

# Drugs for Retinal Disease that are Non-steroidal Anti-Inflammatory

## Abstract

In ophthalmology, NSAIDs are frequently used to treat pain and photophobia following photorefractive surgery as well as to lessen meiosis, inflammation, and cystoid macular edema following cataract surgery. The US Food and Drug Administration has recently approved novel topical NSAIDs and modified previously approved NSAIDs. These modifications might improve drug absorption into the retina and provide additional therapeutic benefits. For instance, therapeutic effects on age-related macular degeneration and diabetic retinopathy may now be possible. We offer an updated evaluation of the clinical application and scientific justification of NSAIDs for retinal illness.

**Keywords:** anti-inflammatory drug • nonsteroidal • neurodegeneration • neuroprotection • retina • optic nerve crush • drug delivery

## Introduction

The analgesic, antipyretic, and anti-inflammatory effect of Nonsteroidal anti-inflammatory medicines (NSAIDs) make them one of the most often prescribed pharmacological classes. NSAIDs are powerful inhibitors of the COX enzyme, which prevents the production of prostaglandins that cause inflammation (PGs). Topical NSAIDs are used in ophthalmology to treat cystoid macular edoema, allergic conjunctivitis, postoperative inflammation, and discomfort, as well as to regulate pupillary dilatation during intraocular surgery (CME) [1]. Topical NSAIDs have a proven track record of treating the aforementioned health issues. Additionally, there is growing evidence that PGs contribute to the pathogenesis of age-related macular degeneration (AMD) and diabetic retinopathy, and in recent years, more research have focused on the therapeutic benefits of NSAIDs for these conditions. This research aims to concentrate on the potential use of NSAIDs to treat retinal disorders.

Aspirin and other chemically related substances have been created in topical ophthalmic formulations relatively recently. These substances have been used systemically for many decades for their analgesic, antipyretic, and anti-inflammatory activities. They have therefore shown promise in improving mydriasis, decreasing postoperative inflammation, and preventing and treating cystoid macular edoema (CME) related to cataract surgery [2]. They can also be used to reduce post-refractive discomfort and photophobia as well as the itching brought on by allergic conjunctivitis. 91, 92, 94, 268, 274 a growing body of research suggests that NSAIDs may be helpful in treating ocular malignancies, age-related macular degeneration, and diabetic retinopathy. 110, 133, 167, 203, 298, 310 More than ten years have passed since the initial in-depth analysis of topical NSAIDs. 94. A chapter in Duane's Clinical Ophthalmology followed in 1994 and was updated in 2006. 92 We give a current assessment of NSAID usage in ophthalmology and talk about potential applications in the future [3].

## Chenguang Wang\*

Department of Ophthalmology, the Second Hospital of Jilin University, China

\*Author for correspondence:

Chenguang60@gmail.com

Tel: 868230613429

**Received:** 28-Sep-2022, Manuscript No. ACTVR-22-76466; **Editor assigned:** 01-Oct-2022, PreQC No. ACTVR-22-76466(PQ); **Reviewed:** 15-Oct-2022, QC No. ACTVR-22-76466; **Revised:** 22-Oct-2022, Manuscript No. ACTVR-22-76466(R); **Published:** 28-Oct-2022; DOI: 10.37532/ACTVR.2022.12 (5).94-96

## Methods and Materials

A multicentre case-control study examining heredity and environment in melanoma is called the Genes, Environment, and Melanoma (GEM) project. The GEM study design has been thoroughly described in a separate publication. Only the University of Michigan respondents were included in this study because further information on medication use was not gathered from other institutions [4].

Participants had to have had their first primary invasive melanoma diagnosed in 2000 as well as a second or higher order invasive primary melanoma diagnosed between January 1, 2000, and August 31, 2003. Subjects having a first primary melanoma in situ diagnosed in 2000 were also enrolled at the University of Michigan site. Additionally, this website registered the melanoma subjects' wives to serve as controls. Current marital status with a melanoma subject and readiness to give informed consent to participate were the inclusion criteria for controls. If a spouse had a personal history of melanoma, they were not allowed to participate in the study as controls. Only cases of newly diagnosed primary invasive melanoma or melanoma in situ are included in the current analysis [5].

At the time of enrolment, participants gave their signed, informed consent. The University of Michigan's Institutional Review Board gave the protocol their blessing. The subjects underwent an interview to determine their drug use, comorbidities, skin phenotype, and personal and family history of cancer. They were asked to recollect drugs used daily for at least three months in order to evaluate prior and present analgesic use.

Statistical evaluations were carried out utilising SAS software (version 9.1). To evaluate the basic correlations between drug use and melanoma risk, contingency tables were used. When cells contained counts of fewer than 10, P-values were calculated using Fisher's exact test. To evaluate the relationship between drug use and melanoma risk, account for confounding factors, and determine any potential effect modification, unconditional logistic regression was performed. P values are always reported on both sides [6].

Age, gender, family history of melanoma, skin

colour, hair colour, eye colour, severe sunburn history, cardiovascular disease history, history of musculoskeletal pain condition, tendency of skin to burn, tendency of skin to tan, age at first sunburn, number of moles on the back, smoking history, history of other medical diagnoses (yes/no), and statin use were among the potential variables evaluated for the multivariate model. If the addition of a covariate to the univariate model of analgesic usage and melanoma risk resulted in a 10% or higher change in the odds ratio, that covariate was included in the first multivariate model. In order to improve the saturated multivariate model's ability to fit the data, these factors were gradually eliminated from it [7].

## Discussion

Our findings imply that if potential confounders are taken into account, neither aspirin nor no aspirin NSAIDs are linked with a substantial and long-lasting reduction in the risk of melanoma. A tiny but significant positive effect of these drugs cannot be ruled out in a larger trial. Due to the small number of regular users in our trial, we were unable to evaluate the effects of COX-2 specific inhibitors or acetaminophen in full.

Four other papers evaluated how NSAIDs affected melanoma. The first discusses a case-control study that included 609 matched controls and 110 women who had melanoma. Women taking NSAIDs had a relative risk of melanoma of 0.45 (95% CI 0.22-0.92). With the inclusion of men, a larger number of melanoma cases, and the evaluation of additional variables, our study expands on this finding. The second study looked at how NSAIDs affected those who had already experienced melanoma [8]. Melanoma metastasis, recurrence, and new primary melanoma were all less common in people on COX inhibitors. Our work shifts the focus of this inquiry from secondary/tertiary prevention to primary prevention. The third study connected the NCI Surveillance, Epidemiology and End Results registry to NSAID usage in the Vitamins and Lifestyle (VITAL) project. These researchers discovered no link between NSAID use and melanoma risk. The number of instances in this study is comparable to ours, but many of them had in situ melanomas as opposed to invasive ones. By using more invasive melanoma cases and

adding information on the number of moles, which was the most important covariate in our analysis, our study builds on this finding? The fourth study examined this issue using the PALGA pathology database and the Dutch PHARMO pharmacy database. They discovered that women who regularly used aspirin had a 46% lower chance of developing melanoma (OR 0.54; 95% CI 0.30-0.99) [9].

Our study has a number of drawbacks. Since many NSAIDs are sold over-the-counter and usage of these drugs is not always fully recorded in the medical record, we were unable to compare reports of medication use with prescription data in our study. Due to the lack of a straightforward, established way to measure this risk factor, sun exposure could not be evaluated as a potential confounder. As potential confounders, we did examine self-reported history of severe sunburns, age at first sunburn, and susceptibility to burn, but these factors had no appreciable impact on the odds ratios. The control group may have been biased by selection because not all spouses of melanoma patients took part. We are unable to determine if the participating spouses were typical of the possible control group as a whole because no data are available on any consenting spouses, additionally overmatching of spouse controls, which may resemble the cases more than those in the general population, may reduce the odds ratios. In conclusion, when relevant confounders are included, our pilot investigation did not find a significant correlation between analgesic use and melanoma risk. Given the contradictory evidence for this question, meta-analysis or examination in a larger cohort with the ability to account for potential confounders may be suitable [10].

## Conclusion

The long-term visual benefits of this procedure are uncertain because CME can resolve on its own, despite the fact that there is strong overall evidence that topical NSAIDs can be used to treat and prevent CME after cataract surgery. Although the pathogenic role of inflammation in AMD, DR, and DME is now well recognised, clinical evidence supporting the therapeutic benefits of NSAIDs in treating these conditions is scarce and largely comes from small, retrospective, or uncontrolled

research. Despite strong scientific support, there is not enough evidence to advise using NSAIDs to treat these disorders until stronger clinical evidence is available.

## Acknowledgments

None

## Conflict of interest

None

## References

- Warren KA, Bahrani H, Fox JE *et al.* NSAIDs in combination therapy for the treatment of chronic pseudophakic cystoid macular edema. *Retina.* 30:260-266 (2010).
- Schoenberger SD, Miller DM, Petersen MR *et al.* Nepafenac for epiretinal membrane surgery. *Ophthalmol.* 118:1482-1482 (2011).
- Friedman DS, O'Colmain BJ, Munoz B *et al.* Prevalence of age-related macular degeneration in the United States. *Arch. Ophthalmol.* 122:564-572 (2004).
- Maloney SC, Fernandes BF, Castiglione E *et al.* Expression of cyclooxygenase-2 in choroidal neovascular membranes from age-related macular degeneration patients. *Retina.* 29:176-180 (2009).
- Hu W, Criswell MH, Ottlecz A *et al.* Oral administration of lumiracoxib reduces choroidal neovascular membrane development in the rat laser-trauma model. *Retina.* 25:1054-1064 (2005).
- Chen E, Benz MS, Fish MH *et al.* Use of nepafenac (Nevanac) in combination with intravitreal anti-VEGF agents in the treatment of recalcitrant exudative macular degeneration requiring monthly injections. *Clin Ophthalmol.* 4:1249-1252 (2010).
- Gomi F, Sawa M, Tsujikawa M *et al.* Topical bromfenac as an adjunctive treatment with intravitreal ranibizumab for exudative age-related macular degeneration. *Retina.* 32:1804-1810 (2012).
- Zhou J, Wang S, Xia X *et al.* Role of intravitreal inflammatory cytokines and angiogenic factors in proliferative diabetic retinopathy. *Curr Eye Res.* 37:416-420 (2012).
- Harris R, Beebe-Donk J, Namboodiri KK *et al.* Inverse association of non-steroidal anti-inflammatory drugs and malignant melanoma among women. *Oncol Rep.* 8:655-657 (2001).
- Asgari MM, Maruti SS, White E *et al.* A large cohort study of Nonsteroidal anti-inflammatory drug use and melanoma incidence. *J Natl Cancer Inst.* 100:967-971 (2008).