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 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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β-Blocking and Racial Variation in the Severity of Retinopathy of Prematurity

In the Early Treatment for Retinopathy of Prematurity Study, the incidence of retinopathy of prematurity (ROP) was the same among African American and non–African American infants; however, once ROP was observed, the incidence of progression to severe (prethreshold) ROP occurred more commonly among non–African American infants. Herein, we expand on these findings and present a possible cause and research approach focused on prevention of some cases of severe ROP.

Methods. Infants weighing less than 1251 g were logged at each participating center. These infants were followed up for the development of ROP and its progression in severity by study-certified ophthalmologists. We report the incidence of progression from the onset of ROP to type 1 ROP in at least 1 eye by birth weight and race. Type 1 is defined as zone I ROP at any stage with plus disease, as zone I stage 3 ROP, or as zone II stage 2 or 3 ROP with plus disease.

Results. The Table shows the percentage of infants by birth weight (<750, 750-999, and ≥1000 g) who had any ROP and the percentage who developed type 1 disease. The estimated incidences of ROP for both African American and non–African American infants weighing less than 1251 g at birth were essentially the same, 68%. However, the proportions of consented infants with ROP who ultimately developed type 1 ROP differed significantly. Non–African American infants had a much higher incidence of type 1 ROP in all weight categories and overall compared with African American infants (overall, 20.9% vs 8.6%, respectively; P <.001).

Comment. In this large study of infants weighing less than 1251 g at birth, a striking reduction in type 1 ROP is seen in African American infants. A similar finding for prethreshold ROP has also been reported in the Cryotherapy for Retinopathy of Prematurity Study. One mechanism to explain some of the observed racial differences in ROP invokes β-blocker receptor polymorphisms, which exist in many African American people. The effect of such polymorphisms renders the person “β-blocked.” If this β-blockade status were protective for ROP, then it could be a reason for the relative immunity to severe disease seen in many African American infants. More importantly, it would suggest that treatment with a β-blocker for infants devoid of β-adrenergic receptor polymorphisms could be beneficial. This polymorphism theory is supported by recent reports indicating an association of cutaneous hemangiomas with ROP and a possible common pathogenesis. Cutaneous hemangiomas show a dramatic reduction with treatment using systemic β-blockers. Cutaneous hemangiomas are far more common in white infants and are very uncommon in African American infants, again suggesting that β-blocker receptor polymorphisms could influence angiogenesis and prevent hemangiomas. β-Adrenergic receptors exist on retinal endothelial cells. However, the exact mechanisms or effects of β-adrenergic receptor blockers on blood vessel growth have not been elucidated.

Prevention is the next frontier in ROP research. It is highly plausible that β-blockers could be effective in preventing ROP. Pharmacokinetic studies indicate that topical betaxolol hydrochloride shows good ocular penetration and reaches the posterior aspect of the eye in good concentrations. By studying the effects of topical betaxolol in infants with birth weight less than 1000 g, we will...
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, eg, 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, intraocular 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.