photographic macular assessment (10.8% of those with dietary assessments). This is one area of concern. The dietary assessment method (food frequency questionnaire, FFQ) was not conducted at baseline, which only allows measurements of association from the follow up examination. Owing to the cross sectional nature of the data, it is plausible and even likely that participants with known signs of early macular degeneration or associated visual changes may have increased their dietary antioxidant intakes (indication bias)—for example, after being told about their signs at the first examination or at other times. This bias may have occurred in particular among those consuming higher linoleic acid diets as higher intakes of linoleic acid have been reported to increase the risk of AMD.[2] An excellent example of this indication bias can be seen in the finding of a significantly increased risk of poor night vision associated with increased consumption of carrots.[3]

The letter also states that a possible protection existed with high LZ intake on AMD among those with low levels of linoleic acid intake. We could, however, not see any data in the results or tables to support this statement.

We thought that these findings needed to be confirmed in other study populations. Given our concerns about the cross sectional data design we explored this association with the incidence of AMD in the Blue Mountains Eye Study cohort. Baseline data were collected in 1992–4 from 3654 residents of the Blue Mountains aged 49 years and over. Eye examinations were conducted on residents at baseline and at follow up using retinal photographs and the Wisconsin AMD grading system.[4] Dietary data were assessed at baseline (n = 2900) and follow up examinations using a 145 item food frequency questionnaire (FFQ).[5] Of the participants examined at either or both the 5 year and 10 year examinations, 2454 had retinal photographs available for the assessment of age related maculopathy (ARM) lesions. Of the 2454 participants 2083 had complete FFQ data, including 818 supplement users. We used the Willett method[6] to energy adjust the linoleic and LZ data and investigated those with less than and greater than median intake for linoleic acid (median = 6.6 g) and 1 standard deviation increases of LZ (mean intake of energy adjusted LZ intake was 819 μg, with an SD of 475 μg), using a multivariate adjusted discrete logistic model to assess factors associated with 10 year incident AMD.

We found no association with energy adjusted LZ intake and the incidence of early, late, or any AMD, whether or not this was stratified by linoleic acid intake (table 1). Given that our median linoleic acid intake was less than the median used by Vu *et al*[1] (6.6 g verses 7.2 g) we also stratified the data by the highest tertile of linoleic acid intakes (cut point 8.5 g) and also found no association between LZ and incident AMD (data not shown).

While the examination of cross sectional data to investigate associations with disease may be useful, conclusions drawn from such data need to be made with care in the light of other known literature. Other data have demonstrated a potential protective effect from foods containing LZ[7] and foods and supplements rich in LZ are known to increase (protective) macular pigment density.[8] In addition, dietary guidelines[9] include recommendations to increase vegetables and fruit that are rich in LZ (for example, broccoli, green beans, silverbeet, brussel sprouts, oranges).[10] The authors' conclusions, based on their very limited data, are non-intuitive and we are concerned that they are potentially misleading. Care needs to be taken to continue to encourage the vegetable and fruit intakes of populations.

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Do retinopathy signs in non-diabetic individuals predict the subsequent risk of diabetes?

We read with interest the article by Wong *et al*,[1] which studied a non-diabetic population consisting of 7992 people aged 49–73 years. Non-mydriatic retinal photographs of one eye were taken and graded for retinopathy lesions using standardised protocols. Surprisingly, the presence of typical retinopathy lesions (microaneurysms or retinal haemorrhages) in people without diabetes did not significantly predict subsequent development of diabetes over a period of 3.5 years. Incident diabetes developed in 4.7% and 3.6% of people with and without retinopathy lesions at baseline, respectively, with a multivariate adjusted odds ratio, OR, 1.1, 95% confidence interval, CI, 0.7 to 1.9. However, in people with a family history of diabetes, presence of retinopathy lesions at baseline predicted a doubling of the risk of incident diabetes (OR 2.0, CI 1.1 to 3.8).

We previously reported findings from the Blue Mountains Eye Study cohort (BMES, n = 3654) that the 5 year risk of developing diabetes in people without diabetes but with retinopathy lesions at baseline was 3.5% (7/202).[2] The BMES examined 3654 participants at baseline (1992–4) and re-examined 2335 participants (75% of survivors) 5 years later (1997–9). Dilated six field retinal photographs of all participants were taken at the baseline and follow up examinations. Diabetes was diagnosed either from medical

Table 1 Odds ratio between baseline dietary lutein and zexanthin (LZ) intake, stratified by linoleic acid (LA) intake (less than and greater than median intake), and 10 year incident AMD in the Blue Mountains Eye Study

	Any AMD OR* (95% CI)	p Value	Early AMD OR* (95% CI) (n = 220)	p Value	Late AMD OR* (95% CI) (n = 59)	p Value
All participants						
Daily energy adj LZ intake	0.94 (0.71 to 1.24)	0.668	0.95 (0.71 to 1.28)	0.758	0.81 (0.45 to 1.50)	0.475
Energy adj LZ intake, <6.6 g LA	1.01 (0.71 to 1.45)	0.943	1.06 (0.73 to 1.53)	0.771	0.66 (0.30 to 1.46)	0.306
Energy adj LZ intake, ≥6.6 g LA	0.85 (0.54 to 1.34)	0.487	0.82 (0.51 to 1.32)	0.820	1.18 (0.51 to 2.77)	0.698
Excluding those who took supplements						
Daily energy adj LZ intake	1.06 (0.74 to 1.52)	0.749	1.11 (0.76 to 1.61)	0.596	0.85 (0.41 to 1.77)	0.666
Energy adj LZ intake, <6.6 g LA	1.10 (0.70 to 1.72)	0.675	1.15 (0.72 to 1.83)	0.559	0.62 (0.23 to 1.68)	0.349
Energy adj LZ intake ≥6.6 g LA	0.98 (0.54 to 1.79)	0.949	1.01 (0.54 to 1.89)	0.975	1.40 (0.49 to 3.96)	0.528

*Adjusted for age, sex, and smoking.

history or fasting blood glucose ⩾7.0 mmol/l at examination. Of the baseline participants without diabetes, 202 had retinopathy lesions (haemorrhages, microaneurysms, soft and hard exudates). Our 3.5% diabetes incidence over 5 years in this group is relatively similar to the 4.7% diabetes incidence over 3.5 years reported by Wong *et al*[1] in people with retinopathy lesions at baseline. These consistent findings suggest that retinopathy lesions occurring in people without diabetes are likely to have multiple aetiologies. In people with a family history of diabetes, retinopathy lesions may indicate a preclinical stage of diabetes. In the great majority of people without diabetes, however, retinopathy lesions are not necessarily linked to blood glucose. Reports from the BMES[3] and Hoorn Study[4] showed that baseline impaired fasting glucose or impaired glucose metabolism did not predict incident retinopathy lesions in people without diabetes. Older age and blood pressure, however, were strongly related.[2 5] It is possible that the same phenotype can result from different pathogenic conditions (such as hypertension[6]) that damage the microvasculature. Given that retinopathy lesions predict systemic vascular outcomes,[7] further research to clarify the causes of these lesions is warranted.

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NOTICES

Back of the eye

The latest issue of *Community Eye Health* (No 57) assesses treatments for age related macular degeneration and other back of the eye conditions. For further information please contact: Journal of Community Eye Health, International Resource Centre, International Centre for Eye Health, Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, UK (tel: +44 (0)20 7612 7964; email: Anita.Shah@lshtm.ac.uk; online edition: www.jceh.co.uk). Annual subscription (4 issues) UK £28/US$45. Free to developing country applicants

Managing human resources

The latest issue of *Community Eye Health* (No 56) assess the use of human resources in the delivery of eye care. For further information please contact: Journal of Community Eye Health, International Resource Centre, International Centre for Eye Health, Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, Keppel Street, Lodon WC1E 7HT, UK (tel: +44 (0)20 7612 7964; email Anita.Shah@lshtm.ac.uk, url: ; online edition: www.jceh.co.uk). Annual subscription (4 issues) UK £28/US$45. Free to developing country applicants.

8th EUNOS Meeting – 2007

The 2007 European Neuro-ophthalmology Society meeting (EUNOS; www.eunos.web.org) will be taking place in Istanbul, Turkey on 26-29th May 2007. For further information please visit www.eunos2007.org or contact Pinar Aydin aydinp@eunos2007.org

Teaching courses on Retinal and Vitreous Surgey

Several teaching courses on Retinal and Vitreous Surgery have been organised throughout 2006 and 2007 around the world in association with the International Faculty. For further information on each of these courses please contact Ingrid Kressig, Univ.-Augenklinik Theodor-Kutzer-Ufer 1-3, 68164 Mannheim, Germany; email: Ingrid.kreissig@augen.ma.uni-heidelberg.de, url: ; website: http://kressig.uni-hd.de/.

EVER 2006

The EVER 2006 meeting will take place in Vilamoura, Portugal on 4-7th October 2006. For further information please contact the EVER Office, Kapucijnenvoer 33, 3000 Leuven, Belgium; website www.ever.be

Recruitment halted on ESCRS study on antibioltic prophylaxis of endophthalmitis following clear beneficial result

THE ESCRS has terminated recruitment for their two year study of antibiotic prophylaxis of endophthalmitis following cataract surgery. Quarterly analysis of the figures to date by the study's statisticians at the University of Strathclyde clearly indicates a beneficial treatment effect. In January 2006 the Data Montioring Committee recommended that the study be unmasked and found the result to be so clear that they recommended to the Study Chairman that recruitment be halted.

The study has found that the risk of contracting endophthalmitis following phacoemulsification cataract surgery is significantly reduced by an intracameral injection of cefuroxinme at the end of surgery.

The escrs Study, a partially masked, randomized, placebo controlled, multi-national study conducted at 24 ophthalmology centres across Europe commenced recruitment in September 2003. a preliminary report on the primary results will be published in the march 2006 issue of the Journal of Cataract and Refractive Surgery. Complete follow-up data and analyses will be reported at the XXXIV Congress of the ESCRS in London in September 2006 and will subsequently be published in the Journal of Cataract and Refractive Surgery.

For further information contact Caroline Fitzpatrick European Society of Cataract and Refractive Surgeons, tel: +353 1 209 1100, caroline.fitzpatrick@escrs.org.

Prevention of Blindness Fellowship Programme

Application are invited for BCPB Fellowships to start in 2007. The aims of the Fellowships are to fund research and training in prevention of blindness for high caliber clinicians and scientists from the UK and overseas. Projects must further the goals of VISION 202: THE RIGHT TO SIGHT, the elimination of avoidable blindness. In 2007, BCPB seeks to fund one Fellow from the UK and one Fellow from a low-income country to undertake projects that focus on Africa.

Priority will be given to applicants who:

- Demonstrate that their project is innovative and increases knowledge of the causes of blindness and/or its prevention in line with the priorities of VISION 2020
- Demonstrate the ability and ambition to pass on their skills in blindness prevention

The fellowships will be worth up to £60,000pa for 2 or 3 years. Applications must be submitted jointly by the Fellowship candidate and the supervisor at the host institution in the UK.

For full information and an application form, see www.bcpb.org, email: or contact Jackie Webber at BCPB, 59-60 Russell Square, London WC1B 4HP or by email: info@bcpb.org. Closing date for applications is 30 September 2006.