ing eye health. Furthermore, the relationship between AMD and this variant in other ethnic groups, and thus the possibility for systemic errors in other groups, remains largely unexplored.

As our results highlight, predictive genetic testing for complex diseases faces many challenges. Until we fully understand how a particular genetic variant acts on disease susceptibility, great care must be taken when translating genetic tests from one race-ethnicity to another.

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focus on infants particularly likely to develop ROP. However, any study of the effects of β-blocker therapy must address the fragility of the patients to be tested and possible systemic and ocular adverse effects. Nevertheless, if topical β-blockers prove to be effective in preventing some cases of ROP, this opens the door for a more individualized approach to prevention of the disease, e.g., using β-adrenergic receptor polymorphisms to guide ROP management.

Table. All Consented Patients With Type 1 ROP in at Least 1 Eye

<table>
<thead>
<tr>
<th>Birth Weight, g</th>
<th>Race</th>
<th>Consented Patients With ROP, No. (N = 2320)</th>
<th>Patients Developing Type 1 ROP, No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;750</td>
<td>Non–African American</td>
<td>623</td>
<td>186 (29.9)</td>
</tr>
<tr>
<td></td>
<td>African American</td>
<td>315</td>
<td>36 (11.4)</td>
</tr>
<tr>
<td>750–999</td>
<td>Non–African American</td>
<td>726</td>
<td>140 (19.3)</td>
</tr>
<tr>
<td></td>
<td>African American</td>
<td>233</td>
<td>18 (7.7)</td>
</tr>
<tr>
<td>≥1000</td>
<td>Non–African American</td>
<td>331</td>
<td>25 (7.6)</td>
</tr>
<tr>
<td></td>
<td>African American</td>
<td>92</td>
<td>1 (1.1)</td>
</tr>
<tr>
<td>Total</td>
<td>Non–African American</td>
<td>1680</td>
<td>351 (20.9)</td>
</tr>
<tr>
<td></td>
<td>African American</td>
<td>640</td>
<td>55 (8.6)</td>
</tr>
</tbody>
</table>

Abbreviation: ROP, retinopathy of prematurity.


Epithelial Downgrowth After Type 1 Boston Keratoprosthesis Manifesting as Tractional Retinal Detachment and Epiretinal Membrane

Type 1 Boston keratoprosthesis (KPro) is a viable treatment option for corneal disease at high risk for graft failure with traditional penetrating keratoplasty. Postoperative complications of Boston KPro include retroprosthetic membrane, glaucoma, sterile vitritis, infectious endophthalmitis, corneal melt, extrusion, and retinal detachment.1 To our knowledge, we report the first case of epithelial downgrowth (ED) of the posterior segment after Boston KPro placement.

Report of a Case. A 52-year-old man with a history of penetrating ocular injury to his right eye had open globe repair and cataract extraction in 1974, placement of a secondary anterior chamber intraocular lens in 1992, 2 failed penetrating keratoplasty procedures in 2004 and 2008, Baerveldt glaucoma tube implantation in 2005, astigmatic keratotomy in 2007, and, most recently, intracocular lens removal and type 1 Boston KPro placement in 2010. Histologic examination of the failed corneal graft excised at the time of KPro placement did not demonstrate ED. One week after KPro placement, visual acuity was 20/40 OD. Two months after KPro placement, he had pain and photophobia in the right eye.