for non-verbal mental ability. This is obtained from the general expression, $h^2 = (r_{MZ} - r_{DZ})/(r_{MZ} - r_{DZ2})$, where $r_{MZ}$ and $r_{DZ}$ are the theoretical correlations for identical and fraternal twins, respectively; essentially the derivation of Jensen[18]. Taking $r_{DZ} = 0.5$, of course, assumes no assortative mating (the presence of which would increase $h^2$) and no dominance or epistasis (which would decrease $h^2$). The magnitude of the influences is difficult to assess with any confidence from existing empirical data, but it is usually assumed that they are very small. Nonetheless, adopting Jensen’s18 estimate of $r_{DZ} = 0.55$ under assortative mating, and thus the maximum feasible correction to the above $h^2$, only increases it to 0.373. This $h^2$ is the consequence of introducing the usually unsuspected ‘treatment effects’ into the data and is at best an upper limit. It has been demonstrated that taking only minimal account of such effects in the data of previous studies reduces the reported $h^2$ estimates to values not significantly different from zero4,6.

In conclusion, our nationally representative sample of twins of uniform age and test subjection, offer supportive evidence for zero or low upper limit heritabilities of mental test performance. Such estimates are considerably lower than the value of 0.8 normally quoted in psychometric research18 and which has permeated the psychological literature. Although it is necessary to be clear about the often-inflated significance of heritability estimates4, the present results are perhaps further indicative of the stringency of sampling required in such studies9.

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Correction to Fisher’s correlations between relatives and environmental effects

Fisher1 has given a novel much used model of assortative mating, and using formulae obtained for it he analysed data on human height, span and forearm. In his model, phenotypic variance is regarded as the sum of three variances, additive, dominance and environmental; and genetic variance is the sum of the additive and dominance components. This was his first contribution to genetics and it would seem that he did not appreciate fully the implications of his model. His formulae for parent-child correlation and sibling correlation, in the presence of assortative mating, are not correct for his model. Moreover, the correct formulae for these correlations for his model suggest a different interpretation when the value for (genetic/phenotypic variance) is greater than 1. I present here a summary of criticisms of Fisher’s formulae; details of which, with derivation of correct formulae will be presented elsewhere.

The additive variance is obtained by fitting a linear regression on the gene content of the genotype; dominance variance is the residual genetic variance, that is, genetic, additive variance. Fisher found that the value of his symbol $c_1$ defined as (genetic variance/phenotypic variance) was greater than 1 for height and span and concluded that this "gives no support to the supposition that there is any cause of variance in these growth features other than genetic differences". His method of partitioning the phenotypic variance into additive, dominance and other components has recently been used for IQ and estimates of its heritability (additive variance/phenotypic variance) have been the subject of some controversy.

Fisher realised that assortative mating will cause association between genotypes ("phases of factors" in Fisher’s terminology). He devised a complicated method to take account of it and found that this association increases the genetic variance of the population. The increase in genetic variance is attributable entirely to an increase in the additive variance of the population8.

Fisher did not consider what effect assortative mating has on the phenotypes of a parent and his progeny. He assumed that the additive deviations are the only cause of resemblance between parent and child as he says, "Hence, since there is no association except of z (additive values) between parent and child, the parental correlation coefficient is

$$c_1c_2 \frac{1 - \mu}{2}$$

Here $\mu$ is the phenotypic correlation between husband and wife and $c_2$ is the total additive variance/phenotypic variance; $c_1$ has already been defined.

Wright1 on the other hand says that "Assortative mating introduces a correlation between dominance deviations of parents and offspring and between dominance deviations of either and additive deviations of the other". Fisher did not investigate such correlations and took no account of them. It can be shown that in Fisher’s model of assortative mating there is a small correlation between the additive and dominance deviations of parent and child. Thus, the assumption on which Fisher obtained his formula for parent-child correlation is not correct.

To obtain his formula for sib correlation, Fisher discarded his model of assortative mating and reverted to random mating. He said, "The variance of a sibship, for example, depends, apart from environment, only on the number of factors in which the parents are heterozygous, and since the proportion of heterozygotes is only diminished by a quantity of the second order, the mean variance of the sibship need not be taken for our purposes to have the value appropriate to random mating..."

It is true that the variance of single sibship will be the same under random mating and assortative mating. The proportions of different types of matings will, however, be different under the two systems. As the mean sibship variance is $\Sigma(\text{frequency \times sibship variance})$, it will not be the same under the two systems.

Moreover, Fisher adds the whole of the increase in variance, $A^2/(1-A)$ where A is the genetic correlation between husband and wife and $a^2$ is the additive variance appropriate to random mating, to sib covariance and no part of the increase to sib variance. This is difficult to justify assuming Mendelian segregation on which his model is based.

Consider now his two formulae together:

Parent-child correlation = $\frac{1}{2}c_1c_2(1-\mu)$

Sib correlation = $\frac{1}{4}c_1(1 + c_2 + 2c_2A)$

In the presence of dominance, the sib correlation will be greater than the parent-child correlation, therefore

$4c_1(1 + c_2 + 2c_2A) > 4c_1(1 + \mu)$

which holds only if $(1 - c_2)/2c_2\mu > 1 - c_1c_2$. Fisher’s model of
assortative mating will hold for those values of $c_1, c_2$ and $\mu$ for which
this inequality holds. It can be verified that it holds only for
certain combinations of values of these parameters. For example,
if $\mu = 0.5$, $c_1 = 0.8$, $c_2 = 0.8$, we get $0.25 > 0.36$,
which is not true.

These considerations lead to the conclusion that Fisher's
formulae for parent-child and sib correlations are not correct
for his model. It is, however, possible to obtain formulae using
Fisher's model which do not suffer from these deficiencies.
These formulae, in Fisher's notation, are

Parent-child
correlation $= c_1 c_2 A(1 - A)^2 + c_1 c_2 (1 - c_2) A(1 - A)$

Sib correlation $= c_1 c_2 A(1 - A)^2 + c_1 c_2 (1 - c_2)$

In both, the first term represents the contribution of additive
deviations to correlation and the second term the contribution
of dominance deviations. Note that the contribution of the
dominance deviations to parent-child correlation is smaller
than their contribution to the sib correlation since $A(1 - A) < 1/4$.
This is another reason why the concept of heritability defined as
additive variance/phenotypic variance cannot be applied to
human populations mating assortatively.

If the coefficient of assortative mating $\mu$ is known, the value
of $c_1$ and $c_2$ can be obtained from the parent-child and the sib
correlation formulae. Effectively, the difference between the two
correlations gives a fraction of the dominance variance. The
formulae given here and those obtained by Fisher, however, are
for genetic correlations which are unknown and phenotypic
correlations have been used instead. If the environments of sibs
are more alike than those of parent and child, the difference
between phenotypic correlations will also contain a fraction of
the environmental variance. When the phenotypic correlational
difference is too large to be explained by the amount of
dominance variance in the population, the value of $c_1$ will
come out to be greater than 1. Thus, such a value, far from
being evidence for no environmental effects, indicates their
presence. On the other hand, a value less than 1 for $c_2$ does not,
of itself, exclude environmental effects, which could mean that
the contributions of dominance deviations, as well as of
environmental effects, are small.

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Effects of artificial selection on reproductive fitness in Drosophila

A common feature of many selection experiments, when
polygenically determined traits are involved, is a reduction in
the reproductive fitness of the selected strains. Experimental
selection for differences in the numbers of abdominal
bristles and sternopleural chaetae in Drosophila melanogaster produced sterility and a reduction in fertility in the selected strains1,2. Later3 found that the "competitiveness" (matings propensity, female fecundity and survival ability) had fallen sharply in strains of D. melanogaster subjected to experimental selection for differences in scutellar bristle number. Selection in Tribolium for changes

in developmental rates produced alterations in productivity, body size and viability of the flour beetles4. The reproductive fitness of chickens under selection for increased shank length was reduced steadily throughout the selection process5.

Lerner6 proposed that an increase in homozygosity is directly
responsible for the reduction in reproductive fitness found in selected strains. He submitted evidence suggesting that heterozygosity is of prime importance in producing a balanced, highly fit natural population. This preselection phenotype is the outcome of numerous generations on which natural selection has operated to produce maximum reproductive fitness. As experimental selection
proceeds, the balanced phenotype and genotype favoured by
natural selection is disrupted. The selection process favours homozygosity for those genes which influence
the selected trait. Homozygosity is also increased by incidental
inbreeding, a consequence of the experimental selection
procedure. On relaxation of the experimental selection
pressures, natural selection is free to restore a balanced
phenotype. The most heterozygous individuals are again
favoured, causing the "gains" made in the experimental
selection scheme to be lost. Lerner has called this pheno-
menon genetic homoestasis and defined it as "the property
of the population to equilibrate its genetic composition and
to resist sudden changes6.

Selection for divergent geotactic and phototactic maze
behaviour in Drosophila is also thought to decrease repro-
ductive fitness. The evidence is indirect and based on the
observation that suspension of selection pressures in strains
of D. pseudoobscura resulted in the reversion of the po-
titive and negative geotactic and phototactic strains towards
their original neutral maze response7. No decrease in pro-
ductivity has, however, been found in divergent geotactic
strains of D. melanogaster during 10 generations of selec-
tion8. This present study was undertaken to investigate
directly the reproductive fitness of strains of D. melanogaster
selected for positive and negative geotactic and photo-
tactic maze behaviour.

Fifteen-unit classification mazes9,10 were used to select
experimentally for divergent geotactic and phototactic
behaviour. Maze scores may vary from 1 to 16 on both
geotactic and phototactic mazes. A score of 1 represents
the most negative maze response possible and the maximum
positive maze response results in a score of 16. A cage
population of D. melanogaster which was established by
hybridising large samples of 20 different wild-type strains,
was used to begin- selection for opposite geotactic and
phototactic behaviour. Divergent behaviour selection was
carried out by collecting approximately 300 females and 300
males which were run separately through either the geo-
mazes or photo-mazes each generation. Sixty pairs of the
most positive and negative individuals were selected as
parents to initiate subsequent generations for both types of
behaviour. Non-virgin females were run through the
mazes so females selected as parents were des.makedirsed by
being placed at -10°C for 10 min11. The geo-strains and
photo-strains thus produced were highly divergent having
undergone opposite selection pressures for over 40 genera-
tions. The degree of divergence is reflected in the mean
geonotactic and phototactic scores of the four strains. The
geotactic generations selected for opposite geotactic act
phototactic behaviour. The mean score of the geonegative
strain was 1.85 and the mean score of the geopositive strain
was 14.90. The photonegative strain had a mean phototactic score of 2.01 while the mean score of the photopositive strain was 12.7. The unselected control
strain was approximately neutral for both geotactic and
phototactic behaviour throughout the selection process.

The following procedure was followed to estimate the
egg-to-adult survival, egg hatchability, larva-to-adult
survival and female fecundity of the strains under study.
Virgin females and males were collected over a 48-h