxidative stress and inhibiting skin aging [25-27]. The results of flow cytometry analysis revealed that taurine was able to cause a significant reduction in the ROS levels, indicating

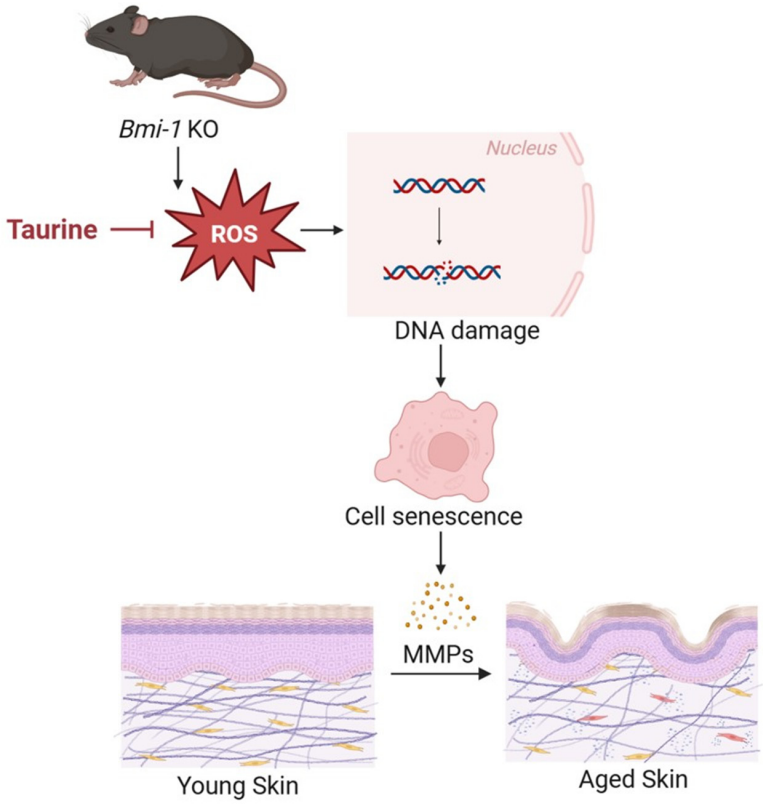

Figure 7. Schematic diagram of taurine prevents skin aging caused by Bmi-1 deletion. Created with BioRender.com.

that it may have antioxidant properties, which could significantly restore the redox balance. In addition, the antioxidant enzyme levels in the skin of Bmi-1 homozygous mice were lower, which were restored by taurine supplementation, and this is further evidence that taurine may be an antioxidant that is helpful in improving skin defense mechanisms against oxidant damage. However, the biological activity of taurine is multifaceted. It can act as a direct scavenger of specific ROS and also as a potent modulator of key signaling pathways, including mitochondrial function [28], calcium homeostasis [29, 30], and the Nrf2/Keap1 antioxidant response [31, 32]. Whether the observed effects result from the combination of these indirect mechanisms or from direct radical scavenging remains to be determined.

Oxidative stress may lead to cellular components degradation and DDR activation [6, 33]. The effect of taurine on skin DNA damage due to Bmi-1 deletion is that these changes were reversed, indicating that taurine may play a helpful role in inhibiting the activation of the DDR pathway and promoting DNA repair. One of the most important enzymes which is involved in the degradation of matrix components is MMPs [34]. The levels of MMPs in senescent skin increase, especially MMP-1 and MMP-3. These MMPs are capable of breaking down collagen and elastin which are structural proteins in the skin [35, 36]. In the current study, taurine caused a significant reduction in the expression levels of the MMPs, and this implied that the anti-inflammatory action of taurine might be important in the skin structural and functional damage associated with Bmi-1 deletion.

This study proved that taurine supplementation could be useful to delay Bmi-1 deletion-induced skin aging by suppressing oxidative stress, enhancing cell proliferation, preventing DNA damage and cellular senescence (**Figure 7**). These findings suggested that taurine may provide an innovative therapeutic approach for combating age-related skin alterations and other skin pathologies associated with oxidative stress and cell senescence.

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Disclosure of conflict of interest

None.

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