Data in Brief 39 (2021) 107572

Contents lists available at ScienceDirect

Data in Brief

journal homepage: www.elsevier.com/locate/dib

Data Article

Data on Connexin 43 hemichannels regulation of cellular redox in lens

Yumeng Quan[a,b], Yu Du[a], Changrui Wu[a], Sumin Gu[a], Jean X. Jiang[a,*]

[a] Department of Biochemistry and Structural Biology, University of Texas Health Science Center, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, USA
[b] Department of Ophthalmology, First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi, China

ARTICLE INFO

Article history:
Received 13 October 2021
Revised 6 November 2021
Accepted 9 November 2021
Available online 13 November 2021

Keywords:
Connexin
Hemichannel
Redox metabolite
Redox regulation
Lens

ABSTRACT

This article describes a dataset that is related to the research paper "Connexin hemichannels regulate redox potential via metabolite exchange and protect lens against cellular oxidative damage". Growing evidence demonstrates that oxidative stress is a key event in cataract formation. Hemichannels (HCs) formed by Connexin (Cx) 43, a Cx subtype only present in the epithelium of lens tissue, mediate the exchange of small molecules between the intracellular and extracellular environments, including redox-related metabolic molecules, such as glutathione (GSH) and reactive oxygen species (ROS). Here, we used a Cx43 heterozygous mouse model, Cx43E2 antibody (a specific Cx43 HC blocker), and knocked down Cx43 expression by siRNA in human lens epithelial HLE-B3 cells to assess the oxidative response of Cx43 HCs to H_2O_2 and UVB radiation. Western blot analysis of heterozygous Cx43-null ($Cx43^{+/-}$) mouse lenses showed the haploinsufficiency of Cx43 protein. We further assessed antioxidative gene expression in response to H_2O_2 and UVB radiation treatment in the Cx43-deficient lens epithelial cells. This dataset will be useful for understanding the critical role of Cx43 HCs in maintaining redox homeostasis in the lens under oxidative stress.

DOI of original article: 10.1016/j.redox.2021.102102
* Corresponding author.
E-mail address: jiangj@uthscsa.edu (J.X. Jiang).

https://doi.org/10.1016/j.dib.2021.107572

Specifications Table

Subject	Cell Biology
Specific subject area	Connexin hemichannels in lens redox homeostasis
Type of data	Table Image Graph
How the data were acquired	Microscopic observation, Western blot analysis, qRT-PCR analysis, Statistical analysis: GraphPad Prism software.
Data format	Raw Analyzed
Description of data collection	The wild type and heterozygous Cx43-null mouse lenses were dissected out of the eyeball, kept in pre-cold PBS for the following experiments. The protein expression was determined by western blot analysis. Human lens epithelial HLE-B3 cells were cultured in Eagle's Minimum Essential Medium (EMEM) containing 20% FBS and penicillin/streptomycin in an incubator at 37°C. Intracellular H_2O_2, ROS and GSH levels in cells were determined by fluorescence-based probes in live cells, and microscopic observation was applied to analyze the fluorescent images.
Data source location	Department of Biochemistry and Structural Biology, University of Texas Health Science Center, San Antonio, TX, USA.
Data accessibility	With the article.
Related research article	Quan Y, Du Y, Wu C, Gu S, Jiang JX. Connexin hemichannels regulate redox potential via metabolite exchange and protect lens against cellular oxidative damage. Redox Biol. 46 (2021) 102102. https://doi.org/10.1016/j.redox.2021.102102

Value of the Data

- This dataset is useful for expanding our understanding of the role of lens epithelium under oxidative stress with aging.
- These data will be beneficial to researchers and scientists whose research interest focuses on age-related cataract and lens redox homeostasis.
- These data could be used as basic research information and will provide support for further investigation of the role of Cx43 in lens epithelium and redox-related disease.

1. Data Description

These data provide additional value to our research article "Connexin hemichannels regulate redox potential via metabolite exchange and protect lens against cellular oxidative damage," which demonstrated that Connexin 43 hemmichannels (Cx43 HCs) are activated in response to oxidative stress and mediate the exchange of oxidants and antioxidants in lens epithelium under oxidative stress [1]. Heterozygous Cx43-null mouse lenses and a human lens epithelial cell line were used to determine the protective effect of functional Cx43 HCs on a human epithelium exposed to oxidative stress.

Lenses from four-month-old mice were used to determine the Cx43 protein expression in membrane extracts. Fig. 1 describes the difference of Cx43 expression between wild type (WT) and heterozygous Cx43-null ($Cx43^{+/-}$) mouse lenses. This figure consists of two parts; the upper panel (Fig. 1A) shows the western blot image of Cx43 and β-actin signals from membrane extracts of four-month-old mouse lenses. The raw images for this western blot can be found in

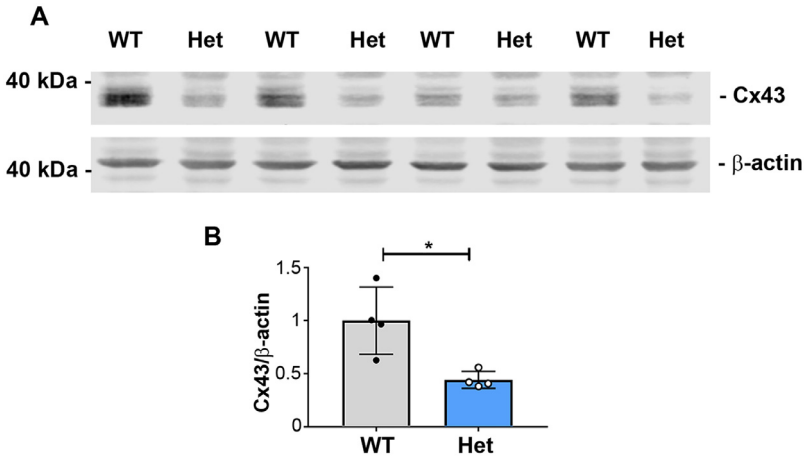

Fig. 1. Cx43 expression in WT and heterozygous Cx43-null mouse lenses. Four-month-old mouse lenses were isolated and membrane extracts were prepared. The membrane extracts were then subjected to immunoblotting with anti-Cx43CT antibody (1:300 dilution) and anti-β-actin antibody (1:5000) (A). The intensity of bands was quantified using NIH ImageJ software and the ratio of Cx43 to β-actin was applied (B). The data are presented as the mean±SEM. (n=4). *, $P < 0.05$ (Student T-test).

Supplementary File 1 (Fig. S1), which shows Cx43 protein (the upper panel) alongside β-actin and GAPDH proteins (the lower panel). Cx43, β-actin, and GAPDH were detected as protein bands of ~39 kDa, ~42 kDa, and 37 kDa, respectively. This observation is in accordance with the calculated molecular weight for Cx43 (39 kDa) (non-phosphorylated isoform) [2], β-actin (42 kDa), and GAPDH (37 kDa). The lower panel (Fig. 1B) is the quantification of the protein levels of Cx43 and β-actin shown in Fig. 1A, and the data are presented as the normalized ratio of Cx43 to β-actin. The data indicated that the protein expression of Cx43 reduced in the membrane fraction of $Cx43^{+/-}$ mouse lenses. The band intensity was quantified using Image studio Lite software and the raw data of quantification is shown in Supplementary File 1 (Table S1).

Lenses are constantly exposed to ultraviolet (UV) radiation, which generates abundant reactive oxygen species (ROS) inside the lens as well as its surrounding environment. To maintain redox homeostasis and transparency, the lens develops a powerful antioxidant defense system consisting of one of the highest tissue levels of reduced GSH (~4-6 mM) [3]. To determine the effect of Cx43 on the redox state of the lens epithelium, we detected the intracellular ROS and GSH in human lens epithelial HLE-B3 cells. Fig. 2 shows the intracellular ROS (Fig. 2A) and GSH (Fig. 2B) levels in Cx43-knockdown live HLE-B3 cells after 4 h of oxidative stress treatment. H_2O_2 treatment or UVB radiation was applied to induce cellular oxidative stress in this assay. The data indicated that elevated ROS levels and reduced GSH levels were observed in Cx43-deficient lens epithelium. The left panel (Fig. 2A) shows the fluorescence-based ROS signal (green) by incubating with Carboxy-H_2DCFDA (ROS probe). The left panel (Fig. 2B) shows the fluorescence-based GSH signal (green) by incubating with ThiolTracker™ (GSH probe). The raw microscopic images of intracellular ROS (Fig. S2 upper panel) and GSH (Fig. S2 lower panel) can be found in Supplementary File 2. All individual raw microscopic images in Fig. S2 are included as raw files in Supplementary File 2. Both cellular ROS and GSH levels were determined by measuring the fluorescence intensity in cells (Fig. 2A, B, right panels), and the quantification of raw data is shown in Supplementary File 2 (Table S2).

It has been reported that Cx HCs could serve as a transport channel for redox-related molecules. In Fig. 3, to determine the effect of Cx43 HCs, Cx43E2 antibody, a specific Cx43 HCs blocker [4] was used to specifically inhibit Cx43 HCs. This figure consists of three parts. The upper two figures (Fig. 3A and B) show the intracellular ROS level in human lens epithelial HLE-B3 cells with the inhibition of Cx43 HCs. The ROS probe mentioned in Fig. 2 was used to

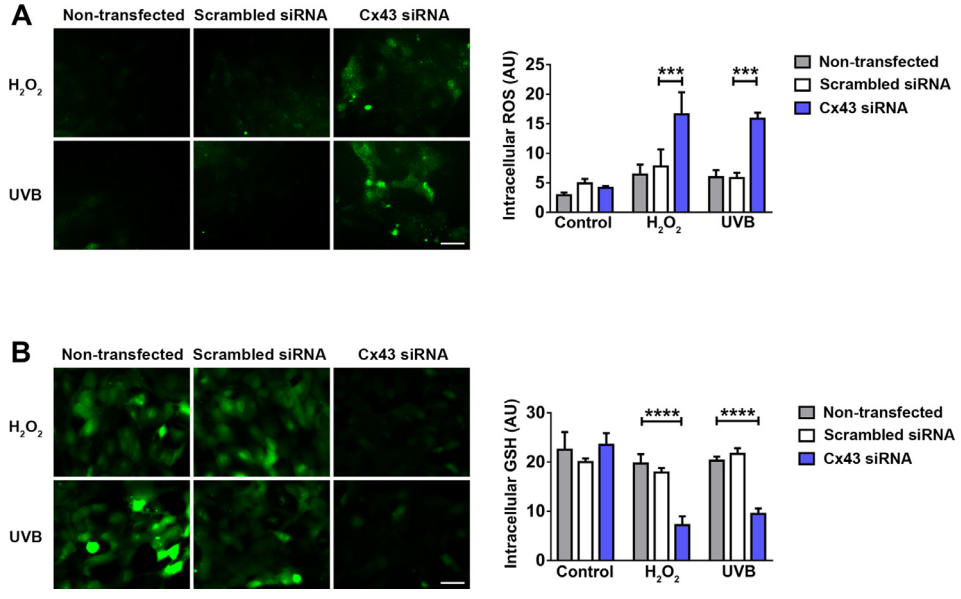

Fig. 2. Intracellular ROS and GSH levels in Cx43 knockdown human lens epithelium. HLE-B3 cells were transfected with Silencer™ Negative Control or Cx43 siRNA and cultured until ready for the experiment. Cells were treated with H_2O_2 or UVB radiation for 4 h and subjected to incubation of fluorescence probes Carboxy-H_2DCFDA (ROS probe) (A, left panel) or Thioltracker™ (GSH probe) (B, left panel). The intracellular ROS (A, right panel) and GSH (B, right panel) levels were determined by measuring the fluorescence intensity in cells. The data are presented as the mean±SEM. (n=3). ***, $P < 0.001$; ****, $P < 0.0001$ (Two-way ANOVA).

measure the ROS level in this assay. The major difference between the two datasets in Fig. 3 is the treatment period; 1 hr in Fig. 3A and 3 h in Fig. 3B. It is observed that the ROS levels in Cx43E2 treated cells were elevated after both UVB radiation and H_2O_2 treatment for 3 h. The trend of ROS levels in Cx43 HC-inhibited cells with these two treatments for 3 h (Fig. 3B) is similar to that observed following these two treatments for 4 h in the Cx43-deficient lens epithelium (Fig. 2A). However, the data in Fig. 3A shows a reduced ROS level after H_2O_2 treatment and an elevated ROS level after UVB radiation in Cx43E2 treated cells compared to the Cx43 WT control groups. Fig. 3C shows the intracellular H_2O_2 level with the inhibition of Cx43 HCs in HLE-B3 cells after H_2O_2 treatment for 30 min. The left panel in Fig. 3C shows the fluorescence sensor-based H_2O_2 signal (green) where the Fluorescent Peroxide Sensor (H_2O_2 probe) was used to determine the H_2O_2 level in cells. The raw microscopic images of intracellular H_2O_2 can be found in Supplementary File 3 (Fig. S3) and all individual raw microscopic images in Fig. S3 are included as raw data files in Supplementary File 3. All cellular fluorescence-based signals for ROS and H_2O_2 were determined by measuring the fluorescence intensity in cells and the raw quantification data is shown in Supplementary File 3 (Table S3).

Fig. 4 shows the results of qRT-PCR analysis for anti-oxidative gene expression in HLE-B3 cells in response to H_2O_2 treatment or UVB radiation. Cx43E2 antibody was used to block Cx43 HCs in this assay. This figure contains five panels, which show mRNA expression of GPX8 (Fig. 4A), GSTM2 (Fig. 4B), GSR (Fig. 4C), MGST1 (Fig. 4D), and HMOX1 (Fig. 4E). Each panel includes two graphs corresponding to mRNA expression under H_2O_2 treatment or UVB radiation, respectively. The raw calculation data are shown in Supplementary File 4 (Table S4).

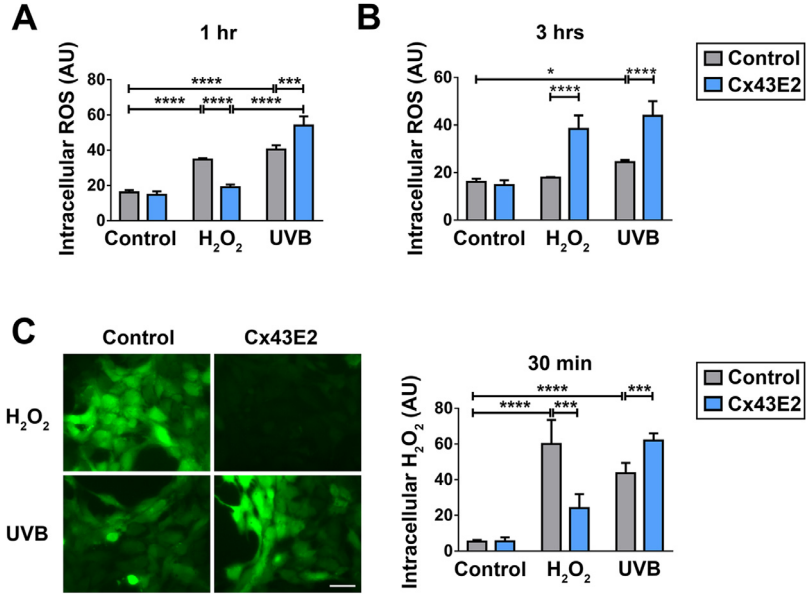

Fig. 3. Intracellular ROS and H_2O_2 levels in impaired Cx43 HCs human lens epithelium. HLE-B3 cells were pre-treated with Cx43E2 antibody for 30 min followed by treatment with H_2O_2 or UVB radiation for various periods. Carboxy-H_2DCFDA (ROS probe) was used to assess the ROS levels for 1 hr (A) or 3 h (B) in cells. Fluorescent Peroxide Sensor (H_2O_2 probe) (green signal, C left panel) was applied to determine the intracellular H_2O_2 level after 30 min of H_2O_2 treatment in live cells, and the H_2O_2 level was determined by measuring the fluorescence intensity in cells (C, right panel). The data are presented as the mean±SEM. (n=3). *, $P < 0.05$; ***, $P < 0.001$; ****, $P < 0.0001$ (Two-way ANOVA).

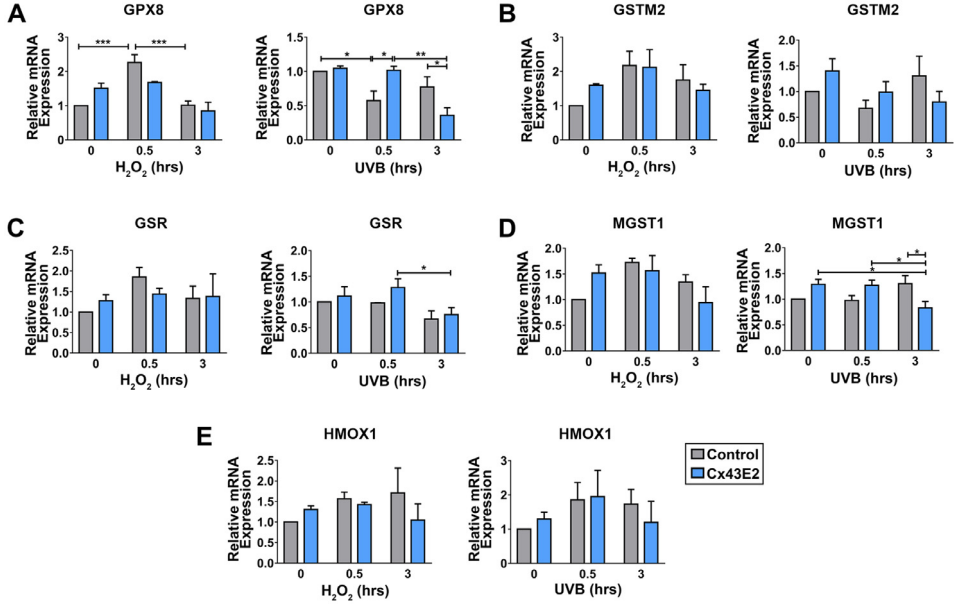

Fig. 4. Anti-oxidative gene expression in response to H_2O_2 treatment and UVB radiation. HLE-B3 cells were treated with H_2O_2 or UVB radiation for 0, 0.5, or 3 h after pre-incubation of Cx43E2 antibody for 30 min. RNA extracts were processed and subjected to qRT-PCR analysis for mRNA expression of GPX8 (A), GSTM2 (B), GSR (C), MGST1 (D), and HMOX1 (E). The data are presented as the mean±SEM. (n=3). *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$ (Two-way ANOVA).

2. Experimental Design, Materials and Methods

2.1. Cell culture

To perform analysis in lens epithelium, human lens epithelial HLE-B3 cells [5] were purchased from American Type Culture Collection (ATCC, Rockville MD, USA), and cells after six passages were used for all cell experiments. The frozen cell vials stored in liquid nitrogen were immediately placed into a 37°C water bath and thawed rapidly (< 1 min). The cells were then plated in a 60 mm culture dish with Eagle's Minimum Essential Medium (EMEM, ATCC, Rockville MD, USA) containing 20% FBS (Hyclone Laboratories, Logan, UT, USA) and penicillin/streptomycin (Invitrogen, Carlsbad, CA, USA) at a high cell density, which helps cell recovery. The culture media was changed the next day. The cells were passaged twice with TrypLE (Invitrogen, Carlsbad, CA, USA) and maintained either in a culture dish or a culture plate in an incubator at 37°C supplied with 5% CO_2, and the culture media was changed every other day. 85% confluent cells were trypsinized and plated into 6, 12, or 96-well plates at required cell densities. The number of living cells was counted using a Countess II FL Automated Cell Counter (Invitrogen, Carlsbad, CA, USA) before seeding cells.

2.2. Generation of heterozygous Cx43-null ($Cx43^{+/-}$) mice

The breeding pairs of $Cx43^{+/-}$ mice [6] were kindly provided by Roberto Civitelli at Washington University School of Medicine. The heterozygous Cx43-null mice were generated by crossing C57BL/6 $Cx43^{+/+}$ mice with C57BL/6 $Cx43^{+/-}$ mice. The mice used in our study were housed in a pathogen-free environment at the Association for Assessment and Accreditation of Laboratory Animal Care–accredited UTHSCSA animal facility following the National Institutes of Health Guidelines for the Care and Use of Laboratory Animals. The experimental protocols for animals were approved by the UTHSCSA Institutional Animal Care and Use Committee (IACUC). The experiments were conducted blindly for $Cx43^{+/+}$ and $Cx43^{+/-}$ mouse models and the identity of the mice became available after completion of the data analysis. Genotyping was performed by real-time PCR technique using genomic DNA isolated from mouse tails and corresponding primers were synthesized by Integrated DNA Technologies, Inc (Coralville, Iowa, USA).

2.3. Lens isolation, preparation of membrane extracts, and western blot analysis

The lenses were isolated from four-month-old mice and crude membrane extracts were prepared. A small incision was carefully made on the side of the optic nerve at the posterior of eyeball. The lens was carefully dissected under a dissection microscope without any damage made during this process [7]. Isolated lenses were kept in pre-cold PBS and homogenized using a mortar and pestle in ice-chilled lysis buffer (5 mM Tris, 5 mM EDTA, and 5 mM EGTA, pH 8.0) containing proteinase inhibitors (5 mM NEM, 2 mM PMSF, 1 mM Na_3VO_4, and 0.2 mM leupeptin). Lens lysate was centrifuged for 5 min at 1,000 rpm to remove cell debris and the supernatant was then subjected to ultracentrifugation at 45,000 rpm for 30 min (TLA55 rotor, Beckman Coulter, Brea, CA, USA) at 4°C. The pellets containing crude membrane extract were resuspended in lysis buffer plus protease inhibitors, and 100 µL of lysis buffer was added into each sample to resuspend the pellet. The crude membrane extracts were then dissolved by the addition of 1% SDS for 5 min at room temperature and boiled for 5 min. Crude membrane extracts were then centrifuged at 10,000 g for 3 min at 4°C, and the supernatants were collected and used for the immunoblotting assay. The protein amount in membrane extracts was quantified using the microBCA assay (Pierce, Rockford, IL, USA), and bovine serum albumin was used as a protein standard. Equal amounts of proteins were loaded onto 10% SDS-PAGE gel and then electrotransferred onto a nitrocellulose membrane. The membrane was immunoblotted with rabbit anti-Cx43CT

Table 1
The list of DNA primers.

Name	Sequence
Gpx1-F	5'-GAATGTGGCGTCCCTCTG-3'
Gpx1-R	5'-CTCTTCGTTCTTGGCGTTCT-3'
Sod1-F	5'-GTGCAGGGCATCATCAATTTC-3'
Sod1-R	5'-GGCCTTCAGTCAGTCCTTTAAT-3'
GSTM2-F	5'-CCCTGAAATGCTGAAGCTCTA-3'
GSTM2-R	5'-TGGTTTCTCTCAAGGACATCATAA-3'
MGST1-F	5'-CACCCAGGTAATGGATGATGAA-3'
MGST1-R	5'-TGCTACACAGTCTTCTGGATTG-3'
GPX8-F	5'-TCTGAAGGAGAACCTGCATTTA-3'
GPX8-R	5'-CCCTCAGGGTTGACAAGATAC-3'
HMOX1 -F	5'-TCAGGCAGAGGGTGATAGAA-3'
HMOX1 -R	5'-GCTCCTGCAACTCCTCAAA-3'
GSR-F	5'-CGGTGCCAGCTTAGGAATAA-3'
GSR-R	5'-GCCATCTCCACAGCAATGTA-3'
18S rRNA-F	5'-ACAGGTCTGTGATGCCCTTAGA-3'
18S rRNA-R	5'-GCAAGCTTATGACCCGCACTTA-3'

(1:300 dilution, developed in our laboratory) [8], mouse anti-β-actin antibody (1:5000 dilution, Invitrogen, Carlsbad, USA), and mouse anti-GAPDH antibody (1:5000 dilution, Invitrogen, Carlsbad, USA). Primary antibodies were detected with goat anti-rabbit IgG conjugated IRDye® 800CW, or goat anti-mouse IgG conjugated IRDye® 680RD (1:15000 dilution, Lincoln, NE, USA) using a Licor Odyssey Infrared Imager (Lincoln, NE, USA). The intensity of the protein bands was quantified by Image Studio Lite software (LI-COR Biosciences).

2.4. RNA extraction and qRT-PCR analysis

HLE-B3 cells were cultured in 6-well plates and treated with H_2O_2 or UVB radiation for 0, 0.5, or 3 hr. The cells in Cx43E2-treatment groups were pre-incubated with Cx43E2 antibody for 30 min at 37°C. TRIzol Reagent (Molecular Research Center, Cincinnati, OH, USA) was used to isolate total RNAs from HLE-B3 cells under various treatments. 250 ng of total RNA were reverse-transcribed into complementary DNA by using a High-Capacity RNA-to-cDNA Kit (Applied Biosystems®, Life Technologies, Carlsbad, CA) according to the manufacturer's instructions. qRT-PCR was performed using an ABI 7900 PCR device (Applied Biosystems®, Life Technologies, Carlsbad, CA) with Maxima SYBR Green/ROX qPCR Master Mix (2X) (Fisher Scientific, Hampton, NH, USA). A two-step PCR protocol (95°C, 10 sec and 60°C, 30 sec) was applied in this assay. The primer sequences used in this assay are listed in Table 1. The 18S rRNA gene was used as a housekeeping gene control and RNA expression level was calculated using the ($2^{-\Delta\Delta Ct}$) ratio.

2.5. Cx43 siRNA treatment

Cx43 small interfering RNA (siRNA) and Silencer™ Negative Control were purchased from Ambion (Life Technologies, Carlsbad, CA, USA). HLE-B3 cells were trypsinized, resuspended in antibiotic-free Opti-MEM reduced-serum medium (Invitrogen, Carlsbad, CA), and then transiently transfected with 30 nM Silencer™ Negative Control siRNA or Cx43 siRNA using Lipofectamine RNAiMAX transfection reagent (Invitrogen, Carlsbad, CA, USA), according to the manufacturer's instructions. 48 hr after the transfection, cells were treated with H_2O_2 or UVB radiation as dictated in the experimental design and assessed for the levels of intracellular ROS and GSH utilizing fluorescence-based probes.

2.6. H_2O_2 and UVB radiation treatment

HLE-B3 cells were plated into 6, 12, or 96-well plates and cultured to 85% confluency, and then subjected to H_2O_2 or UVB radiation. 30% H_2O_2 was diluted, and a fresh H_2O_2 working solution was prepared at the time of use. An incubator with a 302nm UV transilluminator supplied with 37°C and 5% CO_2 was used for UVB radiation treatment. The incubator was pre-exposed to 302nm UV radiation for 20 min, then the UV exposure intensity and the incubator temperature were measured and kept stable before placing the cell culture plates into the incubator. The treated cells were kept in a standard incubator supplied with 5% CO_2 at 37°C for the subsequent experiments.

2.7. Measurement of cellular H_2O_2, ROS and GSH

Fluorescence-based probes, Fluorescent Peroxide Sensor (Sigma-Aldrich, St. Louis, MO), Carboxy-H_2DCFDA (Invitrogen, Carlsbad, CA, USA), and ThiolTracker™ (Thermo Fisher, Waltham, MA, USA), were applied to determine the intracellular H_2O_2, ROS, or GSH levels in live cells, respectively [9]. HLE-B3 cells were pre-incubated with siRNA or Cx43E2 antibody in 12-well or 96-well plates and then treated with H_2O_2 or UVB radiation. After various treatments, cells were rinsed with pre-warm HBSS and incubated with 10 μM Fluorescent Peroxide Sensor for 8 min at 37°C, or with 10 μM Carboxy-H_2DCFDA or ThiolTracker™ for 30 min at 37°C. The cells incubated with Fluorescent Peroxide Sensor or Carboxy-H_2DCFDA were then gently rinsed with warm HBSS twice for 3 min at room temperature, kept in equal amounts of HBSS and immediately followed by fluorescence microscopy analysis (Keyence BZ-X710). ThiolTracker™ incubated cells were rinsed twice for 3 min at room temperature with warm HBSS and then fixed with 2% paraformaldehyde (PFA) before fluorescence microscopy analysis (Keyence BZ-X710). The same imaging parameters were applied for both the control and treated samples in each experiment. For each well of a 96-well plate, one field was taken for H_2O_2 measurement. For ROS or GSH measurement, three fields in each well of a 12-well plate were captured. The images analysis was conducted with NIH ImageJ software. For each field, we measured the average pixel density of the fluorescence intensity of 30 random cells and 5 background regions without cells. The mean background value was obtained by the density of pixels taken from each field and then averaged. The final value was obtained by subtracting the mean background value from the cell value.

2.8. Statistical analysis

GraphPad Prism 7 software (GraphPad Software, La Jolla, CA) was used to perform the statistical analyses for all data. All data are reported as mean ± SEM from at least three measurements for each experiment. The mean difference of two sets of data was analyzed using Student T-test. The mean of quantitative variable changes between two categorical independent variables was analyzed using Two-way ANOVA and Sidak multiple comparison tests. Difference of $P < 0.05$ was considered as statistically significant. Asterisks in all figures were used to describe the value levels of significant statistical differences compared to controls: *, $P < 0.05$; **, $P < 0.01$; *** $P < 0.001$; ****, $P < 0.0001$.

Ethics Statements

This study utilized animals and all animal experiments complied with the National Institutes of Health guide for the care and use of laboratory animals. Experimental protocols were approved by the UTHSCSA IACUC (Approval number: 19980015AR).

Declaration of Competing Interest

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

CRediT Author Statement

Yumeng Quan: Investigation, Data curation, Formal analysis, Writing – original draft; **Yu Du:** Investigation, Data curation, Formal analysis; **Changrui Wu:** Conceptualization, Writing – review & editing; **Sumin Gu:** Writing – original draft, Writing – review & editing; **Jean X. Jiang:** Funding acquisition, Visualization, Writing – original draft, Writing – review & editing.

Acknowledgments

The authors thank Dr. Roberto Civitelli at the Washington University School of Medicine for generously providing $Cx43^{+/-}$ mice. Dr. Eduardo R. Cardenas and Dr. Francisca M. Acosta for proofreading and editing. The study was supported by NIH RO1 EY012085 and Welch Foundation grant AQ-1507 to J.X.J.

Supplementary Materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.dib.2021.107572.

References

[1] Y. Quan, et al., Connexin hemichannels regulate redox potential via metabolite exchange and protect lens against cellular oxidative damage, Redox Biol. 46 (2021) 102102.
[2] R. Kadle, J.T. Zhang, B.J. Nicholson, Tissue-specific distribution of differentially phosphorylated forms of Cx43, Mol. Cell Biol. 11 (1) (1991) 363–369.
[3] V.N. Reddy, Glutathione and its function in the lens–an overview, Exp. Eye Res. 50 (6) (1990) 771–778.
[4] A.J. Siller-Jackson, et al., Adaptation of Connexin 43-hemichannel prostaglandin release to mechanical loading, J. Biol. Chem. 283 (39) (2008) 26374–26382.
[5] U.P. Andley, et al., Propagation and immortalization of human lens epithelial cells in culture, Invest. Ophthalmol. Vis. Sci. 35 (7) (1994) 3094–3102.
[6] A.G. Reaume, et al., Cardiac malformation in neonatal mice lacking Connexin43, Science 267 (5205) (1995) 1831–1834.
[7] S. Gu, et al., Connexin 50 and AQP0 are Essential in Maintaining Organization and Integrity of Lens Fibers, Invest. Ophthalmol. Vis. Sci. 60 (12) (2019) 4021–4032.
[8] B. Cheng, et al., Expression of functional gap junctions and regulation by fluid flow in osteocyte-like MLO-Y4 cells, J. Bone Miner. Res. 16 (2) (2001) 249–259.
[9] D. Wu, P. Yotnda, Production and detection of reactive oxygen species (ROS) in cancers, J. Vis. Exp. (57) (2011).