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# Effect of Travoprost on Cell Viability, Proliferation and Migration in the Sirna-Based Aniridia Limbal Epithelial Cell Model and in Primary Aniridia Limbal Stromal Cells, in Vitro

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**Abstract :** Purpose: Aniridia associated keratopathy (AAK) is a progressive condition commonly observed in individuals with congenital aniridia, with PAX6 haploinsufficiency. AAK can lead to limbal stem cell deficiency and progressive ocular surface damage. The dysfunction of limbal epithelial and stromal cells (LECs and LSCs) potentially plays a key role in AAK pathogenesis. Travoprost, a prostaglandin analog, has been shown to influence cellular behavior in various cell types, but its effects on primary aniridia LECs and LSCs remain unclear. This study aims to evaluate the impact of travoprost on cell viability, proliferation, and migration in LECs, in the siRNA-based limbal epithelial cell model and in primary aniridia LSCs, in vitro. Methods: Primary human LECs were extracted from heathy donors, and siRNA treatment was used to mimic PAX6 haploinsufficiency in congenital aniridia. Primary human LSCs were extracted from heathy and aniridia donors (AN-LSCs). LECs, LSCs and AN-LSCs were treated with 0.039-40 µg/ml travoprost, for 20 minutes. The XTT and BrdU assays were used to evaluate the effect of travoprost on cell viability and proliferation (n=7). Cell migration assay was performed following siRNA-based PAX6 knockdown and subsequent 0.313 and 0.156 µg/ml travoprost treatment for 20 minutes (n=5). Results: LECs and LSCs viability decreased significantly from 0.156 µg/ml and ANLSCs viability decreased significantly from 0.078 µg/ml travoprost concentration ($p=0.0279$, $p<0.0001$, $p=0.0002$). In all cell types, there was a stepwise decrease in cell viability, as the travoprost concentration increased ($p<0.0001$). Travoprost treatment did not change cell proliferation in LSCs ($p\geq0.0892$). In LECs, 20 and 40 µg/ml travoprost concentration, in AN-LSCs 40 µg/ml travoprost concentration exhibited reduced cell proliferation, compared to untreated controls ($p=0.0291$, $p=0.0041$, $p=0.0011$). PAX6-knockdown LECs exhibited lower migration rates at 6, 12, and 24 hours ($p=0.0015$, $p=0.0471$, $p=0.0009$) than control siRNA treated LECs, following travoprost treatment. In contrast, AN-LSCs demonstrated higher migration rates at the same 3 time points, than LSCs, after treatment ($p=0.0225$, $p=0.0383$, $p=0.0155$). In addition, among AN-LSCs, migration rate at of the 0.313 µg/ml travoprost treated group at 6 hours was significantly higher, than in the untreated control group. Conclusions: Our results demonstrate that travoprost may exert different effects on LECs, PAX6-knockdown LECs, LSCs, and AN-LSCs regarding cell viability, proliferation, and migration. AN-LSCs appear to exhibit greater sensitivity to travoprost treatment, than LSCs, therefore, topical antiglaucomatous treatment should be selected with caution for patients with congenital aniridia. Further in vivo measurements are necessary to evaluate the potential role of travoprost on AAK.

**Keywords :** congenital aniridia, travoprost, primary limbal epithelial cells, primary limbal stromal cells
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