Chromosomal rearrangements, speciation and the theoretical approach

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Many theoretical papers investigating the relationship between chromosomal change and speciation are found to have been based on erroneous data. For rather than considering those negatively heterotic, or at least potentially negatively heterotic rearrangements which can have a possible role in speciation, these papers have included substantial amounts of information on rearrangements which are not implicated in this process. Common forms of chromosomal repatterning such as heterochromatic addition and polymorphism are in this category. Their inclusion in theoretical studies, often cited as supporting or opposing a chromosomal involvement in speciation, invalidates these findings. A new approach is suggested.

INTRODUCTION

When we consider the systematic array of organisms studied, and the multitude of lifestyle characteristics they exhibit, it is hardly surprising that a number of modes of speciation have been proposed to explain this diversity. Despite this observation, it is clear that certain mechanisms such as the process of genic allopatry (speciation by isolation and genic mutation), are common to many plants and animals. In a great number of groups, a high correlation between chromosomal reorganisation and species diversity has also been found (see White, 1973). Numerous models have been proposed to explain this relationship in terms of chromosomally mediated speciation. Examples such as saltational speciation (Lewis, 1966), stasipatric speciation (White, 1968; 1978), the monobrachial homologies model of Capanna (1982), and primary chromosomal allopatry (King, 1981, 1984) are but a few of many. In all of these models, negatively heterotic chromosome changes provide the structural basis for reproductive isolation by effecting fertility. There now appears to be a considerable amount of evidence which shows that certain gross chromosomal rearrangements do have substantial fertility effects (see White, 1978; Gropp, 1982; Capanna, 1982), and that they can therefore initiate speciation.

A series of publications has attempted to assess the role played by chromosomal rearrangements in speciation from a theoretical viewpoint (Bush *et al.*, 1977; Imai, 1983), or to ascertain the rate of chromosomal/karyotypic evolution. Their conclusions include the involvement of chromosomes in speciation (Wilson *et al.*, 1975; Coyne, 1984; Larson *et al.*, 1984). Theoretical overviews of this type are often cited as evidence for or against chromosomal speciation, although as in the examples selected, there has been a less than rigorous evaluation of their data base. Indeed. because of errors of unnecessary inclusion, findings made in these studies are at least in doubt and in most cases invalid. The aim of this paper is to highlight some of the problems found in the current theoretical literature and to suggest a more effective approach to this type of analysis.

CATEGORIES OF CHROMOSOME CHANGE

The single most important criterion is to distinguish between those chromosomal changes which are or can be involved in speciation, and those which can not be. Before analysing the data presented in the above papers, it is important to clarify this concept in some detail. Of the spectrum of chromosomal changes that can occur, it is only those few rearrangements which are negatively heterotic or are potentially negatively heterotic,

that can possibly play a role in cladogenic proces-
ses. These include those rearrangements which
malsegregate at meiosis in structural hybrids, pro-
ducing aneuploidy, duplications and deficiencies,
or which disrupt meiosis entirely. Changes that
have the capacity to be powerfully negatively
heterotic are tandem fusions, reciprocal transloca-
tions, centric fusion or fission, multiple centric
fusions, or multiple centric fusions which share
brachial homologies (see White, 1973; Capanna,
1982). However, if any of these rearrangements
produce a balanced meiotic system, in which nor-
mal segregation of the meiotic products occurs,
they will no longer be able to form a postmating
isolating mechanism and will generally result in
chromosomal polymorphisms. It is not at all cer-
tain that these disruptive changes will always be
involved in speciation, although they clearly have
the potential to be so.

A second class of rearrangement is recognisable
and this includes both pericentric and paracentric
inversions. In this case the data are quite
ambiguous as to the involvement of such changes
in speciation. Both forms of inversion have the
propensity to produce lethal or deleterious meiotic
products, the effect of which can be maximised by
the localisation of chiasmata (see White, 1973)
resulting in dicentric and acentric fragments, but
this is not necessarily the course of events. Indeed,
it has been suggested on a number of occasions
that pericentric or paracentric inversion heterozy-
gosity may not have any ill-effects at meiosis (John
and King, 1977; King, 1981, John 1981). That is,
meiosis can proceed without any deleterious effects
in pericentric inversion heterozygotes if chiasma
localisation is changed to a terminal position, or
is outside the inversion loop. Alternatively, meiotic
problems may be prevented by non-homologous
pairing in inversion heterozygotes. Analyses of the
synaptonemal complexes of inversion heterozyotes
in *Mus musculus* (Davisson *et al.*, 1981) and *Per-
omyscus maniculatus* (Greenbaum and Reed, 1984)
and standard meiotic analysis of the grasshoppers
Camnula pellucida (Nur, 1968), *Trimerotropis hel-
feri* (Schroeder, 1968) and *Cryptobothrus chy-
sophorus* (John and King, 1977) found non
homologous pairing. However, paracentric inver-
sions induced in laboratory mice did show loop
formation in heterozygotes (Moses *et al.*, 1982;
Poorman *et al.*, 1981). The status of inversions in
the speciation process is thus in doubt.

Clearly, the formation of single inversion
differences, unless demonstrably negatively
heterotic, may be regarded as a form of chromo-
some repatterning. Nevertheless, multiple pericen-
tric inversions involving many chromosomes in the
complement may have a cumulative effect on fer-
tility. Coates and Shaw (1984) proposed that this
was the case in their analysis of the grasshopper
Caledia captiva where inviability of F2 hybrids
suggests that multiple pericentric inversions have
the potential to be effective postmating isolating
mechanisms. However, in many of the species in
which multiple pericentric inversions have been
encountered such as the rodents *Neotoma micropus*
(Warner, 1976), *Peromyscus* species (Greenbaum
et al., 1978), and the fish *Ilyodon furcidens* (Turner
et al., 1985), they involve high levels of polymorph-
ism and cannot be claimed to be involved in speci-
ation. The impact of pericentric inversions on
speciation is therefore ambiguous and will prob-
ably remain so. Paracentric inversions, which are
a most common form of chromosome repatterning
in organisms such as the *Diptera*, Chironomidae
or Simulidae (White, 1973), and which are also
potentially deleterious in their heterozygous state,
appear to be in a similar situation; they often occur
as balanced polymorphisms and they appear to
have a minor role in speciation (see White, 1973;
Zouros, 1982).

The third class of change includes those struc-
tural rearrangements which we know are not in-
volved in speciation. The two forms of repatterning
implicated are first, those neutral or positively
heterotic rearrangements which occur as transient
or balanced polymorphism, and second, the addi-
tion of heterochromatin.

Transient or balanced chromosomal poly-
morphisms generally have no impact on speciation.
The only opportunity such changes might have for
an involvement is when a series of transient poly-
morphisms for sequential chromosome fusions
form monobrachial homologies. In meiosis,
trivalents or complex chain multiples may result,
their complexity being determined by the number
of chromosomes sharing homologies within that
complement. These may malsegregate with pro-
found effects on fertility, and can therefore act as
a postmating isolating mechanism in speciation as
proposed by Capanna (1982). Thus, neutral rear-
rangements have been transformed into negatively
heterotic changes. It must be stressed that this is
an uncommon situation and involves fixation of
the changes, so this is not a true reflection of the
effect of polymorphism. However, even if this were
the case and complex chain multiples were formed,
with the occurrence of balanced segregation such
changes could provide no barrier to fertility. Gen-
erally, chromosomal polymorphisms which are
balanced in their segregation and are neutral or

adaptive, can not form postmating isolating mechanisms.

There is little evidence to support the involvement of heterochromatic addition in speciation. John and Miklos (1979) have thoroughly dealt with the arguments for and against this form of change. The claims made by Corneo (1976), Fry and Salzer (1977) and Yunis and Yasmineh (1971) remain without support. It is noteworthy that heterochromatin addition is a particularly common form of change in rodents, a group often referred to in speciation studies (see below). Examples showing pronounced C-banding variation include: Patton and Sherwood (1982) in *Thomomys*; Robbins and Baker (1981), Bradshaw and Hsu (1972), Greenbaum *et al.*, (1978) in *Peromyscus*; Baker *et al.*, (1979) in *Onychomys*; Mascarello and Hsu (1976) in *Neotoma*; Baverstock *et al.*, (1982) in *Uromys*. These all show heterochromatic blocks in fixed or polymorphic states. There are now numerous studies investigating the effects of chromosomal addition on fertility. In the pocket gopher *Thomomys bottae* (Patton and Yang (1977)), the white tailed rat *Uromys caudimaculatus* (Baverstock *et al.*, 1982) the grasshoppers *Atractomorpha similis* (John and King, 1983) and *Cryptobothrus chrysophorus* (John and King, 1980), there is no evidence for reduced hybrid fertility between cytotypes possessing gross structural heterozygosity for the amount and localisation of heterochromatin.

At the risk of repetition, it must be emphasised that studies considering the question of speciation and the involvement of chromosomes in this process, should only consider those monomorphic rearrangements which are demonstrably negatively heterotic, or at least have the potential to be so. It is now clear, however, that a number of theoretical papers which are often cited as supporting or opposing the relationship between chromosome change and speciation, are suspect because of the information included in their data bases. These cases are detailed below.

PUBLISHED DATA AND THEORIES

In a recent paper directed at investigating the relationship between karyotype alteration and species differentiation in mammals, Imai, (1983) made the following assessment of the chromosome rearrangements he used: "Visible chromosome rearrangement denotes in this paper a series of rearrangements changing chromosome number and/or arm number (*i.e.*, Robertsonian rearrange-

ment, pericentric inversion, or tandem growth of constitutive heterochromatin in short arms)", (p. 1154).

Further on Imai (1983) tells us: "In a species having chromosome polymorphisms, I count each homomorphic karyotype in a chromosome polymorphism as one," (p. 1155). That is, the analysis that Imai has produced is to a large extent based on chromosomal changes that either have an ambiguous role in speciation (pericentric inversions), or no role in speciation (growth of consitutive heterochromatin and chromosomal polymorphism). Interestingly enough, of the 967 mammals analysed 293 were from the Rodentia, a group in which such chromosome changes predominate. Clearly, these data can not provide a valid comment on speciation. Imai concluded that:

"(1) differentiation of species by speciation without karyotype alteration..."

"(2) substitution of karyotypes in each resulting species..." (p. 1160) predominates in these vertebrates. These findings are at least partially based on data which do not pertain to chromosomal speciation and are therefore spurious.

While Imai's paper argued against chromosome change acting as a primary agent in speciation, a series of other papers which are equally as suspect, have supported the concept of chromosomal speciation. Wilson *et al.*, (1975) Bush *et al.*, (1977) and more recently Larson *et al.*, (1984), have proposed that a relationship exists between social structuring of animal populations, rates of speciation and the involvement of chromosomal change in those processes. The earlier publications have been widely cited in studies involving chromosome change and speciation, although on some occasions not favourably so (see King, 1981; Charlesworth *et al.*, 1982; Bengtsson, 1980). Indeed, King (1980, 1981) pointed out that much of the material used by Wilson *et al.*, (1974), Wilson *et al.*, (1975) when discussing reptiles and amphibians was unreliable. When considering chromosomal change, these authors included modifications in chromosome number and arm number. That is, fusions and dissociations, and also pericentric inversions and chromosome additions were utilised (see fig. 1. Wilson *et al.*, 1974; p. 5056). Moreover, they made no distinction between chromosome changes which were polymorphic or fixed, thus casting doubt on any conclusions relating to chromosome change and speciation.

Bush *et al.*, (1977) tested the hypothesis that small demes promote rapid speciation by esimating the rate of chromosomal evolution in 225 genera

of vertebrates. The authors used what they considered an improved means of estimating the rates of karyotypic change by considering the range in chromosome number and range of chromosome arms per genus. They argued that this approach could estimate the minimum number of chromosomal mutations which must have occurred to produce the observed range of karyotypes. They comment: "Fusions, fissions, and whole-arm inversions are detected, whereas all paracentric inversions, most reciprocal translocations and many pericentric inversions are not. All of the latter are unquestionably important in chromosome evolution and can serve as sterility barriers in speciation" (p. 3942).

This improved means of estimating the rate of chromosomal evolution and of speciation, is still based on the erroneous assumptions found in their earlier publication (Wilson et al., 1974). Heterochromatin addition, a means of chromosomal repatterning common in vertebrates, has been included as have other rearrangements not associated with speciation. It remains unclear whether chromosomal polymorphism has been included or not. Conclusions reached by these authors on the utilisation of structural rearrangements in speciation, or on the life style characteristics which support such processes must remain unsubstantiated.

More recently, Larson et al., (1984) when analysing the relationship between rates of chromosome change, social behaviour and speciation used the same types of raw data, based on the number of chromosomes and chromosome arms. The authors acknowledged: "The structural rearrangements revealed comprise a subset of the total rearrangements taking place: Robertsonian rearrangements (centric fusions and fissions) and whole arm inversions are counted but paracentric inversions are not counted, nor are most interstitial additions and deletions of chromatin" (p. 216).

It would appear that tandem fusions have not been counted, and that whole arm chromosome additions have been. Moreover, it is still not clear whether polymorphisms have been included or not. Once again, the data base incorporates chromosome changes which are irrelevant to the speciation process, and conclusions pertaining to chromosomal involvement in speciation derived from these data are also spurious.

When investigating the correlation between heterozygosity and the rate of chromosome evolution in animals, Coyne (1984) used the data produced by Wilson et al., (1975) and Bush et al., (1977). The conclusions reached by Coyne (1984)

on rates of chromosomal evolution were extended to the relationship of chromosomal change to speciation, and to support of the canalisation model of Bickham and Baker (1979). The selection of inappropriate data from discussions on rates of karyotypic/chromosomal evolution (which may well be valid with such data) to speciation (which are not valid) has been made. Indeed, the inference that rates of chromosomal evolution are directly linked to the involvement of chromosomal change in speciation remains unsupported. Only a small portion of those karyotypic changes which have occurred or have reached fixation, have ever had the capacity or potential to be utilized in speciation. Presumably, many structural rearrangements we see in both fixed and polymorphic conditions are either neutral or in some cases adaptive (John and Miklos, 1979; John and King, 1983). While it is undeniable that a high level of chromosomal mutation is essential for chromosomal speciation, estimates of gross rates of chromosomal evolution can tell us a great deal about the level of karyotypic reorganisation, but nothing about speciation.

CONCLUSION

The purpose of this article has been to suggest that a new approach is required. A more realistic interpretation of the existing chromosomal data on vertebrates could be obtained if they were subdivided into several classes, categorising chromosomal changes which can be expected to play a role in speciation and those which can not. Indeed, further subdivision into individual types of chromosomal rearrangements may be advantageous. Arbitrary subdivisions of this nature when related to such paramaters as social structuring, vagility, and population size, could thus provide a more realistic appraisal of the relationship between chromosomal change and speciation than does the current approach. However, the final arbiter which decides whether a particular rearrangement can or cannot become involved in the speciation process, is the meiotic system of the individual. A complex structural rearrangement may segregate in a balanced fashion in one organism and not in another, and in the final analysis this single event decides whether a rearrangement can ever play a role in speciation.

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