sual acuity with no particular adverse cell proliferation or rejection when ESC-derived retinal pigment epithelial cells were transplanted into the subretinal space to treat age-related macular degeneration.[30] However, ESC therapy is limited by concerns regarding long-term safety and graft survival.

As an alternative, investigators have employed adult stem cell therapy (ASCT).[30] ASCT requires *ex vivo* manipulations that involve isolating, enriching, identifying, and growing adult stem cells before they can be used to replace any cells of the dysfunctional organs via transplantation and cell injection.[30] ASCT aims to allow normal, healthy cells to differentiate into functional cells in the target diseased tissues.[30,32]

Specifically, DSCs can be integrated into ASCT to regenerate and restore ocular tissues. This is because DSCs are derived from cranial NCC and may possess similar properties to neural crest progenitor cells that give rise to many structures of the anterior segment of the eye.[30,32] In fact, the study showed that when undifferentiated, immature human dental pulp stem cells (DPSCs) were transplanted into an animal model of limbal stem cell deficiency, it resulted in a reconstructed corneal epithelium, reduction in neovascularization, and clear cornea. This animal model involves extensive corneal damage and permanent visual impairment, and is often used to study the effects of stem cells in healing damaged tissues. This particular model and its limbal stem cell deficiency manifests as the lack of repopulation of corneal epithelium and is viable for testing the healing capacity of the DSCs. These results clearly demonstrate their capacity to replace limbal stem cells and restore the cornea.[30-32] Furthermore, the study showed that DPSCs may serve as an abundant source of retinal-like stem cells with the ability to differentiate into retinal neurons and photoreceptors.[30]

CONCLUSION

Continued research on ASD has allowed for identification of multiple genes associated with the condition, several of which include *Pitx2*, *Foxc1*, *Tfap2b*. It has been found that these genes do not work independently of one another. Rather, they regulate or affect one another in the process of POM specification into anterior segment tissues such as the components of the corneal layers and structures of the iridocorneal angle. Research has also suggested the possibility of sequential formation of anterior segment tissues. This was clearly demonstrated in the lens ablation experiment, in which the lens allowed for subsequent specification of the POM into the anterior structures through inductive signalling. This review paper aimed to emphasize the importance of proper embryonic development of the anterior structures and the possible complications that can arise as a result of its improper development. These complications include ARS, subcapsular cataracts, and glaucoma. Finally, a potential treatment using adult stem cells, specifically dental stem cells, has highlighted the possibility of regenerating the damaged cornea frequently resulting from ASD. Future steps include further investigation of the AP-2β NCC KO mouse mutants and determination of whether knockouts at different time points during embryonic development yield different clinical manifestations. Regenerative medicine, such as the use of DSCs, should be further validated and advanced to human trials in order to treat the millions affected by ASD.

ACKNOWLEDGEMENTS

Special thanks to Judith West-Mays and Vanessa Martino, a graduate student in West-Mays' lab performing all experiments on AP-2β NCC mutants. These two inviduals have provided guidance towards finding relevant literature that helps with knowledge-building in the field of ophthalmology, specifically with regards to anterior segment dysgenesis and AP-2β. ■

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