Evaluating genetic models of cognitive evolution and behaviour

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Abstract:
Cognitive evolution can be studied at several different levels, ranging from complex societies of interdependent persons to the DNA molecules coding for enzymes that synthesize neurotransmitter molecules. Genetic models of cognitive evolution can be fairly evaluated only if they involve one or two genetic loci, maybe three loci if a massive investment of resources is made. If a simple genetic model is seriously proposed, it ought to be tested by genetic linkage analysis so that future theorizing can be guided and constrained by facts. For more complex behavioural characteristics based on large numbers of genes and intricate interrelations with the environment, genetic analysis and genetic theories are not likely to yield conclusive results. Instead, studying individual differences in the brain and neural correlates of cognitive processes will likely provide more rapid progress toward a deeper understanding of evolution.
Keywords: Corpus callosum; Hippocampus; Epistasis; Linkage analysis; Quantitative trait locus (QTL); Statistical power

Article:

1. Introduction

The evolution of cognition over many generations can occur via environmentally transmitted changes as well as changes in frequencies of genes (Cavalli-Sforza and Feldman, 1978). Without in any way diminishing the importance of cultural factors, the present paper addresses mainly the question of genetic effects. Genes could conceivably influence the progress of long-term cognitive evolution by modifying the ability of the nervous system to sense stimuli, detect associations, store memories, and organize actions. This assertion does not imply that genetic change must therefore precede behavioural change. On the contrary, there are good reasons to believe that behavioural plasticity may give birth to new habits which in turn create conditions favourable for proliferation of particular alleles (e.g. Bateson, 1988; Johnston and Gottlieb, 1990; Plotkin, 1988). Neither is this assertion a claim that genes code for nervous system structure. Genes code for protein structure and act within cells, whereas brain structure emerges through interactions among cells and different kinds of tissues. Suitable genes may be essential for development, but they do not specify the end result of development.

For human cognitive evolution there is not one documented example of a mental characteristic that has changed historically because of genetic changes, although environmentally based trends are well known (e.g. Flynn, 1987). There is no convincing evidence that differences in culture and psychology among nations or tribes has a corresponding genetic cause (e.g. Roubertoux and Capron, 1990; Zuckerman, 1990), although environmental factors are clearly important (Scan and Weinberg, 1976). Faced with this impasse, investigators often adopt one of two approaches to studying genetic changes in evolution.

Selective breeding of laboratory animals for high or low expression of specific behaviours has resulted in gradual but substantial changes in geotaxis in fruit flies (Ricker and Hirsch, 1988a), strength of paw preference (Collins, 1985) and agonistic behaviour (Cairns et al., 1990; van Oortmerssen and Bakker, 1981) in mice, and shock avoidance learning in rats (Brush, 1991), to name a few of the many experiments of this nature. Selection can lead to divergence of lines because of a difference at a single locus (e.g. Korpi et al., 1993) but it seems

likely that most studies involve the combined effects of several different genes. Hence, the kinds of genetic changes effected by these selection experiments remain obscure. These instances of behavioural evolution in microcosm provide glimpses of the kinds of change that could possibly result from extreme differential reproduction based on a single characteristic of an organism. All these selection studies are of the ' what if' variety. Lacking knowledge of the number and properties of relevant polymorphic genes or a thorough quantitative genetic assessment of several crosses, we have no way to predict how widely separated the high and low lines will become, what other characteristics will diverge because of a close relation with the selected behaviour, or whether the lines will converge after selection pressure ceases (see Ricker and Hirsch, 1988b). Because the progress of a selection experiment depends decisively on the nature of the foundation population and environmental conditions obtaining during the study, the results lack generality. Fascinating as the results of these studies of laboratory animals may be, they tell us nothing about the trajectory of human cognitive history. As recognized by Darwin, experiments with domestic animals help to convince us of the plausibility of cognitive evolution. Validating this kind of model is generally not an issue.

The other approach is to devise a model of the genetic structure of a population, the nature of genetic influence on behaviour, and the consequences of the behaviour for reproductive fitness. This hypothesis then yields a prediction of the likely course of evolution by natural selection. Models that posit rigid control of complex behaviours such as altruism, homosexuality, and aggressiveness by one or two genetic loci abound in Wilsonian sociobiology in particular (see critique by Kitcher, 1985). Single locus genetic models of hand preference or cerebral lateralization are also current (McManus and Bryden, 1992). A two locus model of niche construction is presented in this volume (Odling-Smee, 1995).

Experimental studies of evolution using artificial selection provide no comfort for models that posit only one or two relevant loci. Nevertheless, simpler genetic models have achieved some importance in recent theorizing about the evolution of behaviour, and therefore this paper primarily addresses questions about simple models.

2. Two kinds of simple genetic models

If a model begins with genetic variation at a specific locus in the population and predicts that one allele will gain ascendancy at the expense of another, there should no longer be any noteworthy polymorphism in the real world and all individuals should have the same genotype at the locus in question. A model of this kind is presently beyond empirical testing (unless targeted mutations can be used — see Capecchi, 1994; Monastersky, 1993) and is no more persuasive than a ‘just so’ story about how the bobcat lost its tail. Two general challenges have been posed to this kind of theorizing. The `adaptationist program' has been criticized as a circular argument crafted to prove a preconceived conclusion and justify the status quo in human society as biologically inevitable or natural (Gould and Lewontin, 1979; Rose et al., 1984). Equally taxing of the imagination is the implication that evolution has ceased because the best gene won the contest and society is now in a final, stable genetic state, at least until the unpredictable appearance of a novel mutation which will allow another spurt of progress in the ability to think. Because most species still possess considerable genetic variation, there are abundant opportunities for contemporary changes in gene frequencies. Although human fertility has declined in many countries in the past few decades, there are still large differences between individuals in the number of viable offspring (e.g. Grindstaff, 1992), which allows for dynamic changes in gene frequencies in humans as well. Whether such change is indeed happening now is a question to be answered by data. In any event, explanation of how some crucial genetic loci became invariable in the population cannot be evaluated in the same way as those that address individual differences.

If, on the other hand, the model expects genetic polymorphism at the present time, it can be readily evaluated. The best kind of evidence shows that a putative gene affecting behaviour is located close to a known 'marker' gene at a place on a specific chromosome. If the marker and the measured behaviour tend to be transmitted together from one generation to the next, the newly discovered gene is said to be linked to the established locus. The closer the two genetic loci are to each other on the chromosome, the more likely they are to remained joined. Only a decade ago the challenge of detecting genetic linkage was daunting indeed because the locations

of so few marker genes were known and the alternative forms (alleles) of many of these were too rare to be useful in analysing human pedigrees.
The advent of new molecular genetic techniques has made hundreds of new markers on all chromosomes available for research. 'The best of these for linkage analysis involve variations in portions of the DNA molecule (introns) that do not code for functional protein and instead are usually neutral with respect to reproductive fitness and viability of the organism. As of 1993, over 1500 such markers were known in mice (Copeland et al., 1993) and several thousand exist in humans (Cuticchia et al., 1993). Molecular genetic methods are now sufficiently powerful that hundreds of new markers can quickly be identified and used for linkage studies even in species that heretofore had not been studied extensively by geneticists (Andersson et al., 1994).

At last it has become feasible to settle some long-standing disputes about the number, location, and functions of genes that are alleged to influence a wide range of behaviours in human and nonhuman animals. I contend that the time has arrived for advocates of major gene effects on complex behaviours, provided they wish to be taken seriously any longer, to show us some convincing evidence. If people believe a major gene determines who is or is not an altruist, let them show us where it is on which chromosome. Otherwise, we are entitled to regard their writings as sophomoric mind games. The recent availability of thousands of markers for linkage analysis (CHLC, 1994) should herald a hiatus for unfettered speculation and a new period when real genetic knowledge becomes the firm foundation for future theories of cognitive evolution.

3. Learning lessons from nature

No doubt some scientists will be trepedatious about efforts to map genes affecting altruism, religious conservatism, or sexual preference. After all, this might lend dramatic confirmation to a noxious brand of sociobiology. This danger is real, but with the new technology the dagger could strike either side in the dispute, unlike the previous situation where major gene models were virtually impervious to refutation. With so many markers available in most regions of every chromosome, there is now a real possibility of proving that a behavioural or mental characteristic is not influenced by a major gene. Linkage analysis compares (a) the probability of observing the actual pattern of inheritance when a putative gene and a marker are a certain distance apart on the chromosome with (b) the probability that they are not linked at all. If the evidence strongly implies they are not linked, this establishes that for a segment of chromosome on either side of the marker, there is no major gene influencing the behaviour in question. For example, analysis of DNA markers in several Scottish families where there was a high frequency of schizophrenia (St. Clair et al., 1989) established that there was not a gene in a region of chromosome 5 that had earlier been identified as the site of a gene leading to schizophrenia; the probability that the marker was not linked to a gene related to schizophrenia was more than 10^{10} times higher than the probability they were closely linked. If there are enough markers, it becomes possible to prove that schizophrenia, for example, is not a problem caused by one or two major genes located anywhere on the chromosomes. A few years ago a bold claim was made on the basis of linkage analysis that manic-depressive psychosis among the Amish in Pennsylvania, USA, is caused by a gene on human chromosome 11 (Egeland et al., 1987). One commentator remarked triumphantly that "for the first time psychiatry has entered the realm of molecular biology." (Kolata, 1987). However, further data on the same extended Amish family revised the linkage estimate and led to retraction of the claim (Kelsoe et al., 1989; see also Baron et al., 1993). The search for linkage in the same pedigree continued with a large array of 250 markers, and compelling evidence was found that no such gene for manic-depressive psychosis was located in vast regions of the human genome (Ginns et al., 1992). Whether further searching will be futile is difficult to surmise. There is far less to fear from genuine knowledge of biology than from unfalsifiable assertions of rigid genetic determinism made by famous personages.

In many instances the discovery of a major gene effect and elucidation of the mechanisms by which the gene influences development have provided little comfort to deterministic ideology. Experience has shown repeatedly that the imagination of nature is far richer than our own. When a major gene has been identified that influences a behaviour, the pathway between the gene's primary action and an effect of interest to psychologists sometimes proves to be rather indirect. For instance, the 'brindled' mutation in mice *(Mo^{br})*, which is located on

the X chromosome, results in males which have unusually low motor activity, dilute pigmentation, and curly whiskers. Does this constellation of effects (pleiotropic gene action) signal some profound connection between brain mechanisms governing exploratory behaviour and growth on the face? Should we then expect men with kinky beards to be lethargic introverts? Biochemical studies of the brindled mice uncovered a surprising source for the correlated characteristics (Hunt, 1974). The primary defect occurs in the intestine, where there is inadequate transport of dietary copper into the bloodstream. Consequently, the activity of all copper-dependent enzymes in cells far from the gut is reduced (see Fig. 1). One of these is dopamine-β-hydroxylase which converts the neurotransmitter dopamine to norepinephrine, a molecule associated with motor activity. Another is lysyl oxidase that aids the synthesis of long chains of molecules in body hairs. Thus, the source of both defects is in the gut, not the brain. Similarly, the primary enzymatic defect in human phenylketonuria (PKU), which when untreated results in severe mental deficiency, is in the liver, not the brain (Woo, 1991). Simply because a genetic mutation can alter behaviour or cognition does not tell us that the gene acts in the nervous system or that its effects are inevitable. For both the brindled mouse and the PKU child, symptoms can be ameliorated to a large extent by controlling the diet. Indeed, a genetic mutation often renders an individual more sensitive to variation in the environment, rather than confining ontogeny to a deep rut.

Genetic defects often become known because of a dramatic alteration of a prominent brain structure or overt behaviour, yet further investigation reveals the alteration is a secondary consequence of a more subtle disorder of development. Consider hereditary absence of the corpus callosum (CC), a bundle of nerve fibres that normally connects the two cerebral hemispheres (Lassonde and Jeeves, 1994). When first reported (Keeler, 1933), it was believed to be a single gene effect, but subsequent studies proved several genes were involved (Livy and Wahlsten, 1991; Wahlsten and Schalomon, 1994). Observations of the growth of axons in the embryo brain demonstrated that the CC axons are themselves quite normal but they cannot traverse the fissure between the hemispheres because a suitable bridge is missing (Ozaki and Wahlsten, 1993). That bridge is another commissure which connects the two halves of the hippocampus, and the hippocampal commissure usually forms well before the CC axons arrive at the bridgehead (Livy, 1994). Thus, although the defect is generally known as "callosal agenesis" (Lassonde and Jeeves, 1994), absent CC is secondary to an anatomical defect in formation of the hippocampal commissure.

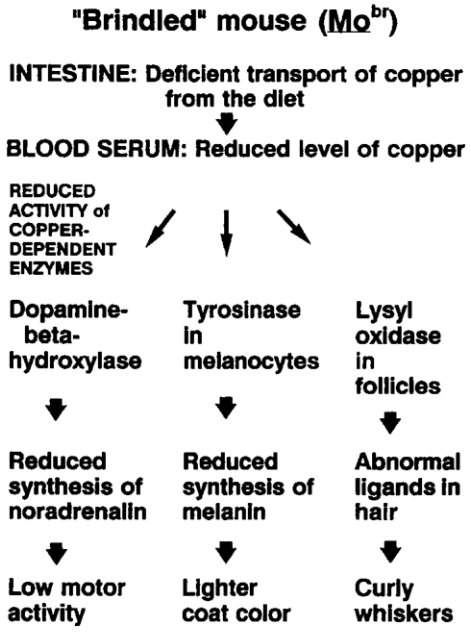

Fig. 1. Pathways by which the 'brindled' mutation in mice (Mo^{br}) affects motor activity, pigmentation, and whiskers. Based on information from Hunt (1974).

These examples point to the necessity of a thorough physiological and developmental study of any hereditary defect that may impact on cognition. Effects on cognition may be highly indirect and secondary to some other effect. Indeed, a novel feature of the brain or behaviour might itself emerge in evolution not because it conferred adaptive advantage on the individual organism but because it was a 'side effect' of some other adaptive change elsewhere in the body. The physiological integration of the organism means that a single gene

can have many diverse consequences, some of which may be very useful while others are neutral or even disadvantageous for survival and reproduction. One of the clear lessons from selective breeding studies is that the experimenter can carefully choose which characteristic will be enhanced or attentuated in the population, yet many other characteristics will change according to their physiological connections with the criterion measure. So it happens that selecting hens to lay more eggs results in smaller eggs. This is one good reason why models of cognitive evolution that consider only one or two supposedly direct effects of changing gene frequencies are not to be taken very seriously. A better approach is to investigate the actual genetic variation in living organisms that is relevant to cognition and be guided in theorizing by nature itself.

4. Simple versus complex genetic effects

In searching for new genetic loci which influence cognition, there is always the gruesome possibility that there are no genes with major effects awaiting discovery and that instead there are dozens of genes with minuscule and obscure effects. Given the availability of thousands of new markers, perhaps the best way to evaluate these alternatives is to proceed with linkage analysis. If a major gene lurks in the labyrinth of DNA, linkage testing should be able to locate it. The quarry in this hunt is the quantitative trait locus (QTL) which presumably modifies the average measure of a behaviour but does not cause a qualitative shift in the distribution of measures (see Hill, 1975; Lander and Schork, 1994; Smith, 1975; Tanksley, 1993).

As useful as this approach may seem in theory, it has profound limitations in practice. The smaller the effect of a QTL gene on a behaviour and the further it is from a marker gene, the more animals must be tested in order to yield a reasonably good chance of detecting its presence. The more QTL genes that are relevant for a behaviour, the smaller will be the average effect of each one and the more likely its effects will pass unseen amidst the welter of more numerous and noteworthy effects, including those of the environment. Especially when working with birds or mammals, the expense and logistics of conducting a large linkage study limit the number of QTL genes that can readily be detected to two or three loci. Statistical difficulties in addition to power and sample size also confront those hoping to document the roles of more than two or three genetic loci acting on the same characteristic (Neumann and Collins, 1991).

Suppose we begin our search with two inbred strains of laboratory mice, each of which is genetically uniform, such that the variance within a strain (σ_N^2) is entirely nongenetic. Taking the midpoint between the strains as 0 and assigning the deviation of each strain mean from 0 the values + g and — g, the genetic variance for the two strains (σ_G^2) is g^2. The magnitude of the strain difference can also be expressed relative to within-strain variation as effect size $\delta_0 = 2g/\sigma_N$.

Now suppose the strains are crossed to produce an F_1 hybrid and then the F_1 hybrids are crossed to yield F_2 hybrids with plenty of genetic variation. Each F_2 mouse is given a test of behaviour and then its DNA is extracted for testing. For a marker locus *(M),* the F_2 sample will include mice with genotypes *MM, Mm,* and *mm.* Comparing behavioural test scores for *MM* and *mm* mice, their means should be significantly different if the *M* locus is close to a QTL which affects the behaviour. The closer *M* and the QTL are on the chromosome, the smaller will be the value of Θ, the probability they will recombine during formation of egg or sperm, and the larger will be 1 — Θ, the probability they remain joined and are transmitted together to the F_2 mouse. If there are in fact L loci and the locus in question has an effect that is approximately the average for these loci, then the difference between the two homozygotes for the QTL should be about g/L.

This model leads to a derivation of the expected effect size for the difference between marker genotypes *MM* and *mm* in terms of δ_0 (size of the original strain difference), Θ (recombination probability) and L (number of relevant QTLs). This value of effect size can then be inserted into a simple formula to determine the necessary sample size *(n)* per genotype when the probability of a Type I error of inference is a and the desired power of the test is 1 — β(see Wahlsten, 1991). This simplified approach to finding sample size to confer adequate power deliberately ignores results for the heterozygote *Mm* because no hypothesis is made a priori about the mode of inheritance and intermediate inheritance makes the same numerical prediction as the null hypothesis that the genes are not linked. The result derived from the simple model is a large equation given elsewhere (Wahlsten,

1995). If the original strain difference is $\delta_0 = 1.0$ standard deviation and power is to be 90%, the necessary sample sizes when recombination probability is 0.1 are shown in Table 1 in relation to the number of relevant QTLs. The required *n* is approximately a function of L^2, and the total number of animals required for the experiment is 4n. Because of continuing dispute about the acceptable level of Type I error (α) in a QTL linkage study, estimates of sample size for several choices of this parameter are given. Lander and Schork (1994) recommend α = 0.0001, two-tailed, for a study of mice.

Table 1
Total number of animals (N) that must be tested to yield statistical power of 90% for detecting linkage of a marker gene with a quantitative trait locus (QTL) when four values of Type I error (α) are used with a two-tailed test [a]

L	Θ	δ	$\alpha = 0.05$	$\alpha = 0.01$	$\alpha = 0.001$	$\alpha = 0.0001$
1	0.1	0.783	146	203	282	350
2	0.1	0.392	556	784	1098	1402
3	0.1	0.262	1229	1737	2437	3116
4	0.1	0.197	2166	3063	4200	5500

[a] These results assume effect size $\delta_0 = 1.0$ for the inbred parent strains. The formula based on Wahlsten (1991) is used to estimate the number of mice needed per group (n), and this figure is multiplied by the number of genotypic groups (4) to obtain the total sample size required ($N = 4n$) in the F_2 hybrid cross.
Symbols in the table are defined as follows: L = number of relevant QTLs; Θ = probability of recombination between a marker and a QTL; δ = effect size for mean difference between genotypes *MM* and *mm*..

The expense of such a study is related to costs of breeding so many mice, doing all the behavioural testing, and screening over 100 genetic markers with biochemical methods. Few laboratories in the world would have adequate financial resources to assess over 500 mice in such a manner, which effectively limits the QTL method to finding only two or three major gene effects. For loci having smaller effects, chances of detecting and then replicating a significant association are not good.

5. Comprehending combined effects of several genes

Once a few QTL effects have been identified, it is then time to find out how the genes work. One approach would be to locate the relevant portion of the DNA molecule with greater precision and then clone and sequence the gene. For those interested mainly in the behavioural consequences, this biochemical enterprise is best left to other labs with the requisite expertise. Interesting experiments are nonetheless possible by combining the various QTL alleles in different ways to determine whether they act independently or instead interact. The question of interaction is of great importance for models of cognitive evolution. Models with independent and additive genetic effects are much simpler mathematically and yield interesting predictions with relatively little effort. On the other hand, when effects of a gene at one locus depend on the organism's genotype at another locus (epistatic interaction), there is no way to predict the outcome of future experiments because so many kinds of interaction are possible. Instead, the central task becomes the understanding of the mechanisms of the interaction itself.

Many interactions among genetic loci have been documented in neurogenetics by creating 'double mutants,' animals which are afflicted by two different genetic disorders at the same time. If each gene modifies behaviour by a certain amount, independence of genetic effects requires that the double mutant behave according to the sum of the separate effects of the two loci. Interaction, on the other hand, might lead to synergistic multiplication of the effects or perhaps sparing. The latter results occurred for the mutations *Lurcher (Lc)* and *staggerer (sg)* which lead to malformations of the cerebellum of mice. 'The *Lurcher* mutation by itself causes the complete loss of all the large Purkinje neurons in the cerebellum, yet when *Lurcher* is combined with the *staggerer* defect, many of the Purkinje cells survive (Messer et al., 1991) for reasons that are not understood. Epistatic interaction reveals that the different genetic loci act jointly during development and that the consequences of a genetic abnormality at one locus depend strongly on what forms of genes are present at other loci.

It seems that there are limitations on our abilities to detect epistatic interactions, just as there are for linkage. Suppose there are two loci (A and *B)* where recessive mutations *(aa* and *bb)* exert similar effects on a measure

of behaviour. Epistasis is evaluated by creating double mutant animals *(aa bb)* and analysing the data for four genotypes with a two-way analysis of variance to test for interaction. In Fig. 2, a model predicts that epistasis will result in a higher score (3g) for the double mutant than would be expected via independent effects (g + g = 2g). The sample size per group that is needed to detect the interaction effect with a specified degree of sensitivity (power = 1 — *β)* can readily be determined (Wahlsten, 1991). Likewise, a model for three loci would require eight groups, one of which will be a 'triple mutant' *(aa bb cc),* and epistasis will be tested by both the two-way and three-way interaction effects. For a model where the group afflicted by all the recessive mutations is one unit of g higher than the respective mutants affected by one less recessive genotype, the requisite number of animals increases rapidly with the number of relevant loci (Wahlsten, unpublished manuscript), as shown in Table 2. It is evident that for genes with modest effects on behaviour, it will be feasible to study the combined effects of only two or three of them in any one experiment.

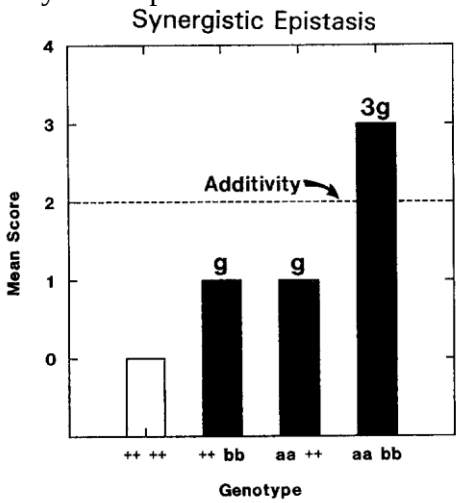

Fig. 2. Expected mean scores on a test of behaviour for four genotypes involving two alleles at two loci in a 2×2 experimental design. The score of the group with neither mutation (genotype + + + +) is taken to be 0, each recessive homozygote (*aa* + + and + + *bb*) is g units above 0, and the double mutant (*aa bb*) is 3g units, one more than expected by additivity of effects at the separate loci (g+g = 2g). These mean scores are used to calculate the size of the interaction effect and the number of animals that must be tested to yield a desired power for the test of the null hypothesis that there is in fact no interaction.

If scientists can expect to investigate in depth the joint functioning of only two or three genetic loci, two conclusions are apparent. First, models of cognitive functioning which involve more than three loci probably cannot be evaluated for practical reasons, and classical genetic and biochemical pathway analyses are likely to remain intractable. Second, we will be able to achieve an adequate understanding of only a small portion of the total system of enzymatic, biochemical relations that form the substrate for cognition. It has been estimated that in humans there are more than 50000 distinct genetic loci, 30000 of which are expressed as unique proteins in the brain at some time during the life span (Sutcliffe, 1988). In other mammals these frequencies will be of the same order of magnitude.

Table 2
Number of animals (*n* in each group) needed to detect epistatic interaction as defined in Fig. 2 when the desired power of the statistical test is 90% [a]

Number of relevant loci	Groups	*n*	Total $N = n \times$ groups
1 [b]	2	23	46
2	4	44	176
3	8	86	688
4	16	170	2,722
5	32	339	10,824

[a] Results in the table assume Type I error probability is 0.05 with a two-tailed test and effect size for the difference between the two homozygous genotypes when only one locus is tested is $\delta_0 = 1.0$.
[b] Epistasis cannot be tested when only one locus is involved, but the *n* required to detect a difference between two genotypes is shown for purposes of comparison.

6. An alternative approach to complex genetic systems

It follows that for the complex systems of gene products (hormones, enzymes, neurotransmitters, etc.) involving dozens, hundreds or even thousands of different genes combining in the process of brain development leading to cognition, analysis at the biochemical genetic level will probably not provide a very satisfactory understanding of cognitive evolution. In this event, cognitive evolution can better be explored by studying processes that occur at a level of reality much closer to the thinking of the whole individual existing in a society. This level involves the primary organ of thought, the brain. Brain-behaviour relationships can be fruitfully explored in the absence of a good understanding of the genetics of the brain (Raichle, 1994). For example, interesting behavioural consequences of an alteration in the anatomy of the mossy fibre projection in the mouse hippocampus have been documented (Crusio et al., 1993; Lipp et al., 1989; Roullet and Lassalle, 1992), even though the genes responsible for the anatomy are not known. Likewise, the behavioural effects of hereditary absence of the corpus callosum can be explored without knowing precisely how many genes are involved (Bulman-Fleming et al., 1992).

7. Conclusions

Cognitive evolution can be studied at several different levels, ranging from complex societies of interdependent persons to the DNA molecules coding for enzymes that synthesize neurotransmitter molecules. Genetic models of cognitive evolution can be fairly evaluated only if they involve one or two genetic loci, maybe three loci if a massive investment of resources is made. If a simple genetic model is seriously proposed, it ought to be tested by genetic linkage analysis so that future theorizing can be guided and constrained by facts. For more complex behavioural characteristics based on large numbers of genes and intricate interrelations with the environment, genetic analysis and genetic theories are not likely to yield conclusive results. Instead, studying individual differences in the brain and neural correlates of cognitive processes will likely provide more rapid progress toward a deeper understanding of evolution.

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