lensing[3] (these are clusters of estimated characteristic central surface mass density above 0.4 g cm^{-2} and situated roughly between redshifts 0.25 and 1).

An arc-like image of a galaxy like that observed can be formed subject to the following two conditions. First, the cluster mass distribution, as projected on the sky, must be close to circular. The degree of the deviation is measured by the reduced shear, γ_c, which is defined in ref. 5. The reduced shear is a combination of the asphericity of the cluster gravitational field and of the additional asymmetry which may possibly be introduced by other massive objects, say other clusters, projected on the sky close to the cluster in question. It also depends on the redshifts of both the lens and the source. The reduced shear determines the angular size of a cone of astroid cross-section within which a point source must reside to produce four or five images. The solid angle inside the astroid is[5]

$$S_5 = \frac{3\pi}{2}\gamma_c^2\,\theta_t^2,$$

where θ_t is a characteristic deflection angle by the cluster. It can be roughly estimated by replacing the cluster by an isothermal sphere of the same line of sight velocity dispersion, v_l,

$$\theta_t \approx 4\pi\,\frac{v_l^2}{c^2}\,\frac{d_{ls}}{d_{os}}$$

where d_{ls} and d_{os} are the lens-source and observer-source angular diameter distances. The clusters relevant for lensing have unusually high velocity dispersions[4]. For the order of magnitude estimates, $v_l \sim 1{,}500$ km s^{-1} and $d_{ls}/d_{os} \sim 0.5$, θ_t is $\sim 30''$.

The second condition for producing large arcs is that the lensed galaxy covers a significant portion of the astroid, say a quarter, for the arcs found by Lynds and Petrosian. The sizes of observable parts and the number density of the relevant, high redshift galaxies[6,7] ($z \sim 0.5-2$ can be used to give a rough estimate of $\sim 2''$ for the angular radii of the observable parts of charateristic (L^*-) galaxies and ~ 100 arc min^{-2} for their number density.

For the astroid to be of a size comparable to that of a galaxy, the shear parameter must be $\gamma_c \leq 0.03$. The distributions of this shear for clusters are not known, but very rich clusters should be well virialized and extremely asymmetric mass distributions are unlikely to be characteristic. The statistics of multiply ranged quasers[3] place the characteristic values for the shear to be ~ 0.5. An estimate on the basis of a uniform distribution between 0 and 1 yields probability $p_\gamma \sim 0.03$ for a shear suitable for producing arcs.

The probability that a galaxy will cover a large part of the astroid is $p_g \sim 0.3$, so that out of the 2,000 clusters about 20 are expected to produce luminous arcs similar to those found by Lynds and Petrosian.

Such a large number may be inconsistent with the absence of such arcs in cluster samples taken by Gunn and Schneider, as noted by Paczyński. It must, however, be clear that the present number is only an order of magnitude estimate. The main difficulty in achieving a much more precise estimate is that the observational data on mass distributions in clusters of galaxies is not available for a part of clusters in the known complete samples. But this estimate shows that we could have anticipated the discovery of the giant luminous arcs, that Paczyński's proposition is completely realistic, and that it may be possible to find some other arcs around the richest of the high-redshift clusters.

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Fitness of insecticide resistance

SIR—Clarke and McKenzie[1] and McKenzie *et al.*[2] have convincingly shown that continued use of diazinon against sheep blowfly, 14 years after resistance to this organophosphate was first detected, has gone on to select modifiers which minimize fluctuating asymmetry and deleterious effects of the resistance allele. These same authors have also shown that blowflies resistant to dieldrin, a cyclodiene, are lacking in modifiers despite high asymmetry and large fitness disadvantages in the absence of dieldrin. But their explanation for the absence of dieldrin-resistance modifiers — that use of dieldrin after resistance evolved was too limited — may be insufficient, for two reasons.

The first is that cyclodiene usage was more extensive than they suggest: γHCH (a cyclodiene-type insecticide known to rapidly select dieldrin-resistant blowflies[3]) had been widely used against sheep ectoparasites for several years prior to the introduction of dieldrin[4] and ought to have selected modifiers had they been available. The second reason is that effective modifiers of dieldrin resistance may not exist at all. This opinion stems from the recently discovered biochemical mechanism of dieldrin resistance in cockroaches[5,6] and work by myself on the fitness and behaviour of dieldrin-resistant mosquitoes. Despite the diversity of test insects used, the conclusions are probably mutually applicable because the dieldrin-resistance mechanism seems to be the same in all species (characteristics held in common include semidominance in heterozygotes, and parallel cross-resistance spectra and resistance factors)[7].

The normal toxic action of dieldrin is to bind to receptors on chloride channels of nerves and thereby prevent entry of chloride ions and block transmission of inhibitory impulses[5]. Dieldrin is ineffective against resistant cockroaches because their receptors have become insensitive to this insecticide[6]. The fitness disadvantage of resistance in a cyclodiene-free environment is behavioural: resistant insects are less active and less responsive to stimuli. Evidence for this comes from observations on four species of mosquito from three continents: in every species homozygotes for resistance spent less time searching for hosts and were less responsive to predator movement than heterozygotes and susceptibles (fitness effects were always recessive and never modified by genetic background); in addition female homozygotes were less responsive to oviposition stimuli and male homozygotes had limited mating success.

My conclusion is that dieldrin resistance raises the response threshold of homozygotes to a range of unrelated stimuli; perhaps the mutation to the dieldrin receptor[6] has increased the permeability of chloride channels, causing hyperinhibition of the nervous system. I suspect that such a far reaching deleterious pleiotropic effect would not easily be neutralized, and that capable modifiers have not yet evolved despite long periods of cyclodiene selection (three of my four species of mosquito were colonized 20 years after cyclodienes were first used).

Insecticide resistance genes give such an enormous advantage in the presence of insecticide that what would otherwise be quite major disadvantages might count for little. While not disputing the importance of time in the selection of modifiers, their existence cannot always be assumed. Before Clarke and McKenzie can pull dieldrin resistance into their argument, they first need to show that modifiers have been selected in areas where cyclodienes are still used and where dieldrin-resistant blowfly are present at high frequency. The Lismore area of New South Wales might be a good place to look[8].

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