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THE ROLE OF LIFESTYLE AND DIET IN THE COURSE OF CATARACT: A LITERATURE REVIEW

Andrzej Wydrych

Independent Public Complex of Health Care Facilities in Płońsk, Henryka Sienkiewicza 7, 09-100 Płońsk

https://orcid.org/0009-0004-8466-2892

andrzej.wydrych@wp.pl

Andrzej Domański

Independent Public Complex of Health Care Facilities in Płońsk, Henryka Sienkiewicza 7, 09-100 Płońsk

https://orcid.org/0009-0001-6041-8359

adomanski00@gmail.com

Małgorzata Dziekońska

Independent Public Complex of Health Care Facilities in Kozienice, Wladyslaw Sikorski 10 Street, 26-900 Kozienice, Poland: Kozienice, Poland, PL

https://orcid.org/0009-0009-9597-6017

ma.dziekonska@gmail.com

Martyna Wójcik

Scanmed Specialist Clinics – Rudolf Weigl Hospital in Blachownia, Sosnowa 16 Street, 42-290 Blachownia

https://orcid.org/0009-0005-6003-3474

martyna.wojcik39@gmail.com

Klaudia Kowalczyk
Independent Public Complex of Health Care Facilities in Płońsk, Henryka Sienkiewicza 7, 09-100 Płońsk
https://orcid.org/0009-0004-6442-0589
8klaudia.kowalczyk8@gmail.com

Zofia Bogiel
State Medical Institute of the Ministry of the Interior and Administration in Warsaw, Wołoska 137 Street, 02-507 Warszawa
https://orcid.org/0009-0002-5425-4906
zofiabogiel112@gmail.com

Antonina Machała
Independent Public Complex of Health Care Facilities in Płońsk, Henryka Sienkiewicza 7, 09-100 Płońsk
https://orcid.org/0009-0004-3684-4579
tosia.machala@gmail.com

Aleksandra Tomczyk
University Clinical Hospital No 2 in Lodz, Żeromskiego 113, 90-549 Lodz
https://orcid.org/0009-0009-6030-3939
tomczyk.aleksandra@yahoo.com

Abstract

Introduction: Cataract, a progressive and largely irreversible opacification of the lens, remains a leading cause of visual impairment and blindness globally, with significant medical, social, and economic consequences. While aging is the main risk factor, lifestyle and environmental factors also influence cataract development.

Aim: This review examines current evidence on the role of lifestyle, diet, and environmental exposures in the onset and progression of cataract, with a focus on identifying modifiable determinants that may inform preventive strategies.

Materials and methods: For this review, we searched databases such as PubMed and Google Scholar using “cataract”, “diet”, “triggers”, “relationship”, “solar radiation”, “smoking”, and

"alcohol". We cited twenty-three articles in this publication. The review covers studies from 1966 to 2025.

Results: Epidemiological and clinical studies indicate that diet quality, smoking, alcohol consumption, ultraviolet (UV) exposure, and corticosteroid use affect cataract risk. Diets rich in fruits, vegetables, antioxidants, and healthy fats are protective, while smoking and alcohol intake consistently increase risk. Corticosteroid use is strongly associated with posterior subcapsular cataracts, and cumulative UV exposure, along with environmental stressors such as heat and low humidity, contributes to regional disparities in disease burden.

Conclusions: Cataract is a multifactorial condition influenced by aging, lifestyle, environmental, and pharmacological factors. Targeting modifiable risk factors through diet, lifestyle interventions, UV protection, and cautious corticosteroid use represents a crucial public health strategy. Future longitudinal, interventional, and epidemiological studies are warranted to clarify causal pathways and interactions, providing a robust evidence base for effective preventive programs aimed at delaying cataract onset.

Keywords: cataract, smoking, alcohol, solar radiation, diet, nutrition, lifestyle factors.

INTRODUCTION

Cataract, defined as a progressive and largely irreversible opacification of the lens, remains one of the leading global causes of visual impairment and blindness. Although cataract is strongly age-related, its impact extends beyond vision loss, carrying substantial medical, social and economic implications. Today, an estimated 95 million people worldwide are affected, and cataract continues to be the most common cause of blindness among adults aged ≥50 years. The burden is particularly heavy in low- and middle-income countries, where limited access to ophthalmic care makes cataract responsible for nearly half of all blindness [6][13]. As populations grow and life expectancy increases, the global prevalence of visually significant cataract is expected to rise further. Alarmingly, studies indicate that up to two-thirds of adults with cataract remain undiagnosed, with many already experiencing bilateral impairment and corresponding functional limitations. This highlights that the true impact of cataract on older populations is likely underestimated. Beyond its visual consequences, cataract is increasingly recognized as a marker of broader age-related vulnerability. Research shows

associations between cataract and frailty, increased fall and fracture risk, depressive symptoms. [6][21]. Given that many determinants of cataract—such as smoking, poor diet, ultraviolet exposure, physical inactivity, metabolic disorders, and overall lifestyle patterns—are modifiable, understanding how lifestyle influences cataract development is of crucial public-health importance. Identifying these determinants and their biological pathways may help establish preventive strategies, reduce disease burden, and improve quality of life among aging populations. This review therefore examines the current evidence on cataract within the broader context of health and aging and sets the stage for exploring **the role of lifestyle in cataract development, progression, and prevention. [6]**

METHODOLOGY

This article reviews the literature on the role of lifestyle and diet in cataract and emphasizes the influence of various external factors on the course of cataract. For this review, we searched databases such as PubMed and Google Scholar using “cataract”, “diet”, “triggers”, “relationship”, “solar radiation”, “nutrition”, “smoking”, and “alcohol”. We cited twenty-three articles in this publication. The review covers studies from 1995 to 2024. We focused on studies including controlled trials, meta-analyses, and systematic reviews. Only English-language publications with full-text availability were included. This review incorporates the most recent and relevant evidence to explore how lifestyle behaviors influence cataract risk, progression, and potential interventions, providing a comprehensive overview for public health and preventive strategies.

RESULTS

1. PATHOPHYSIOLOGY OF AGE-RELATED CATARACT

The lens, a biconvex crystalline structure located behind the iris, constitutes a critical component of the eye’s light-refracting mechanism. Its transparency is precisely regulated by physical and chemical processes, the disruption of which leads to lens damage and opacification. The term “cataract” encompasses any opacity or loss of lens transparency, with such opacities representing the earliest visible changes in disease development. The most common form of cataract is age-related cataract, and in its advanced stages, the whole lens may become opaque [21]. A cataract is a pathologic loss of the transparency of the crystalline lens due to changes in clarity and refractive properties. Along with the cornea, the lens focuses light onto the retina, permitting a sharp image to be formed. For an aberration-free image, the lens must be transparent and must have a high refractive index. To satisfy these optical demands,

the lens has developed a very special structure and composition: it is avascular, and the central fibers (nucleus) lose organelles like nuclei, mitochondria, endoplasmic reticulum, and ribosomes. Tight packing of the fibers minimizes intercellular space, reducing light-scattering structures and supporting lens optical clarity. Maintenance of lens transparency depends on densely packed crystallin proteins (α-, β-, γ-crystallins) within organelle-free lens fibers. Mature fibers lack nuclei and protein-synthesizing machinery, resulting in minimal protein turnover over many decades. While this architecture is essential for optical clarity, it predisposes the lens to the accumulation of molecular damage with aging [22]. A central mechanism in the pathogenesis of age-related cataract is oxidative stress. In young individuals, lens antioxidant systems - including GSH, ascorbic acid (vitamin C), and NRF2-regulated enzymes - neutralize ROS and protect proteins and lipids. With age, the activity of these systems declines and allows ROS formed by metabolism, ultraviolet radiation, and environmental factors to accumulate [2]. The resulting pro-oxidative environment damages lens proteins, particularly crystallins, and disrupts membrane lipids, impairing lens transparency. Oxidative modifications of crystallins include disulfide bond formation, tryptophan oxidation, and protein truncation. These aggregated crystallins form insoluble, high-molecular-weight complexes that scatter light and produce the lens opacities that characterize cataract. Because mature lens fibers are unable to replace damaged proteins, these aggregates progressively accumulate over many decades [12].

2. RISK FACTORS IN DIET

In a cohort of British participants, the diet group was strongly associated with cataract risk. A continuous decrease in risk was observed from high meat eaters, through the low meat and fish eaters, to vegetarians and vegans. Energy, protein, cholesterol, retinol, and vitamin B-12 intakes were positively associated with the risk of cataract, whereas carotene, vitamins C, D, and E, PUFAs, carbohydrates, and fiber showed no significant effect. An analysis limited to meat eaters did not show these associations, suggesting that the observed relationships may reflect differences between dietary groups. These results indicate that vegetarians and vegans have a significantly lower risk of cataracts compared with high meat eaters, particularly among older participants. The risk showed a continuous decline proportional to the reduction in consumption of meat and animal products [1]. In the UK Biobank study, higher consumptions of fruits and vegetables were associated with a lower risk of incident cataract. Higher legume, tomato, apple, and pear consumptions were related to reduced risks of cataract, whereas greater intakes of cruciferous vegetables, green leafy vegetables, berries, citrus fruit, and melon were

not associated with incident cataract. This study also confirmed the positive findings of higher intakes of legumes and tomatoes being protective in previous animal and human studies. Intake of apples and pears showed possible beneficial effects, probably because of their vitamin and antioxidant content. No association was found for intake of cruciferous or green/yellow vegetables, whereas intake of berries showed a nonsignificant trend toward protection. These findings support current dietary recommendations of at least two servings of fruit and three servings of vegetables per day for adults. This evidence is about the possible benefits of selected fruit and vegetable types in the prevention of cataracts. Strengths of the study are its large cohort, extended follow-up, comprehensive dietary data, and energy-adjusted analyses. The limitations are the possible underreporting of cataract cases, incomplete assessment of diet, and generalizability to the population being analyzed [7]. The Women's Health Study (WHS) found an association between frequent consumption of fruits and vegetables and cataract development in a 10-year perspective. Women who consumed ≥3.4 servings/day of fruits and vegetables had a 10-15% decreased risk of cataracts compared to the lowest intake quintile. Also, vitamin C intake at levels >107 mg/day was inversely associated with the prevalence of cataracts, with a 54% reduction in risk at intakes of 143-408 mg/day [8]. In the CAREDES study, dietary quality was associated with cataract prevalence: diets with low saturated fat and sodium with high fruit, vegetable, and overall variety were protective, but diets with high levels of sodium, trans fat, and processed meats conferred risk. We can include the following as protective factors: diets rich in antioxidants, such as vitamin C, beta- and alpha-carotene, and omega-3 fatty acids EPA and DHA. High intake of sodium, saturated and trans fats, and too many simple carbohydrates can lead to increased risk of cataracts. The reduced risk of cataracts can be achieved by at least five servings of fruits and vegetables per day (≥200 g/day); frequent consumption of fish, legumes, eggs, and low-fat dairy products throughout the week; limited intake of red/processed meat; and supplementation when dietary intake is insufficient, such as omega-3 [19][23]. Researchers link reduced oxidative stress to diets rich in antioxidants. Fruits, vegetables, and vitamins can protect the lens from oxidative damage. Analyses of the Healthy Eating Index components further support this. The authors observed a positive effect of the higher intake of fruits and whole grains, combined with lower consumption of refined grains, which is associated with lower cataract risk. Diet quality assessed by the Healthy Eating Index correlates positively with antioxidant levels. Taken together, these findings suggest that reduced oxidative stress resulting from a healthy dietary pattern is a central mechanism by which diet protects lens transparency and reduces cataract risk [23].

3. DRUGS

3.1 STEROIDS

Steroid-induced cataract is a major but often underrecognized contributor to the global burden of lens opacities. Analysis of Australian prescription data (2014–2019) shows that steroid use is both extensive and steadily rising, with nearly 9–10 million annual prescriptions and a 42.9% increase in associated costs over five years. Epidemiological data indicate that approximately 1.5 million Australians used prescription steroids in 2018/2019, with nearly half being ≥45 years old, placing a large population at elevated risk for cataract. Both inhaled and oral corticosteroids significantly increase the likelihood of cataract formation: in Australia, the odds ratio (OR) for PSC is 2.5 for inhaled and 4.1 for oral corticosteroids; nuclear cataracts are similarly elevated (OR 2.0 and 3.5, respectively). The characteristic phenotype of glucocorticoid-induced cataract includes centrally located PSC with vacuoles, likely linked to aberrant posterior migration of lens epithelial cells (LECs). While the clinical association is strong, molecular mechanisms remain incompletely understood. In vitro models show that steroids activate glucocorticoid receptors in human LECs and alter gene expression profiles, including matrix metalloproteinases (MMP-2, MMP-9), MAPK and PI3K/AKT signaling molecules, and cell adhesion markers. However, most studies rely on immortalized LEC lines and short exposures (<24 h), limiting their physiological relevance. Crucially, no existing human LEC studies assess the effect of glucocorticoids on the fundamental lens functions of transparency and focusing. Overall, current evidence highlights glucocorticoids as a well-established cause of PSC and nuclear cataracts. Rising prescription rates, combined with limited mechanistic understanding, underscore the need for improved risk stratification, safer therapeutic alternatives, and deeper exploration of steroid-induced molecular pathways in the lens [4]. Posterior subcapsular cataract (PSC) is the primary and most characteristic cataract subtype associated with corticosteroid use. Early studies in patients with rheumatoid arthritis demonstrated a strong link between oral corticosteroid therapy and PSC formation, and this association has since been consistently confirmed across different patient groups, including individuals with asthma, pemphigus, nephrotic syndrome, systemic lupus erythematosus, and patients receiving high-dose steroids for immunosuppression following renal transplantation. PSC cataract typically begins as fine, granular, vacuolated opacities at the posterior pole of the lens. Numerous studies have shown that PSC formation is particularly associated with high daily doses of corticosteroids (>15 mg prednisone or equivalent) and long-term use (>1 year). However, even short-term treatment as brief as four months has been identified as a significant

risk factor, as demonstrated in a clinic-based case–control study from Oxfordshire. Epidemiological studies of age-related cataract have repeatedly confirmed that corticosteroids selectively increase the risk of PSC cataracts rather than other cataract subtypes. Due to the widespread medical use of corticosteroids, ophthalmologists should carefully review a patient's medication history and be attentive to the characteristic features of steroid-induced PSC [8] [21].

3.2 ALLOPURINOL

Early case reports suggested that chronic allopurinol use might contribute to the development of cortical or posterior subcapsular cataracts. To investigate this potential association, a comparative study examined 51 long-term allopurinol users and 76 non-users, assessing lens opacities and visual acuity through masked ophthalmic evaluation to avoid observer bias. The study evaluated three endpoints: any cataract, posterior subcapsular cataract (PSC), and visually significant cataract (corrected visual acuity ≤20/30), and found no meaningful association between allopurinol exposure and these outcomes. Risk ratios were 1.3 for any cataract, 0.9 for PSC, and 1.3 for visually significant cataract, and adjustment for confounders did not materially alter the results. After an average exposure duration of 6.9 years, long-term allopurinol use showed no evidence of increasing cataract risk. Despite these findings, scattered earlier reports had suggested a possible link between prolonged allopurinol intake and premature PSC development, prompting researchers to explore potential biological mechanisms [5]. Experimental studies conducted on animal lenses and human donor lenses did not confirm that chronic high-dose allopurinol causes cataracts, nor did they provide consistent evidence that allopurinol acts as a photosensitizer in ultraviolet-induced lens damage. Some studies reported that cataractous lenses from patients taking allopurinol for over two years demonstrated a unique photobinding effect not observed in normal lenses, suggesting that cataract formation may require drug retention within the lens, making it more vulnerable to photochemical injury. Further epidemiologic investigations produced inconsistent findings, largely due to small sample sizes and incomplete exposure data. Some cohort studies found no association between allopurinol and cataract development, though the exposure definitions used were not sensitive enough to detect long-term use [21]. Similarly, studies evaluating geriatric cohorts or hospital pharmacy–identified users reported no excess cataract risk, though key exposure parameters were missing and statistical power was limited. A series of patients taking allopurinol for 18 months or longer exhibited various lens changes; however, the lack of a control group limited causal interpretation. Other studies reported associations between

general gout medications and mixed cataracts, but did not isolate allopurinol as the contributing factor. Overall, the collective evidence indicates no credible epidemiologic or experimental support for allopurinol as a cataract-causing medication. Earlier concerns likely reflected confounding by comorbidities common in gout patients, such as age or metabolic disease, rather than a direct effect of the drug itself [5][21].

4. SMOKING

Some report that smoking is an essential factor in in cataract development and is associated with an increased risk of nuclear cataract subtypes, whereas former smokers have a lower risk compared to current smokers. A meta-analysis by Ye et al., which combined data from 13 cohort and 8 case-control studies, confirmed this association and demonstrated a dose-dependent effect of tobacco exposure on cataract formation. The primary pathophysiological mechanisms of smoking-induced cataract involve oxidative stress and direct lens toxicity. Tobacco smoke generates free radicals that promote protein oxidation and lipid peroxidation within the lens, ultimately leading to protein aggregation and lens opacification. In addition, heavy metals present in tobacco, such as cadmium, lead, and copper, accumulate in the lens and exert cytotoxic effects, further contributing to cataract genesis. Despite consistent observational evidence, Mendelian randomization studies, including the analysis by Jiang et al., have not shown a statistically significant causal link between genetically predicted smoking and cataract risk. This discrepancy likely reflects limitations of genetic instruments in capturing the complex behaviors of smoking, including intensity, duration, and exposure to secondhand smoke, rather than indicating a true absence of effect. The evidence supports cigarette smoking as a modifiable risk factor for cataract, and cessation of tobacco use is likely to reduce the long-term risk of lens opacification [10]. A cohort study of 63–64-year-olds in Western Sweden investigated the association between smoking and cataract, confirming a dose-dependent increased risk of cataract surgery associated with cigarette smoking. Risk rose by approximately 5% for every 5 years of smoking and 4% per 5 pack-years, even after adjusting for multiple confounders, with no significant difference observed between current and former smokers, although previous research suggests that long-term cessation (>10 years) may reduce risk. The study included 9,316 participants (51% women, 49% men). The overall prevalence of self-reported cataract was 11.3% (13.5% in women, 9.0% in men), and 6.3% of participants had undergone cataract surgery (7.1% women, 5.4% men). Ever-smoking was reported by 59.3% of participants, including 12.1% current smokers and 47.2% former smokers. Smoking was associated with a significantly increased risk of cataract surgery (HR 1.34, 95% CI 1.04–

1.74), while former smokers had a moderately elevated risk after adjustment for additional factors (HR 1.27, 95% CI 1.03–1.56). Risk increased with cumulative exposure, with each additional 5 years of smoking or 5 pack-years increasing the risk of surgery by approximately 5%, with a stronger effect in women (HR 1.07 per 5 years, 95% CI 1.03–1.10) [17]. An increased risk of lens opacities associated with cigarette smoking has been further demonstrated in eight additional studies. For example, West et al. showed that, in a group of white male fishermen, the cumulative dose of cigarettes smoked was associated with nuclear, but not cortical, opacities. Notably, the risk of nuclear opacities decreased in individuals who had stopped smoking for 10 years. A similar dose-response relationship was observed in cross-sectional data from 1,029 volunteers from diverse social and occupational backgrounds in and around London, showing a significant association between past heavy smoking and nuclear opacities, with an even stronger association among current heavy smokers. Comparable findings were reported across case-control, cross-sectional, and prospective studies. In a prospective study conducted by Christen et al. in a cohort of male physicians, an increased risk of posterior subcapsular cataract (PSC) was also observed, with a dose-response relationship between increasing pack-years and PSC prevalence in men, and a similar, though not statistically significant, trend in women in the Beaver Dam study. However, two clinic-based case-control studies did not find an association between smoking and senile cataract subtypes. In the Indian study, "smoking" included both cigarettes and other tobacco products commonly used in the subcontinent, whose comparability to those studied in the United States and London is unclear. Likewise, the Italian case-control study found no association between smoking and nuclear opacities, although a similar study in Boston did detect a significant link. Cigarette smoke contains substances that impair antioxidant defenses and compounds capable of directly modifying lens proteins. Consistent findings across multiple studies reinforce the role of cigarette smoking as a risk factor for nuclear cataract and potentially for PSC opacities. Considering that approximately 26% of the U.S. population currently smokes, it is estimated that up to 20% of cataract cases in the country may be attributable to smoking [21].

5. ALCOHOL CONSUMPTION

Alcohol consumption has been investigated as a potential risk factor for cataract, but findings remain inconsistent. Genetic studies suggest that alcohol may not directly cause cataract formation. A Mendelian randomization analysis by Jiang et al.[1], using data from the Genetic Epidemiology Research on Adult Health and Aging and the UK Biobank, found no significant association between genetically predicted alcohol intake and cataract risk. Im et al.[2]

reported that heavy drinking (≥280 g/week) significantly increased cataract risk, while low-to-moderate consumption (<140 g/week) appeared borderline protective. Chua et al.[3] observed a reduced likelihood of cataract surgery among low-to-moderate alcohol consumers, particularly wine drinkers, and Kanthan et al.[4] described a U-shaped relationship between alcohol intake and cataract surgery incidence. A meta-analysis by Gong et al.[5] further suggested that moderate alcohol intake may have a modest protective effect, whereas heavy consumption increases risk [10]. Earlier epidemiological research supports these observations. Clayton et al. (1980) reported a "J-shaped" relationship: both heavy drinkers and abstainers had higher cataract risk than occasional drinkers. Studies in Oxfordshire and elsewhere found that heavy beer consumption doubled the risk of cataract, and consuming more than one drink per day increased the risk of surgical posterior subcapsular cataract (PSC) up to four-fold. Similar findings were reported in Wisconsin adults and among white male fishermen, with heavy drinking linked to severe nuclear, cortical, and PSC lens opacities [21]. Alcohol may contribute to cataract formation through several mechanisms. Chronic heavy drinking induces the liver enzyme cytochrome CYP2E1, which produces free radicals that can cause lens protein aggregation. Acute alcohol exposure can disrupt calcium balance in lens cells by impairing calcium pumps and increasing membrane permeability. Additionally, alcohol is metabolized into acetaldehyde, a reactive compound that can modify lens proteins and accelerate cataract development. Genetic differences in alcohol metabolism, such as variants in aldehyde dehydrogenase, may also influence individual susceptibility [10][11]. The link between alcohol and cataract risk is still unclear. Heavy drinking consistently raises the risk, likely due to oxidative stress, free radicals, and direct lens damage. Light-to-moderate drinking may have a small protective effect, but this is not consistent across studies and could be influenced by other factors. The protective effect of moderate drinking remains uncertain. More well-designed studies are needed to confirm whether moderate alcohol intake truly reduces cataract risk [10][21].

6. UV RADIATION EXPOSURE

Lens opacification has been linked to ocular exposure to ultraviolet (UV) radiation, particularly ultraviolet B (UV-B). The lens absorbs both UV-B and UV-A radiation. Changes in lens clarity have been demonstrated in animal experiments following short-term, high-intensity exposure, as well as chronic exposure to UV-B. UVR exposure to the eye is influenced by environmental and anatomical factors, with the temporal limbus being particularly susceptible to incident light. The cornea effectively blocks radiation below 300

nm, whereas the crystalline lens absorbs most UVR up to 380 nm, making it vulnerable to chronic photochemical damage. New evidence shows that UVR damage can accumulate synergistically with repeated exposures, although partial recovery occurs with longer intervals. Lens sensitivity to UVR varies with age, being highest in young individuals, and vitamin E supplementation has shown protective outcomes in animal research. UVB was consistently associated with more profound biochemical alterations than UVA. Pre-treatment with topical steroids amplified metabolic injury, indicating that systemic or topical therapies can modulate susceptibility to UVR damage. Epidemiological data from the Reykjavik Eye Study support the association of high lifetime sunlight exposure with increased risk of cortical cataract, especially among individuals with prolonged outdoor activity during early and middle adulthood [15]. A Chinese study found a significant association between frequent exposure to ultraviolet (UV) radiation and the development of cataracts. The analysis included 185 countries, covering 94.87% of the global population, as well as 206 subnational regions across seven countries. These findings support the suggestion that cataractogenesis is influenced by cumulative UV exposure through mechanisms such as photooxidation and apoptosis affecting lens epithelial cells. The analyses showed that UV exposure modifies the relationship between the Human Development Index (HDI) and the cataract burden, with high levels of UV radiation amplifying the effect of lower socioeconomic status on years lived with disability (YLD) rates. The study also highlights the additional financial and public health burden associated with high levels of UV exposure, particularly in low- and middle-income regions where access to and quality of cataract surgery remain limited. UV exposure varies widely across the world, with countries of lower HDI showing significantly higher daily UV doses. The study confirmed that cataract-related blindness is concentrated in low-HDI regions experiencing higher ambient UV levels. Ultraviolet radiation contributes to cataract formation through dose-dependent photochemical damage, apoptosis, and oxidative stress affecting lens proteins. In countries with a low HDI, the cost and quality of cataract surgery increase inequalities in access to treatment, leading to a higher overall rate of cataract-related blindness in these countries. To mitigate this risk, it is essential to strengthen UV protection measures, including wearing sunglasses, using head coverings, and reducing time spent outdoors during periods of highest UV intensity, especially in developing regions exposed to high levels of ultraviolet radiation. [6] In summary, current epidemiologic evidence supports a role for sunlight exposure in the development of cortical and PSC cataracts. Concern has been raised about potential increases in cataract risk associated with higher UV-B exposure due to ozone depletion. However, data on the range of ocular exposure in human populations are insufficient to quantify any increase

in age-related cataract risk. Further research in this area is clearly warranted [6][15].

7. OBESITY

Cataracts, primarily age-related lens opacities, have been associated with obesity in several population studies. Early work by Glynn et al. (1995) demonstrated a strong correlation between body mass index (BMI) and incident cataracts, particularly posterior subcapsular (PSC) and nuclear cataracts, with a 2-unit BMI increase predicting a 12% higher risk. Subsequent analyses from the Physicians' Health Study (Schaumberg et al., 2000) confirmed that both BMI and waist-to-hip ratio (WHR) are independent risk factors for cataract development. Later studies identified cortical cataracts as the subtype most strongly linked to elevated BMI (Glynn et al., 2009). Abdominal obesity appears to be an even more specific predictor of cataract risk than BMI, with waist circumference, WHR, and waist-to-height ratio (WHtR) reflecting intra-abdominal fat and visceral obesity. Metabolic syndrome and its components predict a higher 5-year incidence of cortical and PSC cataracts in older adults. Mechanistically, obesity is associated with subclinical systemic inflammation and oxidative stress, both of which contribute to lens damage and cataractogenesis. Elevated C-reactive protein (CRP), a marker of systemic inflammation, has been linked to increased cataract risk. Oxidative stress, confirmed in animal models, causes damage to lens proteins, and is exacerbated in obesity [3]. Additionally, obesity is frequently associated with systemic conditions that further increase cataract risk, including hypertension, hyperlipidemia, diabetes, and insulin resistance. Conversely, higher levels of physical activity, such as walking or running, have been associated with reduced cataract incidence, likely due to lower oxidative stress levels. Obesity, particularly central obesity, is a risk factor for age-related cataracts, including posterior subcapsular and cortical subtypes, while physical activity appears protective. Regular physical activity indirectly reduces cataract risk by improving metabolic health, lowering inflammation and supporting vascular function. Physically active individuals show reduced incidence of cataract and delayed progression. These findings highlight the importance of weight management and lifestyle interventions in reducing the risk of cataract development in adults [3][18].

8. DIABETES

Diabetes mellitus (DM) is a major global health problem and a well-established risk factor for accelerated cataract formation. Biochemical studies of lenses from diabetic and galactosemic patients, commonly referred to as sugar cataracts, demonstrate abnormalities in

electrolyte levels, glutathione, and glucose or galactose concentrations. The enzyme aldose reductase converts glucose or galactose into sugar alcohols, leading to hyperosmotic stress within the lens. These metabolic alterations promote lens fiber swelling, vacuole formation, and subsequent opacification, explaining the earlier onset and faster progression of cataracts in diabetic patients [21]. Hyperglycemia-driven mechanisms, including activation of the polyol pathway, sorbitol accumulation, oxidative stress, apoptosis of lens epithelial cells, mitochondrial dysfunction, and impaired autophagy, further accelerate lens degeneration. In diabetic smokers, diminished cytochrome c oxidase activity and reduced antioxidant capacity in the aqueous humor exacerbate oxidative damage, while altered inflammatory mediators, including VEGF, IL-10, and FasL, contribute to lens pathology [24]. Epidemiologic and clinic-based studies consistently report a positive association between diabetes and cataract. The Framingham Eye Study observed that higher blood glucose levels correlate with increased cataract risk, while population-based analyses from both the Framingham Eye Study and NHANES indicate a three- to four-fold increased risk of senile cataract among diabetic individuals under 65 years, and this risk does not persist in those older than 65. Population-specific studies have further highlighted factors influencing cataract development. In younger-onset diabetics, disease duration is the strongest predictor, whereas in older-onset diabetics, age at examination is more relevant. A study in Israel found that glucose intolerance was associated with a six-fold increased risk of senile cataract in women aged 40 to 70 years, and higher glycated hemoglobin levels independently predicted cataract risk, suggesting that poor glycemic control is a major driver of lens opacification. These associations were not observed in men [16][21]. Clinical observations confirm these findings. In a study of 319 diabetic patients, cataracts were present in 32.9 percent of participants, consistent with previous epidemiologic data. Cataract prevalence increased significantly with age, reflecting the combined impact of aging and chronic hyperglycemia. Longer diabetes duration and poor glycemic control, with a median disease duration of 14 years and a median HbA1c of 7.7 percent, were among the strongest predictors of cataract development. Among cataract subtypes, cortical cataracts were most frequent, followed by nuclear sclerotic and posterior subcapsular forms, with poor glycemic control most strongly associated with cortical and nuclear cataracts. Smoking further increased the risk of nuclear and cortical opacities. Overall, diabetic cataracts arise earlier and progress faster than in non-diabetic populations due to the combined effects of metabolic, oxidative, and inflammatory stress on the lens. The consistency of these findings across biochemical, epidemiologic, and clinical studies underscores the importance of regular ophthalmologic screening and rigorous glycemic control in patients with

diabetes [9].

9. HUMIDITY AND TEMPERATURE

Early studies indicate that environmental factors play a role in the development of age-related cataracts. Analysis of population-level data has shown that extreme ambient conditions, particularly higher temperatures and lower relative humidity, are associated with an increased risk of cataract formation in older adults. Specifically, a nonlinear J-shaped relationship between temperature and cataract prevalence suggests that both prolonged heat exposure and low humidity may contribute to lens opacification. Low humidity may elevate cataract risk by causing ocular surface dehydration, hyperosmolarity, and inflammatory responses, which can destabilize the tear film and promote lens damage. Elevated temperatures are thought to accelerate metabolic activity in the lens epithelium and induce protein cross-linking, processes implicated in age-related cataract development. Animal studies further support these mechanisms, showing that lenses exposed to higher temperatures develop cortical and nuclear opacities more rapidly, although direct extrapolation to humans should be made cautiously [13]. Occupational and recreational sunlight exposure has also been consistently identified as a major environmental risk factor. Individuals exposed to sunlight during work had approximately double the risk of developing cataracts, particularly nuclear and posterior subcapsular types. Similarly, recreational exposure to sunlight, such as spending 30 minutes or more per day at the beach, was associated with a more than threefold increase in risk for all cataract types, with the association being statistically significant for nuclear cataract. These results were independent of diabetes or other major comorbidities. Protective behaviors, including the use of hats and sunglasses, were associated with a significantly lower risk of cataract, underscoring the importance of UV protection in cataract prevention [20]. Epidemiological evidence also suggests geographic variability in cataract prevalence, with higher rates observed in warmer regions and plains compared to cooler or mountainous areas. Cumulative thermal exposure of the lens correlates with nuclear cataract prevalence, supporting the role of long-term environmental heat stress in lens aging. While many studies rely on city-level ambient data, which may limit assessment of individual exposure, and potential confounding factors such as air pollution are not always addressed, the consistency of findings across populations strengthens the evidence that both climatic conditions and sunlight exposure are critical determinants of cataract risk. These observations highlight the need to consider environmental and climatic factors in cataract prevention and eye health management, particularly in the context of global climate change [13].

CONCLUSIONS

Cataract is a multifactorial condition resulting from the cumulative effects of aging, lifestyle behaviors, environmental exposures, and medication use. Evidence reviewed in this study indicates that modifiable factors, including diet quality, smoking, alcohol consumption, ultraviolet exposure, and corticosteroid use, play a significant role in cataract development and progression, largely through oxidative stress–mediated damage to lens proteins. Diets rich in fruits, vegetables, antioxidants, and healthy fats appear protective, whereas smoking and heavy alcohol consumption consistently increase cataract risk. Corticosteroids are a well-established cause of posterior subcapsular cataract, highlighting the need for careful prescribing and ophthalmic monitoring, while environmental stressors such as ultraviolet radiation contribute substantially to global disparities in cataract burden. Given the rising prevalence of cataract in aging populations and the substantial healthcare and economic costs of surgical treatment, preventive strategies targeting modifiable lifestyle and environmental factors are of major public health importance. Future research should prioritize longitudinal, interventional, and epidemiological studies to clarify causal pathways and the interactive roles of these and other risk factors in cataractogenesis. Such evidence will form a robust basis for the design of effective public health initiatives aimed at preventing or delaying cataract development.

DISCLOSURE

Authors contribution:

Conceptualization: Andrzej Wydrych, Andrzej Domański

Methodology: Andrzej Wydrych, Małgorzata Dziekońska

Software: Zofia Bogiel, Martyna Wójcik

Check: Andrzej Domański, Zofia Bogiel

Formal analysis: Antonina Machała

Investigation: Klaudia Kowalczyk, Aleksandra Tomczyk

Resources: Martyna Wójcik, Klaudia Kowalczyk

Data curation: Antonina Machała, Andrzej Domański

Writing - rough preparation: Zofia Bogiel, Małgorzata Dziekońska

Writing - review and editing: Aleksandra Tomczyk

Visualization: Andrzej Wydrych, Klaudia Kowalczyk

Supervision: Aleksandra Tomczyk, Martyna Wójcik

Project administration: Antonina Machała, Małgorzata Dziekońska

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