Both genetic and environmental factors are implicated in the aetiology of myopia. Historically, near work activity—for example, reading, has been considered the most important environmental risk factor, although two comprehensive genetic studies have recently suggested that additive genetic factors are responsible for over 80% of the variation in refractive error in European populations, leaving only a minor component that could be due to environmental factors to which people are variably exposed. However, Quinn and co-workers have reported an association between night-time light exposure during the first 2 years of childhood and subsequent myopia development (in a US population group attending a university paediatric ophthalmology outpatient clinic) the magnitude of which was so strong that it would be expected to dominate genetic factors in the aetiology of myopia. Interestingly, the disruption of normal circadian lighting rhythms is known to alter refractive development in chickens resulting in eyes with flatter corneas, shallower anterior chambers, and deeper vitreous chambers (with this latter effect possibly being an emmetropisation response to the anterior segment changes). However, these chick studies would predict that continuous light exposure would predispose to hyperopia rather than myopia, and Smith et al have recently found that in rhesus monkeys continuous light exposure does not induce the dramatic changes in refractive development seen in chicks. In addition, three studies (two in the United States, one in Singapore and China) have failed to replicate the findings of Quinn et al in human populations. We tested the strength of the association between night-time lighting and myopia development in a UK sample for the first time.

Background: It has been reported that exposure to artificial lighting at night during the first 2 years of life was very strongly associated with subsequent myopia development.

Methods: The strength of this association was tested in a UK sample for the first time. The study population comprised 122 university students.

Results: Myopia occurred with approximately equal frequency in those who slept with and without light exposure at night. In contrast, two largely genetic factors, parental myopia and race, were both significantly associated with myopia development, as has been found previously.

Conclusion: This study provides further support for the view that night-time light exposure during infancy is not a major risk factor for myopia development in most population groups. In a subset of this cohort for which spectacle prescriptions were available for both parents (49 trios), the heritability of ocular refraction was estimated to be 0.31.

RESULTS
In this UK sample there was no significant association between night-time light exposure and myopia (Table 1A; \( \chi^2 \), p=0.21). This remained the case when subjects in the “night

<table>
<thead>
<tr>
<th>Subject myopic</th>
<th>Darkness</th>
<th>Night light</th>
<th>Room light</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject not myopic</td>
<td>22</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td>Subject myopic</td>
<td>51</td>
<td>23</td>
<td>4</td>
</tr>
</tbody>
</table>

Table 1 Number of subjects with myopia as a function of night-time light exposure during first 2 years of life

In (B) the data from the night light and room light groups are pooled.
lights” and “room lights” groups were pooled, and the data were analysed in the form of a simplified $2 \times 2$ contingency table—specifically, the presence/absence of myopia in subjects who had/had not received light exposure at night during infancy (Table 1B; Fisher’s, $p=0.12$). (Since the $\chi^2$ test sometimes performs poorly when cells contain values $<5$ the latter test is likely to be more reliable than the former.) When the data collected by Quinn et al were re-analysed in this simplified $2 \times 2$ table manner it retained its high statistical significance ($p<0.0001$). The current study had 95% power to detect an effect of similar magnitude. The current study had 95% power to estimate $\beta$; Fisher’s, $p=0.005$) and in those with at least one myopic parent (Table 1A; Fisher’s, $p=1.0$), but was more common in Asians (Table 3; Fisher’s, $p=0.02$). There was a significant correlation between the refractive error of subjects and their parents (Table 3; Fisher’s, $p=0.002$). This relation provided a heritability estimate for ocular refraction of $0.31$ (95% confidence interval 0.14 to 0.50) for the 49 families for which data were available for both parents.

**DISCUSSION**

In this UK sample there was no evidence of an association between night lights and myopia. A similar conclusion was drawn from studies in the United States by Gwiazda et al and Zadnik et al. However, in subjects from Singapore and China, Saw and co-workers did find a weak association between night lights and myopia that almost reached statistical significance, and in a very recent study, Loman et al found a significant association between myopia and the number of hours of complete darkness to which young adults were currently exposed at night.

The population studied here had a higher prevalence of myopia (64%) and a lower prevalence of hyperopia than the general population (see distribution of offspring mean spheres in Fig 1) suggesting a source of ascertainment bias. As has been noted previously, given that the subjects studied by Quinn et al were aged between 2–16 years, they showed a similar high prevalence of myopia (with 30% of subjects myopic). Although a myopia prevalence of 64% is typical of university students, our study was also likely to have been affected by response bias, with more myopes choosing to participate than non-myopes. Such selection and response biases have the potential to either inflate or mask the effect being investigated. By selecting subjects attending a university paediatric ophthalmology clinic, the population studied by Quinn et al is likely to have suffered from a different source of selection bias, but possibly a similar response bias.

In contrast with the lack of an effect from night lights, this study once again confirms the higher prevalence of myopia in Asians compared to Europeans, and the influence of parental myopia in determining the refractive errors of their children. However, the heritability estimate obtained here (0.31) is much lower than that obtained in two careful twin studies that were carried out recently, both of which used considerably more subjects than were included in the present investigation (n = 506 and 114 twin pairs, respectively). We speculate that several factors might have contributed to this difference. Firstly, our heritability has wide confidence intervals and thus could be a considerable underestimate (and while the linear regression residuals for the calculation do not show a significant deviation from normality, the sample is too small to provide a robust test of this). Secondly, our subjects had a more varied ethnicity, and a higher proportion of high myopes, compared to those examined in the two twin studies. Thirdly, our heritability estimate is subject to a source of bias, since emmetropic parents would be less likely to wear spectacles, and thus would have been excluded from our analysis. Finally, because twin studies make the assumption that “common environment” effects are independent of zygosity, they tend to overestimate heritability in comparison with population studies.

In conclusion, our results suggest that night-time light exposure played a lesser part than genetic factors in the myopia development of this UK student population.

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