apolipoprotein E-deficient mice. *Invest Ophthalmol Vis Sci* 2000; **41**: 2035.
10 Kureishi Y, Luo Z, Shiojima I, Bialik A, Fulton D, Lefer DJ *et al*. The HMG-CoA reductase inhibitor simvastatin activates the protein kinase Akt and, promotes angiogenesis in normocholesterolemic animals. *Nat Med* 2000; **6**: 1004–1010.

A Misra[1], NK Vikram[1] and A Kumar[2]

[1]Department of Medicine
All India Institute of Medical Sciences
New Delhi 110029, India

[2]Dr RP Center for Ophthalmology
All India Institute of Medical Sciences
New Delhi 110029, India

Correspondence: Dr A Misra
Center for Human Nutrition
Department of Internal Medicine
The University of Texas
Southwestern Medical Center at Dallas
5323 Harry Hines Blvd, Dallas
TX 75390-9052, USA
Tel: +1 214 648 3198
E-mail: anoopmisra@hotmail.com
Anoop.Misra@Utsouthwestern.edu

*Eye* (2004) **18,** 107–108. doi:10.1038/sj.eye.6700530

Sir,

**Lipids, diabetic maculopathy, and cardiovascular risk**

Broader messages on diabetic eye care are deducible from the review by Chowdhury *et al*[1] evaluating the role of serum dyslipidaemia in diabetic maculopathy. The evidence they have surveyed indicates that dyslipidaemia has somewhat congruously joined poor glycaemic control and hypertension as cardiovascular risk factors that affect macular health in diabetes. In contemporary practice, a characterisation of risk factors for diabetic maculopathy is of monumental importance in public health terms. Type 2 diabetes is now manifesting its explosive demography in Britain with an astonishing volume of diabetic eye disease, of which maculopathy is a foremost concern. This iatrogenic fear is generated, for example, by the many patients with advanced ischaemic diabetic maculopathy who have disease untreatable by laser. Exudative maculopathy is similarly not always responsive to laser and noticeably problematic are the larger lipid deposits (especially plaques) that form in the central macula. In some diabetic eye services in Britain, the prevalence of this fundal picture is striking. Gross lipid exudation invites the same therapeutic nihilism associated with ischaemic maculopathy, because regardless of whether the tissue insult is ischaemic or exudative, the end-organ damage becomes irredeemable. In the wake of the UKPDS, preventative strategies therefore are eminently rational and a manipulation of the lipid profile as a potential adjunct to laser seems particularly compelling.

Moreover, it is arguable that ophthalmologists should develop more than a passing interest in cardiovascular risk factors since beyond the eye these factors are a leading cause of systemic morbidity and death. After all, in counterbalance, physicians undertake retinopathy screening with modest equipment. Rather than being a perfunctory exercise, a purposeful inquiry into a patient's prevailing glycaemic control, blood pressure, and lipid status (among other factors) in an ophthalmic consultation will stimulate remedial referral patterns. Indeed, even in its simplest guise the interchange with the ophthalmologist should be an opportunity to reiterate the messages behind diabetes care to our patients. By judiciously checking blood pressure and glucose on clinical suspicion, or looking up glycaemic and lipid indices, we are not (as may be the criticism) becoming diabetologists, but rather contributing in managing an unwieldy and major public health problem. This approach ought to be the strategy for any ophthalmologist examining diabetic retinopathy and not exclusively the mantra of those providing a medical retina service. To contextualise the issue for ophthalmologists yet again, diabetic eye disease is by far the most common cause of poor vision in our society among people of working age. Since the 1970s our selectively efficacious solution for maculopathy remains a Hamletesque 'to laser or not to laser' (a stabilising intervention), and even this decision is sometimes debatable and subject to the treatment ethos of a given clinician or department.

Beyond reducing macrovascular complications, an expansion of the remit of lipid-lowering therapy in managing maculopathy would represent a highly desirable therapeutic convenience in diabetes care. A large randomised controlled trial to pass final judgement on this wishful speculation can conceivably be the next landmark study in the medical management of diabetic retinopathy.

**References**

1 Chowdhury TA, Hopkins D, Dodson P, Vafidis GC. The role of serum lipids in exudative diabetic maculopathy:

is there a place for lipid lowering therapy? *Eye* 2002; **16**(6): 689–693.

JS Gandhi

Department of Ophthalmology
Charing Cross Hospital, Fulham Palace Road
London W6 8RF, UK

Correspondence: JS Gandhi
Tel: +44 20 8846 1497
Fax: +44 20 8846 1911
E-mail: jag_gandhi@hotmail.com

*Eye* (2004) **18,** 108–109. doi:10.1038/sj.eye.6700531

Sir,

**Reply**

I read with keen interest, the article titled 'contact lenses in the management of high anisometropic amblyopia by Roberts and Adams.[1] The authors pointed out that high anisometropic amblyopia is challenging to treat, and there is a good chance of improvement in visual acuity using contact lenses and occlusion in anosometropia of 6 diopter.[2]

In India, social and climatological circumstances make the wearing of contact lenses by children difficult, since most of our patients are from lower socioeconomic strata. These patients are commonly lost to follow-up, and are not available for timely examination of cornea and optical adjustments in contact lens wearing. We are of the opinion that in such cases, clear lens extraction with intraocular lens implantation is appealing, because most ophthalmologists use this procedure and good visual outcome can be achieved. Lyle and Jin[2] achieved a visual acuity of more than 6/12 in 89% eyes with clear lens extraction and intraocular lens implantation. We also observed an improvement in visual acuity of more than two lines in 80% patients.

**References**

1 Roberts CJ, Adams GGW. Contact lenses in the management of high anisometropic amblyopia. *Eye* 2002; **16**: 577–579.
2 Lyle WA, Jin GJC. Clear lens extraction for correction of high refractive error. *J Cataract Refract Surg* 1994; **20**: 273–276.

S Dadeya

Guru Nanak Eye Center, Maulana Azad Medical college, New Delhi 2, India

Correspondence: S Dadeya
Tel: +91 11 656 4225
Fax: +91 11 6166 1013
E-mail: dadeya86@hotmail.com

*Eye* (2004) **18,** 109. doi:10.1038/sj.eye.6700532

Sir,

**Reply**

We thank Dr Dadeya for his comments on our paper. We are well aware of the social and climatological difficulties that can be experienced when suggesting the use of contact lenses in children and note his comments on the difficulties of follow-up in certain patient groups. However, we do have significant concerns about intraocular surgery for anisometropia in the paediatric age group because of the potential for serious complications including posterior capsule opacification, glaucoma, marked anterior uveitis with synechiae or membrane formation, and retinal detachment. Unpredictable refractive outcomes and higher reoperation rates are well recognised in paediatric cataract surgery. The issues surrounding paediatric lens implantation have recently been highlighted in an editorial in the *Journal of AAPOS*.[1] It is true that some groups have reported excellent visual results from implant surgery in older children; however, close follow-up is important and posterior capsular opacification is still an issue. Occlusion therapy for amblyopia is still required to gain a visual result after surgery and will require regular attendance at clinic. In the context of a society in which follow-up may be challenging, the amblyogenic effect of capsule opacification is of great concern and certainly may prove to be more amblyogenic than the original presenting anisometropia.

**References**

1 Levin AV. IOLs, innovation and ethics in pediatric ophthalmology: let's be honest. *J AAPOS* 2002; **6**(3): 133–135.

CJ Roberts[1] and GGW Adams[2]

[1] Royal Eye Unit, Kingston Hospital
Glasworthy Road, Kingston-upon-Thames KT2 7QB
UK

[2] Moorfields Eye Hospital
City Road
London EC1V 2PD, UK