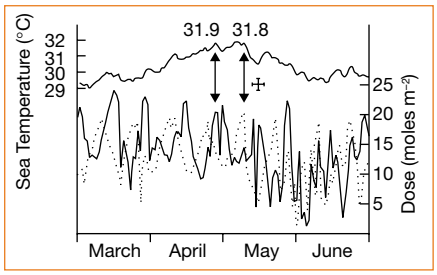

Figure 1 Daily dose of photosynthetically active radiation (PAR) underwater at the depth of *G. aspera* colonies in 1995. Solid line, west surface; dotted line, east surface. Top trace, daily mean sea temperature; double-headed arrows, maximum combined sea temperature and PAR doses for east (8 May) and west (26 April) surfaces; cross, bleaching first recorded (14 May).

G. aspera colonies to temperature bleaching in May 1995. First, given that different genotypes of the symbiotic algae on the Caribbean coral *Montastrea annularis* show differential bleaching susceptibility[6], we tested whether the east and west surfaces of *G. aspera* hosted different algal genotypes. Molecular analysis of the ribosomal RNA gene complex revealed no variation between algae from the two surfaces, either by analysis of restriction-fragment length polymorphisms of the small-subunit-rRNA gene[6] or by sequence analysis of the ITS1–5.8S–ITS2 region, which resolves finer molecular differences[7,8]. We conclude that the bleaching pattern observed in *G. aspera* is not due to genetic differences between their algae of the sort previously reported in *M. annularis*[6].

Second, we investigated whether the east surfaces of *G. aspera* colonies received more solar radiation than the west surfaces at the time of temperature bleaching in May 1995. During solar bleaching (January–March), the dose of PAR was consistently higher on the west than on the east surfaces, but from late April this difference disappeared. The maximal combined sea temperature (31.8–31.9 °C) and solar radiation were recorded on 26 April (for the west surfaces) and 8 May (for the east) — before the first observation (14 May) of temperature bleaching (Fig. 1). So the east surface did not receive more solar radiation than the west at the time of temperature bleaching.

Finally, to establish whether west surfaces are more tolerant to increased temperature, we subjected core samples from the west and east surfaces to different temperatures (elevated, 34 °C; ambient, 27 °C) under identical irradiance for 3 days (Fig. 2). At 27 °C, the algal density and chlorophyll *a* content per algal cell were similar for the east and west cores, and did not vary between the start and end of the experiment. Exposure of the east cores to 34 °C, however, resulted in significantly reduced algal density (Mann–Whitney *U*-test: $P < 0.01$) and caused an increase in the chlorophyll *a* content per algal cell

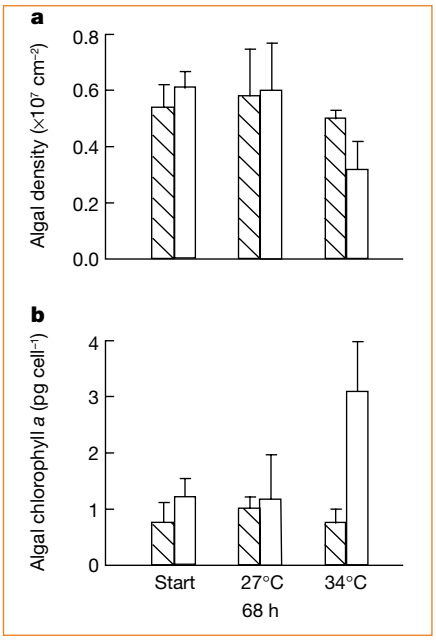

Figure 2 Physiological parameters in east and west cores of *G. aspera* before and after exposure to elevated (34 °C) and ambient (27 °C) temperatures for 68 h at an irradiance of 100 μmol m^{-2} s^{-1}. Mean values for each parameter are shown; error bars show one standard deviation ($n = 5$). **a,** Algal density in west (hatched) and east (clear) cores at the start and end of the experiment at the different temperatures. **b,** Algal chlorophyll *a* concentrations.

($P < 0.008$), whereas west cores were unaffected. Algal chlorophyll *a* was increased in other recently bleached corals[9,10].

Our findings indicate that the west surfaces of *G. aspera* colonies could have been protected against temperature bleaching in May 1995 because they were more tolerant than the east surfaces towards the combined stress of temperature and solar radiation. This tolerance probably arose as a result of the increased solar radiation received by the west surfaces during January–March. We conclude that experience-mediated tolerance, as well as algal genotypic differences[6], contributes to the variation in bleaching susceptibility among reef corals. Many coral colonies are long-lived[11], and experience effects may have an important influence on the bleaching responses to global climate change over the coming decades.

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Vision

Myopia and ambient night-time lighting

Myopia is a common affliction (one in four adult Americans is near-sighted[1]), and juvenile-onset myopia is believed to be due to a combination of genetic and environmental factors[2]. Results from animal experiments indicate that light cycles may affect the development of myopia[3,4], and Quinn *et al.* claim to have extended these to humans[5]. They reported a strong association between childhood myopia and night-time lighting before the age of two: there were five times more children with myopia among those who slept with room lights on than in those who slept in the dark, and an intermediate number among those sleeping with a dim night-light[5]. However, we have been unable to find a link between night-time nursery lighting and the development of myopia in a sample of schoolchildren.

We examined the issue of nursery lighting in a subsample of children from the multicentre Collaborative Longitudinal Evaluation of Ethnicity and Refractive Error (CLEERE) Study. Parents reported their use of night-time lighting and their own refractive status, and the child's refractive error was measured by cycloplegic autorefraction. Our sample consisted of 1,220 children with a median age of 10.2 years: 11.5% of them were African–American, 19.1% Asian, 47.9% Caucasian and 21.6% Hispanic; overall, 18.1% of them were myopic (at least −0.50 dioptres spherical equivalent). The proportion of children with myopia did not differ across nursery-lighting groups ($\chi^2 = 2.62$, $P = 0.271$). Eighty-four of 417 children (20.0%) who slept in darkness were myopic; 128 of 758 children (16.8%) who slept with a night light before age two were myopic, and 10 of 45 children (22.2%) who slept with full room lights on before age two were myopic.

We found an association between the number of myopic parents and nursery lighting before age two ($\chi^2 = 35.02$, $P < 0.001$), as well as an association between ethnicity and room lighting ($\chi^2 = 89.22$, $P < 0.001$). This sample carries a statistical power of 0.99 to be able to detect an odds ratio of 2.00 between nursery lighting and childhood myopia.

Our results do not replicate those of Quinn *et al.*[5]. In fact, the proportion of myopic children in those subjected to a

range of nursery-lighting conditions is remarkably uniform. The association we find between parental myopia and nursery night-time lighting suggests that Quinn *et al.*'s study should have controlled for parental myopia.

Another possible difference is that Quinn *et al.*'s sample is not representative of juvenile myopes. It was drawn from a tertiary referral, paediatric ophthalmology outpatient clinic, and the sample had a median age of eight (young for a sample of myopes) with a very high proportion of myopia (30%). Our sample had fewer myopes and fewer hyperopes, and the children were older. Also, the proportion of parents reporting that their infants slept under full lighting is different in our study: more than 15% of their clinic-based sample had full nursery lighting, whereas only 3.7% of our representative, school-based sample had full room lighting at night.

Our results indicate that myopia is unlikely to develop in children as a result of exposure to night-time lighting as infants.

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Quinn *et al.* report a strong association between myopia in children and their exposure to night-time lighting during their first two years[1]. We have been unable to confirm this surprising result, but we find that myopic parents are more likely to employ night-time lighting aids for their children. Moreover, there is an association between myopia in parents and their children[2,3].

We acquired child and parent refraction information as part of a 24-year longitudinal study of visual development in children. These children were research subjects and are not a clinical population. Refractions from 213 children and their parents are included; all children were refracted in the laboratory by non-cycloplegic retinoscopy.

One limitation of Quinn *et al.*'s study is a lack of information about the refractive status of the parents. Parents in our study were either tested in the laboratory or their spectacle prescriptions were used; if they had never worn glasses and could see clearly at a distance, they were classed as non-myopic.

Subjects (100 females and 113 males) ranged in age from 2 to 24 years, with a mean of 11 years. The data were divided into two groups: myopes, with a spherical equivalent refractive error ranging from −9.0 to −0.5 dioptres (mean, −2.50 dioptres), and non-myopes, with a spherical equivalent refractive error more positive than −0.5 dioptres (range, −0.38 to +4.38 dioptres; mean, +0.87 dioptres). Answers to questionnaires on nursery lighting conditions at night were collected from parents over the telephone, using the questions of Quinn *et al.* and a few extra ones. One asked parents to rate their confidence in the reliability of their recall of night-time lighting conditions from years earlier: 98% were confident in their responses.

The prevalence of myopia in our sample of children was not associated with ambient light exposure at night during their first two years, or later in life: 20% of those who slept with night lights before age 2 were myopic — the same incidence as in children who slept in the dark. There were no myopes among the small group who slept with full room illumination. This result was not related to either age of onset (mean, 10.5 years) or the severity of myopia.

Families with two myopic parents, however, reported the use of ambient lighting at night significantly more than those with zero or one myopic parent ($\chi^2 = 7.42$, $P < 0.025$). This could be related either to their own poor visual acuity, necessitating lighting to see the child more easily at night, or to the higher socio-economic level of myopic parents, who use more child-monitoring devices. Myopia in children was associated with parental myopia, as reported previously[2,3]. The proportion of myopic children with two myopic parents was significantly greater than the proportion of myopic children with zero or one myopic parent ($\chi^2 = 4.42$, $P < 0.05$).

Based on these results, we question whether parents need to be concerned about causing myopia in their children by lighting their nurseries at night.

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Quinn et al. *reply* — In not being able to find the strong association reported by us[1] of childhood myopia with night-time ambient lighting before age 2 years, Zadnik *et al.* and Gwiazda *et al.* ascribe our results to a tendency of myopic parents to illuminate their children's rooms at night. Family studies of myopia typically have difficulty separating environmental from genetic factors, however, as sib–sib correlations for myopia decrease with increasing age difference[2] and within-family refractive similarities decrease with adjustment for the 'classic' environmental factors of education and close work[3]. Thus, shared inter-generational behaviour (such as use of night lighting) cannot be excluded *a priori* as contributing to any familial association for myopia.

There are major differences among the studies. Our subjects were younger (mean age, 8 years) and had a considerably higher myopia prevalence of 28% — itself quite high for a United States population of this young age. Accordingly, early-onset myopes, who ultimately tend to become more severely affected, are overrepresented in our tertiary-care population. Thus, it remains to be determined whether the lack of a daily period of darkness during infancy either accelerates myopia onset or provokes the condition in a subset of children who may be predisposed to a more severe form of the condition.

Neither of the subsequent studies considers possible reporting bias. Our findings received widespread publicity, and parents of myopic children might not accurately report or may even under-report a behaviour they fear could have harmed their children. Misclassification errors may also have been introduced into the later results, from non-cycloplegic childhood refractions in one and from self-reported parental refractions[4] in the other.

Our results[1] and others demonstrating the influence of lighting on ocular development in animals[5] support the notion that disrupting the daily light–dark illumination cycle may affect eye development in children. Rather than offering reassurance to parents at this time, the disparities in the available clinical reports are better directed to guiding the design of future research into the interactions of light, dark and refractive development.

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