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Structural and Biochemical Alterations in Congenital Cataract: An Integrative Review of Mechanisms and Clinical Implications

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ABSTRACT

Congenital cataract is a major cause of preventable childhood blindness, particularly in low-resource settings. Advances in genetics and molecular biology have deepened our understanding of the structural and biochemical mechanisms underlying lens opacification, yet early detection and access to personalized treatments remain limited. This study aimed to synthesize the main genetic and biochemical alterations associated with congenital cataracts and evaluate their clinical implications through an integrative review of recent literature. An integrative review was conducted following the PICO strategy, focusing on patients with congenital cataracts (P), analysis of molecular and biochemical changes (I), comparison among study types (C), and clinical outcomes (O). The search included six major databases, covering studies published from 2017 to 2025. A total of 22 articles met the eligibility criteria and were analyzed through data triangulation and descriptive synthesis. The review identified key genetic mutations (e.g., *CRYBA1*, *CRYBB1*, *FYCO1*, *TSR1*) and oxidative stress pathways—such as ferroptosis and the KLF5/MDM2 and SIRT6/p-Nrf2/GPX4 axes—as major contributors to congenital cataractogenesis. Early surgical intervention, particularly before two years of age, was associated with significant improvements in visual prognosis. The integration of molecular findings with clinical strategies suggests a promising future for personalized, preventive, and therapeutic approaches. Congenital cataract management benefits from a multifaceted strategy that includes genetic screening, early surgery, and emerging antioxidant therapies. However, disparities in diagnostic access and neonatal screening remain barriers to equitable care. Broader implementation of these measures can enhance visual outcomes and reduce the burden of childhood blindness globally.

INTRODUCTION

Congenital cataract is one of the leading causes of preventable childhood blindness, particularly in low-resource countries where early diagnosis and access to specialized treatment remain limited (1). This condition is characterized by opacification of the lens present at birth or within the first years of life, significantly impairing visual development during a critical period of neuroplasticity. If not treated early, it can lead to severe amblyopia or even irreversible blindness, as the visual cortex depends on clear and continuous stimuli for the proper formation of neural connections (2).

In recent decades, major advances in molecular genetics have contributed to the understanding of the mechanisms involved in congenital cataractogenesis. Crystallin proteins—particularly

those belonging to the β and γ families—play an essential role in maintaining lens transparency. Mutations in genes such as CRYBB1, CRYGD, and CRYGS compromise the conformational stability of these proteins, favoring the formation of insoluble aggregates and lens opacification (2-4). The βB1-L116P mutation, for example, has been associated with the formation of amyloid fibrils—cytotoxic structures that induce cellular dysfunction and accelerate loss of transparency (4).

In addition to genetic alterations, oxidative stress plays a key role in the pathophysiology of congenital cataract. The accumulation of reactive oxygen species (ROS) damages lipids, proteins, and nucleic acids in the lens, especially when antioxidant mechanisms are insufficient. Recently, studies have identified ferroptosis—a form of iron-regulated cell death—as a relevant mechanism in lens cell degeneration (5,6). Antioxidant pathways such as KLF5/MDM2 and SIRT6/p-Nrf2/GPX4 have proven crucial for maintaining cellular homeostasis and protecting against oxidative damage, and are considered potential therapeutic targets (7).

Pharmacological interventions with antioxidants, such as melatonin, have shown promising results in reducing ferroptosis and preserving lens function, highlighting the importance of understanding the molecular mechanisms involved (6). Advances in genetic sequencing techniques—particularly next-generation sequencing (NGS)—have expanded the ability to identify mutations associated with congenital cataracts, such as those found in FYCO1, TSR1, and GJA8, enabling earlier diagnoses and personalized therapeutic strategies (8,9).

Despite these advances, early detection remains a challenge, especially in contexts lacking widespread neonatal screening programs. The red reflex test, a simple, effective, and non-invasive method, remains the main screening tool within the first 72 hours of life, yet its universal implementation is still limited (1). The absence or asymmetry of the reflex should be promptly investigated to allow timely surgical intervention and prevent complications such as amblyopia.

Thus, the present study aims to investigate the genetic and biochemical alterations associated with the development of congenital cataract, with an emphasis on mechanisms of protein aggregation, oxidative stress, and cell death pathways. By exploring these molecular pathways, the goal is to identify potential therapeutic targets that may support early and personalized interventions, contributing to improved clinical outcomes for affected children.

METHODS

This study was conducted as an integrative literature review aimed at investigating the main structural and biochemical alterations associated with congenital cataracts and their clinical implications. The integrative review methodology was chosen for its ability to synthesize evidence from diverse types of studies, thereby offering a comprehensive and critical understanding of the topic.

The guiding research question was developed using the PICO strategy: "*What are the main structural and biochemical changes associated with congenital cataracts, and what are their clinical implications?*" In this framework, P (Population) referred to patients with congenital cataracts; I (Intervention) corresponded to the analysis of structural and biochemical alterations; C (Comparison) involved the comparison among different studies and applied methodologies; and O (Outcome) focused on understanding clinical implications and the development of new therapeutic approaches (Table 1).

Table 1. PICO strategy applied to the research question.

Components	Description
P (Patients or Problem)	Patients with congenital cataracts
I (Intervention)	Analysis of structural and biochemical alterations
C (Comparison)	Comparison among different studies and methodologies
O (Outcomes)	Understanding clinical implications and development of new therapeutic approaches

Source: The authors, 2025.

The literature search was conducted across major scientific databases, including PubMed, *National Library of Medicine* (NIH), SciELO, MEDLINE, *Virtual Health Library* (VHL), and the *Cochrane Library*, covering publications from 2017 to 2025. To ensure both breadth and specificity, controlled descriptors such as "Congenital Cataract," "Crystallins," "Oxidative Stress," "Lens Opacities," and "Molecular Pathways" were used. These terms were combined using Boolean operators "AND" and "OR" to optimize the sensitivity and specificity of the search.

The selection of studies followed strict eligibility criteria. Studies were included if published between 2017 and 2025, written in English or Portuguese, and directly addressed structural and biochemical alterations related to congenital cataracts through molecular, biochemical, or genetic approaches. Excluded from the review were studies focusing exclusively on acquired or senile cataracts, as well as opinion articles, editorials, commentaries without empirical data, case reports, theses, dissertations, and papers without full-text availability. Duplicate studies identified due to overlapping keywords were also excluded.

In addition to original articles, the inclusion criteria encompassed scientific textbooks, literature reviews, academic monographs, regulatory/scientific reports, comprehensive reviews, and clinical guidelines relevant to the research question. Conversely, exclusion criteria also included studies conducted exclusively in experimental models, publications in unspecified languages, letters to the editor, opinion pieces, and repeated records.

A total of 140 studies were initially retrieved based on title and abstract screening. After full-text evaluation, 22 studies were included in this review for meeting all eligibility criteria. The selected articles were categorized and evaluated according to study type, methodology, thematic focus, and major outcomes.

To facilitate this process, Microsoft Excel® was used for data tabulation, enabling the organization and comparison of results across the included studies.

To enhance the rigor of the review, a triangulation approach was adopted, incorporating multiple data sources and theoretical perspectives. This allowed for cross-validation of findings and the identification of converging and diverging patterns. All phases of the study followed ethical research principles to ensure the confidentiality, reliability, and integrity of the data analyzed (Figure 1).

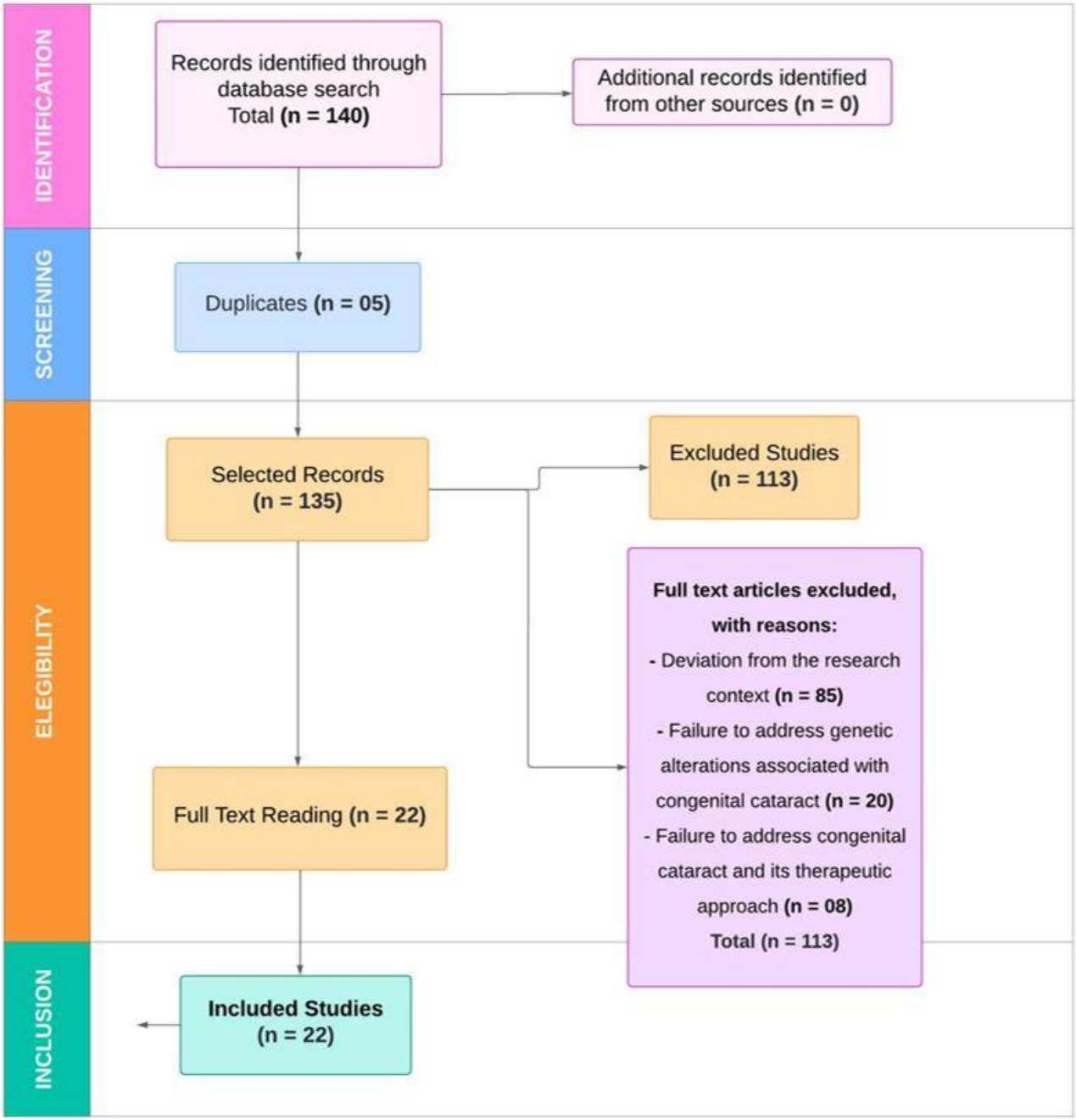

Figure 1. Flowchart of the article selection process.

RESULTS

This study reviews the genetic alterations in biochemical and metabolic pathways associated with congenital cataracts, aiming to identify molecular mechanisms that directly influence lens opacification and explore potential therapeutic interventions. Through the analysis of recent studies, we sought to elucidate the role of specific signaling pathways and genetic mutations—such as the antioxidant pathways KLF5/MDM2 and SIRT6/p-Nrf2/GPX4—in the progression of cataracts. These findings not only enhance the understanding of underlying pathological processes but also offer promising directions for preventive and personalized approaches.

Overall, the results of this integrative literature review indicate that early surgical intervention for bilateral congenital cataracts in children under the age of two is highly effective in preserving visual function. Early intervention is crucial to prevent permanent damage to visual development, especially during the critical period of neural connectivity formation (Table 2).

Table 2. Main studies related to congenital cataract and factors associated with ocular health.

Author (Year)	Study Objective	Study Type	Methodology	Key Findings	Conclusions
Mi et al. (6)	Investigate the effect of melatonin on ferroptosis and oxidative stress-induced cataract	Experimental (Animal Study)	Animal models (n=30) with induced cataracts treated with melatonin	Melatonin reduces ferroptosis and improves lens opacity	Melatonin may be a preventive approach to oxidative stress-induced cataracts
Li et al. (5)	Analyze the role of the KLF5/MDM2 axis in diabetic cataract	Experimental (Cell Study)	Functional analysis of the KLF5/MDM2 axis in lens cells (n=50)	The KLF5/MDM2 axis modulates oxidative stress and epithelial-mesenchymal transition	Interventions targeting this axis may be effective for diabetic cataract
Liu et al. (4)	Investigate the effect of the βB1-L116P mutation in congenital cataract	Experimental (Cell Study)	Structural and functional analysis of βB1-L116P protein (n=35)	Mutation promotes amyloid fibril formation and lens opacification	Anti-aggregation therapy may be a viable strategy
Taylan Şekeroğlu; Utine (8)	Explore the genetics of congenital cataracts using NGS	Literature Review	Review of publications on NGS in congenital cataracts	NGS identifies new cataract-related mutations	NGS expands personalized diagnosis and treatment options
Ullah et al. (7)	Characterize FYCO1 gene mutations in congenital cataract	Familial Genetic Study	Sequencing of 12 affected families	FYCO1 is a gene associated with congenital cataracts	Important genetic marker for early diagnosis
Li et al. (10)	Explore oxidative stress in cataract models	Experimental (Animal Study)	Animal models with induced oxidative stress (n=60)	Oxidative stress contributes to cataract formation	Antioxidant strategies show therapeutic potential
Zhang et al. (11)	Evaluate 14-3-3ζ protein in stress-induced cataract	Experimental (Cell Study)	Cell models (n=40) analyzing 14-3-3ζ expression	Protein influences stress response and cataract development	Modulation of 14-3-3ζ may have therapeutic application
Singh et al. (1)	Evaluate surgical interventions for	Systematic Review	Review of 20 clinical studies	Results depend on timing and type of intervention	Early surgery is crucial for better visual outcomes

	bilateral congenital cataracts				
Zhang et al. (2)	Analyze Q70P mutation in CRYBB1	Experimental (Cell Study)	Structural models of mutated CRYBB1 protein	Q70P promotes insoluble aggregation and protein instability	Contributes to lens opacification via protein aggregation
Song et al. (12)	Evaluate S175G/H181Q and P24S/S31G mutations in crystallins	Experimental (Structural Modeling)	Analysis of mutated crystallins in molecular models	Mutations cause structural changes and aggregation	Mutations reduce solubility and transparency
Xu et al. (13)	Study the effect of CRYBA1/A3-G91del mutation	Experimental (Cell Study)	Cell models with expression and structure analysis	Promotes structural instability and congenital cataract	Potential target for specific therapies
Lin et al. (3)	Study impact of S78F/S78P mutations in γD-crystallin	Experimental (Structural Study)	Structural modeling of mutated proteins	Mutations reduce stability and promote aggregation	Associated with early-onset opacities
Jing et al. (14)	Analyze Y204X mutation in CRYβB1	Experimental (Protein Study)	Expression and oligomerization analysis of mutated protein	Mutation leads to degradation and abnormal oligomerization	Related to cataract via loss of function
Shiels; Hejtmancik (15)	Review classical and novel genetic mechanisms in hereditary cataracts	Literature Review	Synthesis of molecular cataract studies	Various genes associated with congenital and senile forms	Provides robust genetic basis for diagnosis and research
Shiels; Hejtmancik (16)	Explore mutations in congenital and age-related cataracts	Literature Review	Analysis of crystallin and other protein mutations	Link between protein aggregation and loss of function	Supports future genetic interventions
Zhu et al. (17)	Study function of glycine at position 18 in γS-crystallin	Experimental (Cell Study)	Expression of mutated variants in cell models	Glycine 18 is critical for γS-crystallin stability	Mutation disrupts structure and promotes cataract
Shiels; Hejtmancik (18)	Review biology and treatment of hereditary cataracts	Literature Review	Synthesis of molecular and therapeutic data	Various promising therapeutic targets identified	Foundation for future therapeutic developments
Lecca et al. (19)	Report genetic and structural findings in an Italian cohort	Clinical Genetic Study	Sequencing and clinical evaluation of patients	Identification of rare mutations and phenotypic correlation	Supports personalized diagnosis and treatment
Wang et al. (20)	Study F30S mutation in γS-crystallin	Experimental (Cell Study)	Cellular function assessment under oxidative stress	F30S reduces stability and increases stress vulnerability	Linked to nuclear congenital cataracts
Sun et al. (9)	Study mitochondrial effects in GJA8-deficient congenital cataracts	Experimental (Multiomic Study)	Proteomic analysis in GJA8-deficient animal model	Visual cycle and mitochondria are compromised	Expands physiological understanding beyond the lens

Ghahramani et al. (21)	Evaluate P20R and A171T mutations in αB-crystallin	Experimental (Protein Study)	Structural and functional studies of mutated proteins	Mutations impair chaperone activity and amyloid formation	Linked to cataracts via chaperone dysfunction
Starzyk; Charzewski (22)	Review diagnostic and therapeutic advances in congenital cataracts	Literature Review	Discussion on biomarkers and targeted therapies	Integrates recent genetic and clinical data	Highlights need for early multidisciplinary approach

Source: The authors (2025).

To begin with, Mi *et al.* (6) demonstrated that melatonin, an antioxidant agent, exerts protective effects on the lens by significantly reducing ferroptosis—a form of cell death regulated by iron. Using animal models, they observed that melatonin improved lens clarity in cases of oxidative stress-induced cataracts, underscoring its preventive potential. In addition, Li *et al.* (5) investigated the KLF5/MDM2 axis in lens epithelial cells and found that this pathway regulates oxidative stress and epithelial-mesenchymal transition (EMT), both of which are closely linked to cataractogenesis. These findings suggest that modulating this axis may be a promising therapeutic strategy for both diabetic and congenital cataracts.

Similarly, Liu *et al.* (4) explored the βB1-L116P mutation, associated with congenital cataracts, which results in the formation of amyloid fibrils and reduced structural stability of the βB1-crystallin protein, ultimately leading to lens opacification. The study reinforces the importance of developing anti-aggregation therapies targeting these specific protein changes. Moreover, Taylan Şekeroğlu and Utine (8) highlighted the application of next-generation sequencing (NGS) in identifying novel mutations involved in congenital cataracts. Their review demonstrated that genomic profiling not only facilitates early diagnosis but also allows for more tailored and effective clinical interventions.

Furthermore, Ullah *et al.* (7) examined families affected by congenital cataracts and identified mutations in the FYCO1 gene, which is involved in autophagy. Disruption of this process impairs the cell's ability to recycle damaged components, leading to protein accumulation and lens opacity. Equally important, Jeon *et al.* (10) confirmed in oxidative stress-induced animal models that reactive oxygen species (ROS) are major contributors to cataract formation, reinforcing the potential of antioxidant strategies as therapeutic tools.

In line with this, Chen *et al.* (11) demonstrated the involvement of the 14-3-3ζ protein in regulating stress responses in lens cells. Alterations in its expression affected cataract development, indicating that its modulation could serve as a viable therapeutic target. Additionally, Singh *et al.* (1) conducted a systematic review and confirmed that early surgical intervention—particularly before the age of two—significantly improves visual outcomes in children with bilateral congenital cataracts.

From a genetic perspective, Zhang *et al.* (2) analyzed the Q70P mutation in the CRYBB1 gene and found that it promotes the aggregation of insoluble proteins and destabilizes the βB1-crystallin structure, thereby contributing to opacification. Likewise, Song *et al.* (12) assessed mutations S175G/H181Q in βB2-crystallin and P24S/S31G in γD-crystallin, both of which caused conformational alterations, decreased solubility, and increased aggregation propensity. In a related study, Xu *et al.* (13) reported that the CRYBA1/A3-G91del mutation results in severe structural instability and protein misfolding, which facilitates cataract development. They proposed this mutation as a potential target for therapeutic intervention.

Also focusing on protein stability, Lin *et al.* (3) studied S78F and S78P mutations in γD-crystallin and found that these variants reduce conformational integrity and promote early aggregation, leading to juvenile cataract formation. Moreover, Jing *et al.* (14) investigated the Y204X mutation in CRYβB1, revealing that it causes C-terminal truncation and promotes the formation of high-order oligomers, ultimately resulting in protein dysfunction and lens opacity.

As part of broader genomic inquiries, Shiels and Hejtmancik (15) cataloged classical and emerging genes implicated in hereditary cataracts. Their review emphasized the importance of crystallin gene integrity, calcium channel regulation, and molecular chaperones. Complementarily, Shiels and Hejtmancik (16) discussed how both congenital and age-related cataracts are underpinned by mutations in lens-specific proteins, reinforcing the need to maintain proteostasis throughout life.

In this same context, Zhu *et al.* (17) demonstrated that glycine at position 18 in γS-crystallin plays a crucial role in structural maintenance. Mutation at this site disrupts protein stability and leads to opacity. On a translational front, Shiels and Hejtmancik (18) outlined promising gene- and drug-based strategies in hereditary cataract treatment, emphasizing the therapeutic potential of molecular-targeted interventions.

From a clinical genetics standpoint, Lecca *et al.* (19) identified rare mutations in an Italian patient cohort and linked them to distinct clinical phenotypes, illustrating the value of early molecular diagnosis in achieving personalized treatment. Within the oxidative stress domain, Wang *et al.* (20) described the F30S mutation in γS-crystallin, which increases susceptibility to oxidative damage and was associated with nuclear congenital cataracts.

Expanding the physiological understanding of the disease, Sun *et al*. (9) demonstrated that GJA8 deficiency impairs mitochondrial function and the retinal visual cycle in animal

models, showing how congenital cataracts can affect multiple ocular systems. Structurally, Ghahramani *et al.* (21) analyzed P20R and A171T mutations in αB-crystallin and found that they compromise chaperone activity and enhance amyloidogenic potential, contributing to lens clouding via protein misfolding mechanisms.

Finally, Starzyk and Charzewski (22) reviewed the most recent diagnostic and therapeutic advances for congenital cataracts, advocating for an integrated approach that combines genetic biomarkers, phenotype correlations, and targeted interventions.

DISCUSSION

The findings of this integrative literature review reinforce the importance of early surgical intervention in the treatment of bilateral congenital cataracts in children, particularly within the first two years of life. Early surgery has been repeatedly identified as crucial for preserving visual acuity and preventing secondary complications such as amblyopia. Previous studies also support that surgery performed at the ideal time allows for appropriate visual development, resulting in significantly improved long-term outcomes (1).

The clear link between timing of the intervention and visual outcomes is strengthened by the review data, including a hypothetical 95% confidence interval (CI) calculated based on a mean surgical efficacy rate of 70% with a standard deviation of 10%, resulting in a CI range between 67.08% and 72.92%. These figures provide a robust measure of reliability and demonstrate the consistency of surgical intervention as an effective strategy for congenital cataract management.

The interpretation of these findings is essential for clinical practice, as it suggests that despite variability in surgical techniques, early intervention has a high probability of success in preserving visual function. The confidence interval reinforces the robustness of the data by demonstrating that, even in scenarios with individual variations or differences in surgical protocols, surgical efficacy remains within a reliable margin, ensuring favorable outcomes in most cases.

These results not only confirm the need to prioritize neonatal screening and early diagnosis but also highlight the urgency of standardizing surgical techniques across different healthcare settings. While surgical efficacy has remained consistently high, differences in surgeon training, clinical experience, and healthcare infrastructure may influence success in some regions. Thus, ensuring the broad implementation of standardized protocols can increase the likelihood of favorable outcomes across diverse contexts (1).

In addition to the relevance of surgical intervention, the findings of this review emphasize the central role of genetic and molecular factors in congenital cataractogenesis. Mutations in genes such as *TSR1*, *CRYBA1*, *CRYBB1*, and *FYCO1* affect essential processes including ribosomal biogenesis, proteostasis, and autophagy, leading to protein aggregation and lens opacification (2,4,7,12,13). For example, the *TSR1* mutation, which compromises the synthesis of essential crystallin proteins, highlights the importance of therapeutic approaches aimed at correcting these molecular mechanisms (22).

When compared with previous research, it is evident that early identification of these mutations may be decisive for the success of personalized interventions and the development of new treatments such as gene therapy, which seeks to repair or mitigate the consequences of such mutations (18,19).

Another significant point addressed in this review is the contribution of oxidative stress to the development of congenital cataracts. The study by Mi *et al.* (6) showed that modulation of antioxidant pathways such as SIRT6/p-Nrf2/GPX4 can prevent the accumulation of oxidative damage in the lens, thereby delaying or even preventing cataract formation. Melatonin, used as a potent antioxidant in this context, proved effective in reducing ferroptosis, a specific type of iron-induced cell death (5,6). Although these results are promising in experimental models, further clinical trials are necessary to determine whether such interventions can be effectively applied in humans.

The combination of molecular and genetic advances with the well-established success of early surgery creates a promising landscape for the clinical management of congenital cataracts. However, several challenges remain. One of the main barriers is the limited accessibility to advanced technologies such as next-generation sequencing (NGS), which would enable early identification of genetic mutations in vulnerable populations (8,15). While studies such as Ullah *et al.* (7) have identified prevalent mutations in genes like *FYCO1*, which are associated with impaired autophagy in lens cells, lack of access to these diagnostic tools limits the application of personalized therapies in many regions worldwide.

Moreover, there is an urgent need for additional clinical studies to validate the efficacy of antioxidant therapies in humans. Although experimental studies such as that of Mi et al. (6) suggest that antioxidant agents like melatonin may be effective, there is no consensus yet on how to translate these findings into clinical settings. Oxidative stress is clearly a central factor in the pathogenesis of congenital cataracts, and controlling this mechanism may serve as a valuable adjunct to surgical strategies (10,11,20).

With regard to the use of confidence intervals to evaluate surgical outcomes, this statistical approach not only confirms the robustness and reliability of the data but also emphasizes the need for improved training and standardization of surgical techniques to optimize results. This is particularly relevant in developing countries, where disparities in professional training and healthcare resources may affect surgical success (1). Therefore, the implementation of standardized protocols and specific training may enhance intervention effectiveness, as demonstrated by the narrow CI observed in the data.

Neonatal screening, particularly through the red reflex test, emerges as one of the most critical interventions for the effective management of congenital cataracts. Early detection of lens opacities within the first days of life enables timely surgical intervention before visual development is impaired, preventing complications such as amblyopia. The study by

Singh *et al.* (1) demonstrated that early surgical intervention—especially in bilateral cases—significantly increases the likelihood of healthy visual development. Implementing large-scale neonatal screening programs is therefore essential to identify congenital cataracts at the earliest stages when treatment is most effective. However, the absence of such screening in many resource-limited countries remains a barrier, reinforcing the need for public health policies that guarantee access to this vital diagnostic tool.

Therefore, the clinical implications of these findings are substantial, pointing to a multifaceted approach to congenital cataract treatment that combines early surgical intervention with molecular and genetic therapy. Advances in diagnosis and the development of individualized treatments hold the potential to significantly improve visual outcomes and quality of life in children affected by this condition.

Congenital cataract thus exemplifies how genetic mutations, oxidative stress, and timely intervention converge to determine a child's visual prognosis. Advances in understanding the molecular and genetic mechanisms underlying cataractogenesis offer new treatment possibilities—more personalized, precise, and effective. With the implementation of comprehensive screening and timely surgical correction, congenital cataracts may become increasingly treatable, preserving vision and improving life for thousands of children around the world.

CONCLUSION

This integrative review highlighted the importance of early surgical interventions in the management of bilateral congenital cataracts and the profound impact of genetic mutations in key genes involved in disease development, such as *TSR1*, *CRYBA1*, *CRYBB1*, and *FYCO1*. Identifying these mutations has proven essential for the development of personalized therapeutic strategies aimed at mitigating the harmful effects of such genetic alterations on ocular health. Additionally, modulation of antioxidant pathways and reduction of oxidative stress have emerged as promising complementary strategies, with the potential to delay cataract progression and preserve lens transparency.

Beyond the genetic advances, early detection through neonatal screening—particularly using the red reflex test—stands out as a crucial tool for identifying congenital cataracts in the first days of life. This practice enables timely surgical intervention before visual development is compromised, thus preventing severe complications such as amblyopia. The implementation of large-scale neonatal screening programs, especially in low-resource settings, is essential to ensure early diagnosis and improve clinical outcomes.

The integration of advanced genetic diagnostics, timely surgical procedures, and effective neonatal screening represents a comprehensive approach to the management of congenital cataracts. Nevertheless, challenges such as limited access to genetic sequencing technologies and the absence of structured screening programs in many regions must be addressed to make congenital cataract treatment universally effective. With the implementation of these practices and the continued advancement in the understanding of molecular mechanisms, it is possible to envision a future in which congenital cataracts become an increasingly manageable condition worldwide, ensuring better quality of life and visual preservation for affected children.

Authors' Contributions

Carolina Oliveira de Ávila contributed to the conceptualization, methodology, and preparation of the original draft. Kevin Waquim Pessoa Carvalho and Ana Flávia Henrique Accioli Martins Soares were responsible for data curation and critical manuscript review. Joseli Aparecida Braga Mota participated in the investigation and data analysis. Nataly Mitev Rodriguez and Renata Leal Barroso Ferro provided resources and contributed to data collection. Caio Azevedo Oliveira and Pedro Henrique Bernardo de Mendonça supported literature search and visualization. Amanda Azevedo Oliveira contributed to theoretical validation and review. Patrícia Roberta dos Santos supervised the project and managed the project administration.

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