https://doi.org/10.52973/rcfcv-e35659 Revista Científica, FCV-LUZ / Vol. XXXV

Effect of Indomethacin and Dimethylsulfoxide on the oxidative status of humor aqueous in ocular burn

Efecto de la indometacina y el dimetilsulfóxido sobre el estado oxidativo del humor acuoso en quemaduras oculares

***Semih Altan*[1]*, *Feray Altan*[2], *Zeki Ogurtan*[3]**

[1]*Dokuz Eylul University, Faculty of Veterinary Medicine, Department of Surgery. Izmir, Türkiye.*
[2]*Dokuz Eylul University, Faculty of Veterinary Medicine, Department of Pharmacology and Toxicology. Izmir, Türkiye.*
[3]*Selcuk University, Faculty of Veterinary Medicine, Department of Surgery. Konya, Türkiye.*
**Corresponding author: semih.altan@deu.edu.tr*

ABSTRACT

The study aims to investigate the efficacies of dimethyl sulfoxide (DMSO) and indomethacin on the oxidative status in the aqueous humor of rabbits' eyes with experimentally induced hydrofluoric acid ocular burns. For this purpose, thiobarbituric acid (TBARS) and total antioxidant status (TAS) were measured. Seventy–two male New Zealand rabbits were allocated into four groups, each containing 18 rabbits (Group D, I, DI, and C). After general anesthesia, 2% hydrofluoric (HF) acid was dropped into the right eye for 60 s, creating a chemical burn. Solution of 40% DMSO (4 drops of QID) and 0.1% indomethacin (4 drops of QID) were used alone and in combination. At the end of the follow–up periods (2, 7, and 14 treatment days), animals were euthanized, and the humor aqueous was collected from the burned eyes by anterior chamber paracentesis. The TBARS and TAS levels were assessed using enzyme–linked immunosorbent assay (ELISA) in aqueous humor. On days 7 and 14, the TBARS levels in the aqueous humor of group D differed significantly from those in the other groups (P<0.05). Additionally, TAS values were considerably higher in groups D and C compared to groups I and DI (P<0.05). This study is thought to be a model for further studies in ocular diseases such as chemical eye burns, which may result in impaired ocular healing due to oxidative stress. DMSO could decrease oxidative stress and improve tissue healing of chemical eye burns.

Key words: Antioxidants; DMSO; humor aqueous; hydrofluoric acid; Indomethacin

RESUMEN

El estudio tuvo como objetivo evaluar el efecto del dimetilsulfóxido (DMSO) y la indometacina sobre el estado oxidativo del humor acuoso ocular de conejos con quemaduras oculares inducidas por ácido fluorhídrico. Para ello, se midieron el ácido tiobarbitúrico (TBARS) y el estado antioxidante total (TAS). Setenta y dos conejos machos de raza Nueva Zelanda se dividieron en cuatro grupos, con 18 conejos en cada grupo (Grupo D, I, DI y C). Tras la anestesia general, se les aplicó ácido fluorhídrico (HF) al 2 % en el ojo derecho durante 60 s, lo que provocó una quemadura química. Se utilizó una solución de DMSO al 40 % (4 gotas cuatro veces al día) e indometacina al 0,1 % (4 gotas cuatro veces al día), tanto solas como en combinación. Al final de los períodos de seguimiento (2, 7 y 14 días de tratamiento), los animales fueron sacrificados y se recogió el humor acuoso de los ojos quemados mediante paracentesis de la cámara anterior. La TBARS y la TAS se midieron mediante un ensayo inmunoabsorbente ligado a enzimas (ELISA) en el humor acuoso. Se observó una diferencia significativa entre la TBARS en el humor acuoso del grupo D, mayor que en los otros grupos, en los días 7 y 14 (P<0,05). Los valores de TAS fueron mucho mayores en los grupos D y C que en los grupos I y DI (P<0,05). Este estudio se considera un modelo para futuros estudios sobre enfermedades oculares, como las quemaduras químicas oculares, que pueden afectar la cicatrización ocular debido al estrés oxidativo. El DMSO podría disminuir el estrés oxidativo y mejorar la cicatrización tisular de las quemaduras químicas oculares.

Palabras clave: Antioxidantes; DMSO; humor acuoso; ácido fluorhídrico; Indometacina

Received: 24/03/2025 Accepted: 26/06/2025 Published: 14/07/2025

INTRODUCTION

The eye is one of the organs that directly affects a living being's relationship with the external environment. Ocular traumas that lead to a decrease or complete loss of function can significantly change one's quality of life [1, 2]. Although considerable progress has been made in its prevention and management in recent years, chemical eye burns continue to be one of the most prevalent causes of ocular trauma, resulting in reduced or total loss of visual function [3, 4]. Inflammation, tissue damage, infection, and oxidative stress occur in the eye immediately after a severe injury, such as one caused by liquid, aerosol, or solid materials [5, 6, 7]. One of the primary causes of pathophysiological changes observed immediately following a severe injury, like a chemical eye burn, is oxidative stress [7].

Oxidative stress (OS) is a consequence of an imbalance between the detrimental effects of reactive oxygen species (ROS) and the cellular antioxidant capacity [8]. Various mechanisms have been suggested to clarify the possible relationship between oxidative stress and ocular burns. ROS generated at the molecular level in burn cases may harm corneal tissues by degrading collagen and oxidizing unsaturated fatty acids in the cell membrane of epithelial–stromal cells [9]. Antioxidative markers, such as total antioxidant status (TAS) and thiobarbituric acid–reactive substances (TBARS), can be assessed as indicators of lipid, protein, and DNA damage caused by oxidative stress [10]. Tissues have various enzymatic and non–enzymatic antioxidant defense systems. In chemical eye burns, a reduction in the antioxidant protection mechanism and increased lipid peroxidation are observed [11]. Numerous researchers have emphasized the correlation between the pro–oxidative state and the imbalance of the antioxidant defense mechanism, commonly referred to as oxidative stress (OS), in chemical eye burns [11, 12]. Aqueous humor is a clear, slightly alkaline fluid produced and secreted by the ciliary bodies, lining the space between the cornea and the lens. It is a crucial component of the ocular surface and is rich in low molecular weight, water–soluble antioxidants that aid the corneal defense mechanisms against oxidative stress [13]. Although the changes in humor aqueous concentration of these markers, which are implicated in the pathogenesis of alkaline eye burns [11], are known, there remain uncertainties regarding acidic eye burns.

Hydrofluoric acid (HF) is a hazardous substance widely utilized in industrial settings and is known to cause chemical burns [14]. Ocular chemical burns from HF exposure represent an ocular emergency that requires immediate treatment to eliminate the inflammatory agent and manage the resulting inflammation [10, 15, 16, 17]. Antioxidants and anti–inflammatories such as dimethyl sulfoxide and indomethacin are employed in treatment to avert irreversible dystrophic changes in the eye tissue, such as alkaline burns [5, 15, 18].

Although it is estimated how DMSO and indomethacin affect the oxidative status of the humor aqueous in HF eye burns, the specific mechanism is unknown. This study was conducted to investigate how DMSO and indomethacin affect the oxidant–antioxidant balance (TBARS and TAS) in the aqueous humor of rabbits with eye burns induced by hydrofluoric acid.

MATERIALS AND METHODS

Animals and group design

Humor aqueous samples were obtained from 72 male New Zealand breed rabbits used in the authors' previous research [15]. Briefly, after general anesthesia with 10 mg·kg^{-1} of 2% Xylazine HCL (Rompun, Bayer, Türkiye), followed by 30 mg·kg^{-1} of 10% Ketamine HCL (Ketasol, Interhas, Türkiye), the right eye was subjected to burning through the instillation of 0.05 mL of 2% HF (38–40% Merck, USA) for just 60 s [15]. Afterward, the eye was gently rinsed with 500 mL of saline. Following this, rabbits were allocated into four groups each containing 18 rabbits. Group D received 40% DMSO (99.9%, Merck, USA; four drops QID), Group I received 0.1% indomethacin (Indocolir 5 mL, Abdi–İbrahim, Türkiye; four drops QID), and Group DI received DMSO along with indomethacin in the same doses as Groups D and I. Group C did not receive any therapeutic agents and functioned as the control group. These groups were divided into three subgroups based on follow–up periods of 2, 7, and 14 treatment days (d). Dipyrone (Devalgine 0.5 g·mL^{-1}, Vetaş, Türkiye) was administered intramuscularly at 14 mg·kg^{-1} before the burning and continued every 6 hours for two days.

Subsequent to the euthanasia of the animals at the end of the follow–up periods, aqueous humor was collected through anterior chamber paracentesis, conducted with a 25G needle attached to a 1 mL syringe from the burned eyes (FIG. 1). The samples were transferred to centrifuge tubes labeled accordingly to evaluate TAS and TBARS. The control values of humor aqueous were collected from six healthy left eyes of rabbits. The samples were frozen at -80°C (Nüve DF 490, İstanbul, Türkiye) until further analysis.

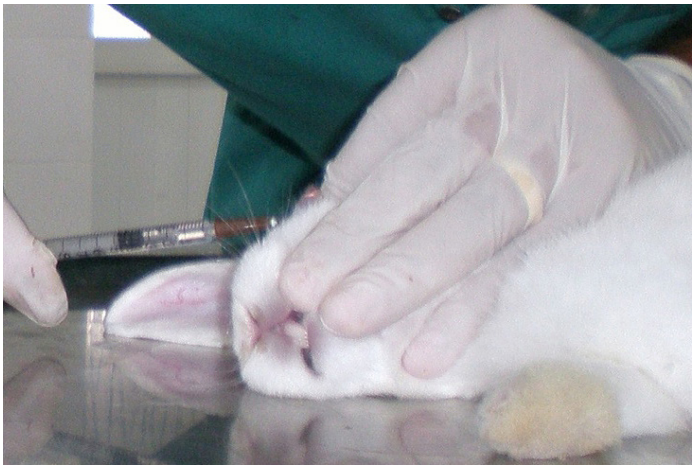

FIGURE 1. Parasynthesis of the humor aqueous

Measurement of oxidative stress status

Commercial enzyme–linked immunosorbent assay (ELISA) kits (SunRed Biotechnology Company, Shanghai, China) were used to measure oxidative levels through TBARS and antioxidant levels through TAS. Both tests were performed according to the kit protocol.

Statistical analysis

Statistical analyses were conducted using SPSS 20.0 software (SPSS 20.0, Chicago, IL, USA). Analysis of variance (ANOVA) was utilized to determine whether there was a difference between treatment days and groups. Subsequently, Duncan test for multiple comparisons was applied to assess the significance level of the differences between the mean values in groups. $P<0.05$ was considered statistically significant. Results are presented as mean ± standard error (SE).

RESULTS AND DISCUSSION

Thiobarbituric acid reactive substances and total antioxidant status values are shown in TABLES I and II and FIGS. 2 and 3. TBARS values of all groups were maximum on d 2 and minimum on d 14. TBARS values were less marked in group D than in groups C and DI and with no observed difference between groups D and I on d 2 ($P>0.05$), and group D was less marked than other groups with a significant difference at d 7 and 14 ($P<0.05$).

Total antioxidant status was significantly higher in groups D and C than in groups I and DI ($P<0.05$), with no statistically significant difference between groups D and C and between groups I and DI on d 2 ($P>0.05$). TAS values were much higher and statistically significant in the group DI than in other groups on d 14 ($P<0.05$).

The eye is particularly vulnerable to oxidative damage induced by reactive oxygen species generated by exposure to various chemical agents. In the aqueous humor, reactive oxygen species are constantly generated through hydrogen peroxide, superoxide anion, singlet oxygen, and peroxyl radicals. The humor aqueous, which serves as the eye's physical and chemical barrier, is crucial in combating free radicals through its enzymatic and non–enzymatic antioxidants. Additionally, it is important for the nutrition and protection of the anterior lens epithelium and corneal endothelium.

One of its functions is to remove metabolic waste and biochemical byproducts generated by the cornea and lens. Evaluating ocular biomarkers in aqueous humor provides valuable information for revealing eye disorders [10, 19]. Recently, studies have shown that the composition of aqueous humor is affected by the pathophysiology of diseases requiring ocular emergency treatment, such as eye burns [11] and other ocular disorders [20]. Therefore, severe oxidative stress in eye burns can be assessed by measuring the aqueous humor's antioxidant capacity and enzyme activity.

Prior research has demonstrated DMSO and indomethacin's clinical and histopathological effectiveness in treating acidic eye burns [15]. Furthermore, results demonstrating the anti–inflammatory activity of various substances in alkaline eye burns have also been reported [11]. This study investigated the impact of DMSO and indomethacin on the oxidative status of aqueous humor obtained from hydrofluoric acid–induced eye burns (FIG. 3).

TABLE I
TBARS (μM) values of the humor aqueous HF–induced eye burn in rabbits (mean ± SE)

Groups	day 0	day 2	day 7	day 14
C	$0.050 \pm 0.014^{C,a}$	$1.050 \pm 0.122^{A,ab}$	$0.533 \pm 0.076^{B,a}$	$0.179 \pm 0.039^{C,a}$
D	$0.050 \pm 0.014^{B,a}$	$0.604 \pm 0.101^{A,c}$	$0.143 \pm 0.049^{B,b}$	$0.066 \pm 0.007^{B,b}$
I	$0.050 \pm 0.014^{D,a}$	$0.897 \pm 0.072^{A,bc}$	$0.466 \pm 0.07^{B,a}$	$0.229 \pm 0.033^{C,a}$
DI	$0.050 \pm 0.014^{C,a}$	$1.286 \pm 0.157^{A,a}$	$0.340 \pm 0.047^{B,a}$	$0.243 \pm 0.028^{BC,a}$

Different letters in the same line ($^{A, B, C, D}$) and column ($^{a, b, c}$) with statistically significant ($P<0.05$). D: Dimethyl sulfoxide, I: Indomethacin, DI: Dimethyl sulfoxide+ Indomethacin, C: Control

TABLE II
TAS (mmol·L^{-1}) values of the humor aqueous of HF–induced eye burn in rabbits (mean ± SE)

Groups	day 0	day 2	day 7	day 14
C	$9.550 \pm 0.471^{A,a}$	$9.110 \pm 0.475^{AB,ab}$	$6.776 \pm 0.475^{B,a}$	$6.933 \pm 0.609^{B,b}$
D	$9.550 \pm 0.471^{A,a}$	$9.916 \pm 2.845^{A,a}$	$7.876 \pm 0.961^{A,a}$	$5.726 \pm 0.856^{A,b}$
I	$9.550 \pm 0.471^{A,a}$	$4.183 \pm 0.953^{B,b}$	$9.010 \pm 0.436^{A,a}$	$5.723 \pm 1.041^{B,b}$
DI	$9.550 \pm 0.471^{A,a}$	$5.293 \pm 1.382^{B,ab}$	$8.740 \pm 0.836^{A,a}$	$9.700 \pm 0.471^{A,a}$

Different letters in the same line ($^{A, B, C, D}$) and column ($^{a, b, c}$) with statistically significant ($P<0.05$). D: Dimethyl sulfoxide, I: Indomethacin, DI: Dimethyl sulfoxide+ Indomethacin, C: Control

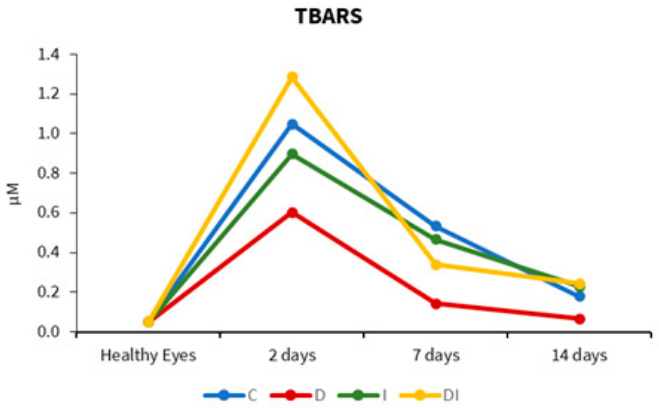

FIGURE 2. Bar graph illustrating the change in TBARS means between days for each group. D: Dimethyl sulfoxide, I: Indomethacin, DI: Dimethyl sulfoxide+ Indomethacin, C: Control

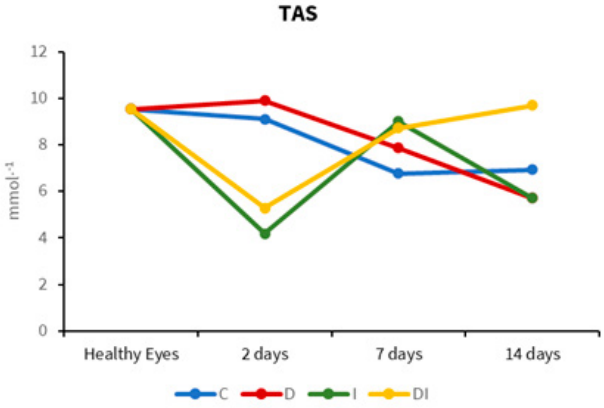

FIGURE 3. Bar graph illustrating the change in TAS means between days for each group. D: Dimethyl sulfoxide, I: Indomethacin, DI: Dimethyl sulfoxide+ Indomethacin, C: Control

This study found that DMSO alone was more effective in reducing TBARS levels than either indomethacin alone or the indomethacin–DMSO combination in the aqueous humor. These results are consistent with those observed in TBARS levels associated with the use of DMSO in certain pathological conditions [21]. It is well known that TBARS are terminal products of lipid peroxidation, and therefore, the TBARS content is commonly utilized to estimate the extent of oxidative stress [22]. DMSO possesses a chemical property that allows it to be readily miscible with water and to dissolve highly lipophilic substances [23]. As a free radical scavenger, DMSO is clinically employed as an antioxidant in treating various diseases [23, 24]. DMSO may have reduced TBARS levels due to its chemical structure and its ability to scavenge hydroxyl radicals properties.

Total antioxidant status is a biomarker that describes the dynamic balance of oxidative stress between pro–oxidants and antioxidants [20, 13, 25]. Its levels decrease due to high catabolism and consumption in burn cases [26]. Indomethacin, an anti–inflammatory drug, is used to treat alkali eye burns because of its anti–inflammatory properties [15]. Nonsteroidal anti–inflammatory drugs, commonly used topically to prevent inflammation in ocular therapy, are known to cause rare corneal complications [27]. DMSO is known for its anti–inflammatory effects and its role as a free radical scavenger, effectively reducing oxidative stress following chemical burns [28, 29]. The TAS values were considerably elevated in group D, exhibiting a significant difference ($P<0.05$) in comparison to the control group on d 2. In group DI, TAS values were also higher, with a statistically significant difference ($P<0.05$) than the other groups on d 14. However, TAS values were markedly lower on the 2nd and 14th days in the groups treated only with indomethacin than the others. This increase in TAS in group D can be attributed to the use of DMSO because of its antioxidant properties.

The antioxidant effect of DMSO in this study was consistent with that of Altan et al. [15], which demonstrated that the DMSO–administered group exhibited both clinical (no corneal haziness, no conjunctival inflammation, minimal corneal erosion, among others) and histopathological (minimal inflammatory cell density) significant anti–inflammatory effects. The low level of inflammatory cell density, a major source of ROS, in the DMSO–treated group, is thought to correspond with the reduced oxidative stress observed in the aqueous humor of this group in the present study.

CONCLUSION

This study examined the TBARS and TAS levels related to oxidative status in the humor aqueous. It was not possible to evaluate other parameters indicating oxidative and antioxidant status due to insufficient samples collected from the aqueous humor. Although this might be viewed as a limitation of the study, the results suggest that DMSO is a potent antioxidant in aqueous humor. Consequently, this study is considered a model for further research on ocular diseases such as chemical eye burns, which may impede corneal healing due to oxidative stress and ultimately lead to vision loss.

Ethical approval

This research was conducted following the approval of the animal experiment by the Selcuk University Local Ethics Committee (Decision number: 2010/15).

Conflict of interest

The authors declare no conflicts of interest regarding this article's research, authorship, and/or publication.

Author's contributions

All authors contributed equally to this project's execution.

BIBLIOGRAPHIC REFERENCES

[1] Boret C, Brehin C, Cortey C, Chanut M, Houzé–Cerfon C–H, Soler V, Claude I. Pediatric ocular trauma: Characteristics and outcomes among a French cohort (2007–2016). Arch. Pediatr. [Internet]. 2020; 27(3):128–134. doi: https://doi.org/gkrhmc

[2] Feng K. Epidemiology of Ocular Trauma. In: Yan H, editor. Anatomy and Examination in Ocular Trauma. Singapore: Springer; 2019. p.105-122. doi: https://doi.org/pvq9

[3] Kyriakaki EDO, Symvoulakis EK, Chlouverakis G, Detorakis ET. Causes, occupational risk and socio–economic determinants of eye injuries: a literature review. Med. Pharm. Rep. [Internet]. 2021; 94(2):131–144. doi: https://doi.org/pvrb

[4] Moreno–Arrones Quesada J, Merayo Lloves J, Varillas Delgado D. Ocular chemical burns in the workplace: Epidemiological characteristics. Burns [Internet]. 2020; 46(5):1212–1218. doi: https://doi.org/pvrc

[5] Bizrah M, Yusuf A, Ahmad S. An update on chemical eye burns. Eye [Internet]. 2019; 33:1362–1377. doi: https://doi.org/gncvdz

[6] Eyer F, Zilker T. Expositionen mit Ätzstoffen. Ther. Umsch. [Internet]. 2009; 66(5):379–386. doi: https://doi.org/b3r7tb

[7] Parihar A, Parihar MS, Milner S, Bhat S. Oxidative stress and anti–oxidative mobilization in burn injury. Burns [Internet]. 2008; 34(1):6–17. doi: https://doi.org/dd79dv

[8] Lichtenberg D, Pinchuk I. Oxidative stress, the term and the concept. Biochem. Biophys. Res. Commun. [Internet]. 2015; 461(3):441–444. doi: https://doi.org/f7dtn8

[9] Gakhramanov FS. Effect of natural antioxidants on antioxidant activity and lipid peroxidation in eye tissue of rabbits with chemical burns. Bull. Exp. Biol. Med. [Internet]. 2005; 140:289–291. doi: https://doi.org/dj37h2

[10] Cejka C, Cejkova J. Oxidative stress to the cornea, changes in corneal optical properties, and advances in treatment of corneal oxidative injuries. Oxid. Med. Cell. Longev. [Internet]. 2015; 2015:591530. Available in: doi: https://doi.org/gb5pzb

[11] Bashkaran K, Zunaina E, Bakiah S, Sulaiman SA, Sirajudeen K, Naik V. Anti–inflammatory and antioxidant effects of Tualang honey in alkali injury on the eyes of rabbits: Experimental animal study. BMC Complement. Altern. Med. [Internet]. 2011; 11:90. doi: https://doi.org/dqt8qk

[12] Shoham A, Hadziahmetovic M, Dunaief JL, Mydlarski MB, Schipper HM. Oxidative stress in diseases of the human cornea. Free Radic. Biol. Med. [Internet]. 2008; 45(8):1047–1055. doi: https://doi.org/d7qhtg

[13] López–Contreras AK, Martínez–Ruiz MG, Olvera–Montaño C, Robles–Rivera RR, Arévalo–Simental DE, Castellanos–González JA, Hernández–Chávez A, Huerta–Olvera SG, Cardona–Muñoz EG, Rodríguez–Carrizalez AD. Importance of the use of oxidative stress biomarkers and inflammatory profile in aqueous and vitreous humor in diabetic retinopathy. Antioxidants [Internet]. 2020; 9(9):891. doi: https://doi.org/gjs9z6

[14] Bajraktarova–Valjakova E, Korunoska–Stevkovska V, Georgieva S, Ivanovski K, Bajraktarova–Misevska C, Mijoska A, Grozdanov A. Hydrofluoric acid: burns and systemic toxicity, protective measures, immediate and hospital medical treatment. Open Access Maced. J. Med. Sci. [Internet]. 2018; 6(11):2257–2269. doi: https://doi.org/jtb8

[15] Altan S, Oğurtan Z. Dimethyl sulfoxide but not indomethacin is efficient for healing in hydrofluoric acid eye burns. Burns [Internet]. 2017; 43(1):232–244. doi: https://doi.org/pvrd

[16] Claassen K, Rodil Dos Anjos D, Broding HC. Current status of emergency treatment of chemical eye burns in workplaces. Int. J. Ophthalmol. [Internet]. 2021; 14(2):306–309. doi: https://doi.org/pvrf

[17] Sharma N, Kaur M, Agarwal T, Sangwan VS, Vajpayee RB. Treatment of acute ocular chemical burns. Surv. Ophthalmol. [Internet]. 2018; 63(2):214–235. doi: https://doi.org/gc5x6n

[18] Hoang C, Nguyen AK, Nguyen TQ, Fang W, Han B, Hoang BX, Tran HD. Application of dimethyl sulfoxide as a therapeutic agent and drug vehicle for eye diseases. J. Ocul. Pharmacol. Ther. [Internet]. 2021; 37(8):441-451. doi: https://doi.org/pvrg

[19] Chen Y, Mehta G, Vasiliou V. Antioxidant defenses in the ocular surface. Ocul. Surf. [Internet]. 2009; 7(4):176–185. doi: https://doi.org/gjzcqz

[20] Goel M, Picciani RG, Lee RK, Bhattacharya SK. Aqueous humor dynamics: A review. Open Ophthalmol. J. [Internet]. 2010; 4:52–59.doi: https://doi.org/cv8r6v

[21] Silveira PC, Victor EG, Schefer D, Silva LA, Streck EL, Paula MM, Pinho RA. Effects of therapeutic pulsed ultrasound and dimethylsulfoxide (DMSO) phonophoresis on parameters of oxidative stress in traumatized muscle. Ultrasound Med. Biol. [Internet]. 2010; 36(1):44–50. doi: https://doi.org/bc98s6

[22] Dasgupta A, Klein K. Antioxidants in food, vitamins and supplements, prevention and treatment of disease [Internet]. Amsterdam (Netherlands): Elsevier Inc; 2014. Chapter 2, Methods for measuring oxidative stress in the laboratory; p. 19–40. doi: https://doi.org/pvrh

[23] Pielesz A, Gawłowski A, Biniaś D, Bobiński R, Kawecki M, Klama–Baryła A, Kitala D, Łabuś W, Glik J, Paluch J. The role of dimethyl sulfoxide (DMSO) in *ex–vivo* examination of human skin burn injury treatment. Spectrochim. Acta A Mol. Biomol. Spectrosc. [Internet]. 2018; 196:344–352. doi: https://doi.org/gc996s

[24] Di Giorgio AM, Hou Y, Zhao X, Zhang B, Lyeth BG, Russell MJ. Dimethyl sulfoxide provides neuroprotection in a traumatic brain injury model. Restor. Neurol. Neurosci. 2008; 26(6):501–507. PMID: 19096138. Available in: https://goo.su/noXaEnP

[25] Güler MC, Tanyeli A, Erdoğan DG, Eraslan E, Çomaklı S, Polat E, Doğanay S. Urapidil alleviates ovarian torsion detorsion injury via regulating oxidative stress, apoptosis, autophagia, and inflammation. Iran J. Basic Med. Sci. [Internet]. 2021; 24(7):935–942. doi: https://doi.org/gpbpq8

[26] Gürünlüoğlu K, Demircan M, Taşçı A, Üremiş MM, Türköz Y, Bag HG, Akıncı A, Bayrakcı E. The effects of two different burn dressings on serum oxidative stress indicators in children with partial burn. J. Burn Care Res. [Internet]. 2019; 40(4):444-450. doi: https://doi.org/pvrj

[27] Schalnus R. Topical nonsteroidal anti–inflammatory therapy in ophthalmology. Ophthalmologica [Internet]. 2003; 217(2):89–98. doi: https://doi.org/fqjrsz

[28] Patil M, Shivaprakash BV. Pharmacology and clinical use of dimethyl sulfoxide (DMSO): A review. Int. J. Mol. Vet. Res. [Internet]. 2013; 3(6): 23-33. doi: https://doi.org/pvrk

[29] Sanmartín–Suárez C, Soto–Otero R, Sánchez–Sellero I, Méndez–Álvarez E. Antioxidant properties of dimethyl sulfoxide and its viability as a solvent in the evaluation of neuroprotective antioxidants. J. Pharmacol. Toxicol. Methods [Internet]. 2011; 63(2):209–215. doi: https://doi.org/dcsbwt