28 March 1933.

Major L. Darwin, F.C.D.,
Cripps Corner,
Forest Row,
Sussex.

Dear Major Darwin:

On the question of the heritability of different grades of defect the current belief in the lower heritability of the more extreme defects seems to be based partly on a very simple fallacy. A man who is interested in an extreme and rare form of idiocy finds among the parents of his idiots a very small proportion who are themselves idiots, so small a proportion, indeed, that he probably finds none in the sample of cases examined. He, therefore, does not encounter any striking positive evidence of heredity and when his work is summarised with that of 20 others there will be found several strong and apparently well substantiated cases with respect to the inheritance of mild mental defect but no signs of conviction among those who speak of the severer forms. The fallacy arises through a failure to realise that though one must particularise the type and degree of defect in starting point of the pedigree, yet all kinds of defect must be recorded in the relatives, but

actually very few medical investigators realise that there is any necessity to draw a distinction between the starting point and his relatives. Are they not all relatives of one another?

You have, I think, often noticed the influence on statistics collected in this way of the fact that most low grade types cannot or do not breed, and this naturally most affects the apparent inheritance of types of defect most frequently related to the non-breeding types. Less generaly one may perhaps look at some cases in this way--suppose a single recessive mutation causes idiocy, as in the amaurotic blind sort, these never breed. The supply of idiots must be maintained by carriers only and the supply of these by mutations, supposing the carriers to be entirely normal and the defect entirely recessive. In these circumstances I cannot see that the gene can avoid being diffused with equal frequency into all localities and all social classes. Such defects will presumably be very rare, perhaps one in a million, since mutation rates higher than this are probably rare.

As soon as the situation is altered so as to allow the recessives to breed, as in the case of a similar mutation producing a milder defect, the same mutation rate may maintain defectives at a thousand-fold greater frequency, the limit being now set by a counter selection against the defect, which may be very small, or might even vanish. This greater

frequency being due, ultimatley, entirely to reproduction by the defectives themselves, will be strongly influenced by local and class circumstances, so that an investigator working in a bad patch may obtain irrefutable evidence of inheritance, although, had he taken similar cases from a different class or from a district where the defect was rare, he would have got no convincing evidence at all.

As to the classification endogenous versus exogenous, etc., the Chairman and most of our witnesses have frequently used a three-class clasification, though not always quite consistently. Some witnesses have favoured the belief that in a large proportion of cases both hereditary and environmental factors were responsible, with the underlying idea that, if this were so, we should all agree that the environmental ones were the only ones to remedy.

Yours sincerely,