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CHROMOSOMAL EVOLUTION, SPECIATION, AND SYSTEMATICS: SOME RELEVANT ISSUES

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WE present in this paper a brief review of several aspects of chromosomal evolution and speciation that are only superficially discussed in the accompanying paper by Kluge (1994). Our intent is to highlight some of the issues relevant to a deeper understanding of mechanisms by which new chromosomal mutations become established in natural populations, the "speciation potential" that fixation of such rearrangements may have in either initiating and/or subsequently contributing to the divergence process, and what implications these processes may have on the utility of chromosomal characters for phylogenetic inference. We purposefully omit any detailed consideration of molecular chromosomal architecture and mutation mechanisms, because such a discussion is beyond the scope of this paper and outside of our primary interests. We take it as axiomatic that different classes of chromosomal rearrangements arise as spontaneous mutations in natural populations, although likely at varying rates [some of the mutational mechanisms are reviewed by Fontdevila (1992) and Redi et al. (1990)], and that the frequencies with which these rearrangements become fixed vary considerably as a function of many

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interrelated internal (e.g., molecular, cytomechanical, meiotic) and external (e.g., effective population size, gene flow and metapopulation structure, dynamics of demic extinction and recolonization) factors (Barton and Rouhani, 1991; Spirito, 1992). Some of these are considered in more detail below.

This review will overlap a few of the points raised by Kluge (1994), but we do not wish to participate in the debate between Kluge and King over the details of gekkonid phylogeny and classification per se. We emphasize at the outset, however, our philosophical commitment to phylogenetic principles (Hennig, 1966) and adopt the position that testing the direction of character change—in this case patterns of chromosomal evolution within monophyletic groups—requires a cladistic hypothesis based on independent (non-chromosomal) characters. As we point out below, formulating and testing rigorous hypotheses about mechanisms of chromosomal evolution and speciation must involve investigations of population-level processes, but a well corroborated phylogenetic framework must also be developed to provide a historical perspective on patterns of chromosomal change in genealogical and adaptational contexts (Brooks and McLennan, 1991; Harvey and Pagel, 1991).

Investigations on the nature and role of chromosomal evolution require multidisciplinary studies on a variety of interrelated issues in chromosomally polytypic taxa that may be undergoing some form of chromosomally mediated divergence. Many of these issues were enumerated by White (1978*a*:11–12) and will be further discussed here. A point that we stress is that very few examples of such phenomena are known in amphibians and reptiles. We have not attempted to include in this review all known cases of chromosomal polytypy in the herpetological literature, because most of these have been reported only in an anecdotal sense. Rather, we emphasize some of the major issues, and then consider these with respect to two groups of lizards in which well characterized chromosome races have been discovered within taxa that were previously considered to be single morphological species. These include the *Sceloporus grammicus* complex (Iguania: Phrynosomatidae) in Mexico and the *Liolaemus monticola* complex (Iguania: Tropiduridae; both families are recognized on the basis of the hypothesis presented by Frost and Etheridge, 1989) in Chile. *Sceloporus* and *Liolaemus* are in many respects independent "experimental" replicates exhibiting strikingly similar chromosomal phenomena at equivalent hierarchical levels, suggesting that similar mutational/selection forces and/or constraints may be operating on both radiations. Both genera contain species complexes that show maintenance of within-species chromosomal polymorphisms, the formation of parapatric hybrid zones between chromosome races, and independent evolution of several chromosomal lineages presumably derived from a similar ancestral karyotype. These features make both groups well suited for in-depth studies of many of the issues described in this paper. Of necessity however, most of the phenomena that we describe are taken from non-herpetological taxa (chiefly eutherian mammals and orthopteran insects). This bias simply reflects the preponderance of examples, and in some cases the depth and sophistication of long-term studies, that are available for these groups. Readers should keep in mind, however, that most of the questions investigated in the mammalian and insect groups are relevant to the same issues in amphibians and reptiles.

Hypotheses of Stochastic Chromosomal Evolution

A number of process oriented hypotheses have explicitly proposed a causal role between the fixation of one or more new rearrangements and the initiation of speciation. These models may require a single (White, 1968) or multiple rearrangements (Hall, 1980, 1983; Walsh, 1982; White, 1978*b*) but require stochastic (genetic drift) rather than deterministic (natural selection) forces for establishment of a new mutation. The early models were based on the observation that closely related species (which were seldom defined in a phylo-

genetic context) frequently differed in their karyotypes by the fixation of one or more rearrangements that were presumed to have been underdominant upon their origin as individual heterozygotes. Within-species cytogenetic surveys, when undertaken, usually revealed that intra-population polymorphisms for rearrangements diagnostic of the between-species differences were rare or absent (White, 1973). These observations were taken together as prima facie evidence for stochastic fixation of such rearrangements and their subsequent causal role in the divergence process. Because such mutations originate in a heterozygous condition, they should be immediately eliminated by selection in a random mating population. Consequently, a necessary corollary of these hypotheses is that the population structure must be sufficiently subdivided into small, isolated (or nearly so) breeding units in which sampling error may occasionally override selection and fix a strongly underdominant rearrangement. In small demes, genetic drift can increase the frequency of a new rearrangement above the unstable equilibrium frequency (0.5), after which it can be fixed by selection favoring the new homozygote. Such a transition corresponds to a shift between adaptive peaks on a "Wrightian" population landscape, an important part of Wright's (1970) "shifting balance" theory of evolution. It is important to note that, when underdominance is severe, fixation requires extremely small effective population sizes (on the order of $N_e = 10$; Bengtsson and Bodmer, 1976; Lande 1979; Hedrick, 1981; Walsh, 1982; see also Hedrick and Levin, 1984; Templeton, 1980, 1981, 1982). Many of the implications of population structure have been reviewed elsewhere (Fontdevila, 1992; Patton and Sherwood, 1983; Sites and Moritz, 1987; for theoretical treatments see Barton, 1992; Barton and Rouhani, 1991; Spirito et al., 1991), and below we qualify and extend the premises of some hypotheses in light of recent findings. Alternative mechanisms to selection and drift for fixation of new rearrangements (i.e., meiotic drive) have been considered recently by Coyne and Orr (1993) and remain unsubstantiated as general factors of evolutionary importance.

Upon fixation of a new rearrangement in a small population, selection favoring the new homozygote is then hypothesized to effect its spread via expansion or colonization by the population into a new niche, or by displacement of part of the ancestral population. The driving force in the evolution of reproductive isolation occurs upon contact between the ancestral and derived chromosome races, which establishes a "tension zone" (Key, 1968)—a sharp clinal transition in diagnostic markers—with the chromosomal differences between hybridizing taxa functioning as a partial post-mating isolating mechanism. The important point here is that the chromosomally F_1 and possibly some backcross combinations are less fit than pure parental karyotypes on either side of the zone due to any number of potential meiotic anomalies (White, 1973). The hybrid zone itself thus acts as a partial barrier to gene flow, and selection presumably favors the evolution of pre-mating isolating mechanisms to reduce the formation of hybrids. This is viewed as a type of "reinforcement" process that drives speciation to completion (Dobzhansky, 1951, 1970).

A number of permutations exist on the general theme outlined above, especially with regard to the number of rearrangements required to initiate speciation (one versus several), the meiotic consequences of chromosomal heterozygosity, the geographic relationships of ancestral and derived chromosome races, the permissible population structure, and the nature of the barrier to gene flow across a hybrid zone (see Table 2 in Sites and Moritz, 1987). Some of the most restrictive hypotheses are those proposing major underdominance effects from single mutations (the stasipatric model of White, 1968), and therefore they require an extremely "Wrightian" population structure for fixation. The theoretical objections to this mechanism are considerable (Barton, 1979; Charlesworth et al., 1982; Futuyma and Mayer, 1980; Mayr, 1978; Spirito et al., 1983), and no convincing empirical examples are known (Fontdevila, 1992; Searle, 1993; Sites and

Moritz, 1987). Perhaps even more restrictive are the "chain process" (White 1978*b*) and "cascade" (Hall, 1980, 1983) models of chromosomal speciation, which require sequential fixation of a series of such rearrangements, all by sampling error. Again, no convincing cases have yet been demonstrated (Searle, 1993; Sites and Moritz, 1987; and see below). Other models of "invasive" or "primary chromosomal allopatry" involve colonization and range expansion by a newly arisen chromosome race, but they depend upon the same initial assumptions as do many of the models just listed (albeit in a different geographic context: King, 1981, 1984). In most of these cases, the formation of a hybrid zone between ancestral and derived chromosomal races initiates speciation in the manner described above. King (1987) has recently stressed the point that certain kinds of chromosomal rearrangements (i.e., heterochromatic changes) appear never or at least only rarely to cause meiotic anomalies and are therefore not implicated in any of these processes.

Another set of hypotheses, although differing in many details, collectively provide less restrictive alternatives by postulating the establishment of several rearrangements within a population, each individually with small underdominant or approximately neutral effects. These mutations can thus be fixed in less strongly subdivided populations (Barton and Rouhani, 1991; Lande, 1984). Strong underdominance comes into play only as a secondary consequence of their combined effects in a hybrid zone, where rearrangements may contribute substantially to postmating fitness loss. Walsh (1982) provided a general model to show how this could occur (see also Barton and Bengtsson, 1986), and Capanna (1982), Baker and Bickham, (1986), and Moritz (1986) have implicated monobrachial fusions as one class of rearrangements likely to operate in exactly this manner (see also Searle, 1993). In this case, chromosomal evolution may be viewed as a dual level process: one set of factors determines the frequency with which new mutations arise and become established within demes; and a completely different set (effects of altered genetic background or chromosomal context on meiosis) determines the fitness levels of F_1 and backcross genotypes resulting from hybridization (King, 1992).

A third alternative is the "recombinational breakdown" hypothesis (Shaw, 1981) developed on the basis of observations in the *Caledia captiva* complex of grasshoppers (Orthoptera), which postulates a barrier to gene flow resulting from the disruption of coadapted gene complexes by altered recombinational patterns in heterozygous F_1 individuals (see also Shaw and Coates, 1983). In a hybrid zone, the F_1 generation shows no decrease in fertility, but developmental breakdown is severe in progeny of this generation (F_2). Hybrid breakdown is not due to disruption of meiosis but rather occurs because pericentric inversion heterozygosity has shifted chiasmata into regions previously shielded from recombination in both parental types. This mechanism contrasts strongly with the previous models, all of which stress the production of a high frequency of aneuploid gametes in chromosomal heterozygotes (Table 2 in Sites and Moritz, 1987). However, the model resembles those based on monobrachial homology in that its requirements for a particular population structure are much less restrictive, and rearrangements are only deleterious when they occur in novel combinations within hybrid zones. The examples cited above show that pericentric inversions are frequently not underdominant as within-population polymorphisms (see also Coyne et al., 1991, 1993) and could therefore become fixed in a variety of population structures. Chromosomal evolution again may be viewed as a dual level process by this mechanism (King, 1992).

Hybrid Zone Dynamics

In the simplest cases, speciation could occur as a sole consequence of chromosomal rearrangements under two conditions (Searle, 1993). First, if the karyotypic differences between populations render hybrids completely sterile, strictly as a result of structural chromosomal heterozygosity (i.e., genic differences, if present,

do not contribute to the sterility), speciation would be immediate. This is not the case in the overwhelming majority of hybrid zones studied to date (Harrison, 1990; Searle, 1993). An alternative possibility is fertility reduction in hybrids resulting from chromosomal heterozygosity that provides a partial barrier to gene flow, thereby facilitating genic divergence, and presumably also favoring selection for reinforcement of premating isolating mechanisms.

Determining exactly how chromosomal rearrangements may contribute to fitness loss in hybrid zones requires several lines of sometimes difficult-to-attain evidence. First, data from eutherian mammals (Searle, 1993) suggest that reduced fertility in chromosomal hybrids arising from meiotic anomalies should have different consequences for each sex. Aneuploid gametes formed in males may suffer germ cell death, but unless this fraction of the total gamete production is very large, or unless aneuploid gametes mature and fertilize at the same rates as euploids, aneuploidy may be of little consequence because of the number of gametes produced. Female chromosomal heterozygotes undergoing high levels of nondisjunction and forming aneuploid oocytes, or being fertilized by aneuploid spermatocytes, may show two kinds of fitness effects (Mercer et al., 1992). *Primary infertility* may result from germ cell death at implantation (which is manifested as a smaller-than-average clutch), and *secondary infertility* may result from abortion of embryos later in gestation (manifested as a normal clutch size, but containing one to several aborted conceptuses). In eutherian mammals, primary infertility is the rule for monosomics, while secondary infertility is the rule for trisomics (Searle, 1993).

Compounding these issues are several others that may also contribute to hybrid unfitness independently of chromosomal heterozygosity, but are again frequently difficult to assess. These include possible selection for a particular karyotype in a particular habitat (Haldane, 1948; and see below), so that chromosomal clines are partly maintained by selection against introgression of particular rearrangements into the wrong environments or onto the wrong genetic background. Decreased fitness of hybrids may also result from effects of other genes (linked or unlinked) that are in linkage disequilibrium with the rearrangement(s). For example, continued immigration of parental types from either side into the zone will generate linkage disequilibria between diagnostic chromosomes, which if strong enough will keep chromosomal clines concordant with each other and other selected genes (Barton, 1983). Further, if hybridizing populations are characterized by heterogamety, then a significant amount of post-zygotic reproductive isolation may be caused by the sex chromosomes, which may have very large effects on hybrid sterility/inviability independent of autosomal interactions (Coyne, 1992; Coyne and Orr, 1989*a*,*b*). We emphasize the point that a direct test for the role of chromosomal rearrangements in divergence depends on assessing the relative contributions to fitness loss of these other factors.

Hypotheses of Adaptive Chromosomal Evolution

A fundamental dichotomy of ideas about the evolutionary significance of the fixation of chromosomal rearrangements is quickly apparent from the recent literature. Those reviewed above represent mechanisms of stochastic chromosomal evolution while a second category of ideas includes several different hypotheses on the adaptive nature of the karyotype. Bickham and Baker (1979) hypothesized that an optimal karyotypic configuration is selected for early in the radiation of a monophyletic group as it is beginning to colonize a new adaptive zone, while Imai et al. (1986) suggested that particular chromosomal rearrangements are selected for because they minimize the probabilities of deleterious intracellular between-chromatid interactions (i.e., breakage and/or nonreciprocal exchange).

The "canalization model" of Bickham and Baker (1979) predicts three stages of karyotypic evolution during an adaptive radiation. First, during the initially rapid species diversification, many karyotypic

rearrangements (mostly non-Robertsonian) occur in such a manner as fundamentally to reorganize many linkage groups in the genome. These linkage groups are selected toward a near-optimum set of linkage relationships for the adaptive zone being filled. The second stage of karyotypic change occurs more slowly as the overall structure of the karyotype is "fine tuned" by Robertsonian or tandem fusion changes that do not alter whole-arm linkage groups. The final stage (stage 3) is reached when the karyotype is "canalized" at its optimum and karyotypic stability prevails because further rearrangements are maladaptive in the presently occupied adaptive zone. This model has been criticized for different reasons by King (1985) and Sites and Moritz (1987). Its chief prediction is that, for a given monophyletic group displaying between-taxon chromosomal variability, rearrangements should be hierarchically structured so that non-Robertsonian changes characterize the deep history of the group, while Robertsonian changes prevail at the younger nodes. This model was based on patterns originally described in bats and turtles, but it has not been supported in subsequent tests (see Sites and Moritz, 1987, for details).

The "minimum-interaction hypothesis" of Imai et al. (1986) is based on the idea that the main deterministic force governing karyotypic evolution has been selection for reduced opportunity for spontaneous negatively heterotic chromosomal mutations. The majority of spontaneous chromosomal rearrangements occur in synaptonemal complexes via crossovers and mis-resolution of interlockings during pachynema, a stage of meiotic prophase in which bivalents are extremely elongated and fixed at their telomeres to the nuclear membrane. This arrangement of bivalents in prophase nuclei is highly structured and nonrandom, and it represents a universal configuration in eukaryotes referred to as the "suspension-arch structure" by Imai et al. (1986). Because potentially deleterious rearrangements such as reciprocal translocations result from interactions between nonhomologously associated chromosomes, the configuration of bivalents significantly affects their occurrence probabilities. Specifically, the size of autosomal chromosomes (which is usually inversely correlated with number) and nuclear volume chiefly determine the configuration of the suspension-arch structure, and therefore the frequency with which different bivalents interact.

Simulation studies carried out by Imai et al. (1986), based on observed ranges of diploid numbers in ants of the genus *Myrmecia*, and the frequencies of several classes of rearrangements (mostly in *Drosophila* and humans), revealed two major trends. When nuclear size (as measured by radius, r) is constant, the frequency of reciprocal translocations decreases with an increase in total chromosome number, and when the 2n is fixed, the frequency of translocations decreases as r increases. Selection thus appears to reduce the interaction probabilities by acting on the chromosome size(2n)/nuclear volume(r) ratio of oocytes and/or spermatocytes at pachytene. The hypothesis predicts that when the 2n:r ratio is low, the frequency of reciprocal translocations should be low and an increase in the diploid number would not be strongly favored. Conversely, under high 2n:r ratios, there should be strong selection for an increase in diploid number to reduce the size of at least the largest autosomes. To our knowledge, this hypothesis has not yet been tested in any comparative phylogenetic framework.

More recently, studies of chromosome races of the *Caledia captiva* complex by Shaw et al. (1988) have provided evidence for another adaptive role for the specific chromosomal structure of a eukaryote genome. These authors demonstrated a very gradual, concordant clinal shift from completely metacentric, through all intermediates, to completely acrocentric morphologies in the six largest autosomal chromosomes along a north–south transect spanning the entire 1500 km distance between the Moreton and Lakes-Entrance races. These shifts are apparently due to small asymmetrical pericentric inversions occurring sequentially in parallel in all markers, and they appear to track an environmental gradient in length of summer

and patterns of rainfall. Shaw et al. (1985) had previously argued that patterns of karyotypic variation in *Caledia* reflected an underlying adaptive significance to the chromosome structure, for the first time arguing that the fixation of chromosomal rearrangements was driven by positive selection for direct phenotypic adaptations, rather than by sampling error (see also Shaw et al., 1990). A more recent study by Groeters and Shaw (1992) revealed a significant correlation along the transect between embryonic development time and chromosome structure, and the authors postulated that this pattern is due to changes in voltinism which tracks the north–south variation in length of available growing season. Unlike the canalization model, the hypothesis advanced by Shaw and collaborators specifies an adaptive phenotypic role—directly tied to environmental variables—likely to act at the population level. The hypothesis is also free of constraints about how different kinds of chromosomal mutations should evolve (e.g., non-Robertsonian changes first followed by Robertsonian "fine tuning"), and it does not require postulating that *Caledia* is beginning an adaptive radiation.

The Cases of the *S. grammicus* and *L. monticola* Complexes

The *Sceloporus grammicus* complex of central Mexico illustrates several aspects of karyotypic divergence. Early cytogenetic studies revealed a complex of seven distinct chromosome races, or cytotypes (2N = 31/32–45/46 [male/female]) within a single taxonomically recognized morphological species (Hall, 1980, 1983). The cytotypes are defined primarily by Robertsonian fission rearrangements which were hypothesized to have been derived in a linear fashion from a 2N = 32 karyotype with biarmed macrochromosomes (Hall, 1980). To date, seven hybrid zones involving interactions between six different combinations of chromosome races have been documented (Arévalo et al., 1991, 1993; Hall, 1980, 1983; Hall and Selander, 1973; Porter and Sites, 1986; Sites et al., 1993), and additional contacts are suggested by both molecular and cytogenetic data.

The pattern of karyotypic diversity within the *S. grammicus* complex, the allopatric or parapatric distribution of the cytotypes, and the presence of hybrid zones between several races persuaded Hall (1983) to develop a detailed model of chromosomally mediated speciation. This Cascade model was based on three main premises: (1) chromosomally differentiated species originate as small founder populations; (2) chromosomal differentiation is influenced by mutation rate, meiotic processes and products, mating system, and population structure; and (3) these factors are under genetic control. As with most models of chromosomal speciation, underdominance for chromosomal rearrangements was the central assumption of the Cascade model. However, at the time of its conception, such underdominance had not been demonstrated in a hybrid zone context.

The *S. grammicus* complex has provided a "living laboratory" in which to study several key aspects of chromosomal evolution. Over the past few years, several parallel lines of investigation have tested specific aspects of the Cascade model and several others that require a Wrightian metapopulation structure and chromosomal underdominance. Both comparative (Thompson and Sites, 1986) and computer simulation studies of population structure (Sites et al., 1988) suggest that current populations lack the appropriate population structure required of the most restrictive models. Population cytogenetic studies (Arévalo et al., 1991; Porter and Sites, 1986; Sites, 1983) have shown that in non-hybrid zone contexts, chromosomal polymorphisms are widespread and present in Hardy-Weinberg genotypic proportions, an unexpected observation with strong underdominance assumptions. Further, studies of the meiotic consequences of chromosomal heterozygosity have validated conclusions based on the population structure and population cytogenetic studies. Heterozygosity for fission (Porter and Sites 1985, 1987; Reed et al., 1992*c*) and pericentric-inversion rearrangements (Reed et

al., 1992*b*) were found to be of little meiotic consequence when present within populations (i.e., in non-hybrid zone contexts). These studies have collectively served as "controls" to provide background data against which to compare cytogenetic and molecular studies of hybrid zones.

The first detailed study of a hybrid zone was carried out by Hall and Selander (1973) for an HS(32) × F6(34) contact on the eastern divide of the Valley of Mexico. These workers found evidence of limited hybridization and backcrossing in which diagnostic alleles (one chromosome and two isozyme markers) did not introgress beyond a width of about 3 km, and they concluded that these two races were genetically isolated and behaving as good biological species because selection against hybrids limited gene flow. Arévalo et al. (1993) investigated a poorly defined contact between the same races on the opposite side of the Valley of Mexico and again found evidence of limited introgression. Sites et al. (1993) established a transect across a F5(34) × FM2(46) hybrid zone in eastern Hidalgo (the "Tulancingo" transect) and showed similar patterns. The studies of Arévalo et al. (1993) and Sites et al. (1993) included morphological, chromosomal, isozyme, and mtDNA and rDNA restriction-site markers, and generally supported the conclusions of Hall and Selander (1973)—introgression was limited, probably because of selection against hybrid and/or backcross genotypes in the hybrid zone (see also Sites and Davis, 1989). None of these studies, however, specifically addressed what role might be played by the fixed chromosomal differences between the hybridizing populations in contributing to fitness losses in the hybrid zones.

Because of its accessibility and the degree of chromosomal divergence between the hybridizing populations, the Tulancingo transect has been selected for more detailed study by the authors and collaborators E. Arévalo, N. Barton, S. Davis, and I. Greenbaum. This work is still in the data analysis stage, but we can summarize some of the major findings to this point. A study of microgeographic population structure shows that (1) the zone is characterized by steep, concordant clines for all three diagnostic autosomal markers (chromosomes 1, 2, and 6); (2) there is a strong deficit of heterozygotes across modest geographic sampling scales; (3) strong linkage disequilibrium is characteristic of all paired combinations of marker chromosomes; (4) there is a strong association between karyotype and habitat (F5 is predominant on oak, FM2 elsewhere); (5) dispersal distances are small, on the order of 80–160 m/generation; (6) the zone width is about 830 m (95% confidence limits are 770 and 930 m), and the zone forms an extremely irregular, mosaic contact; (7) strong selection against hybrids is implicated in maintaining the structure of the zone; and (8) selection appears to be based in part on an animal's genotype (those being closer to an F_1 genotype having smaller clutches) *and* its position along the cline (even parental genotypes suffer some reduction in fecundity if they are located toward the center of the cline). Detailed meiotic analyses reveal a lack of underdominance associated with simple fission and inversion rearrangements in this hybrid zone. In contrast to the single rearrangements of chromosomes 1, 3, 4, and 6 which delineate the F5 and FM2 cytotypes, macrochromosome 2 differs between the cytotypes by a minimum of two independent fission events (Reed et al., 1992*a*,*c*). Structural differences in chromosome 2, however, produce increased meiotic disruption (malsegregation and aneuploidy) in hybrid males, but we cannot yet determine if these meiotic irregularities produce measurable reduction in fitness (see also Mercer et al., 1992). Examination of female reproductive parameters reveal increased levels of both primary (germ cell death manifested as smaller clutches) and secondary infertility (inviable embryos) within the hybrid zone. Part of this fitness reduction could be attributed to the effects of heterozygosity for chromosome 2, but clearly "non-chromosomal" genic effects were involved in the reduction of fitness in hybrid females. The non-chromosomal "genic" contribution to the overall loss of fitness may

be substantial, because phylogenetic studies show that the Tulancingo transect is a secondary contact between non-sister taxa that may have been separated for a considerable time. Two of the major conclusions from this work are that chromosome 2 appears to make a substantial contribution to a reduction in female fecundity, strictly as a post-mating isolating mechanism, and that a substantial influx of parental genotypes from both races must be needed to maintain the strong linkage disequilibrium among markers. If these conclusions are correct, reinforcement may not easily evolve in this system, and the hybrid zone may persist and foster divergence in the manner envisioned by Hewitt (1989).

Based on non-differentially stained preparations, chromosomal evolution in *Liolaemus* appears to have included primarily Robertsonian fission/fusion events (Espinoza and Formas, 1976; Lamborot and Alvarez-Sarret, 1989; Lamborot et al., 1979, 1981), although some changes in chromosomal morphology may be the result of other rearrangements (translocations and inversions). Population cytogenetic studies of the *L. monticola* complex have revealed chromosomal polytypy similar to that seen in *S. grammicus*; two currently recognized subspecies (*L. m. chillanensis* and *L. m. villaricensis*) possess invariant 2N = 32 karyotypes, while *L. m. monticola* consists of a "southern" race is characterized by a 2N = 34 karyotype, and a northern race with a 2N = 38–40 karyotype. Differences between the Southern race and the other subspecies of *L. monticola* can be attributed to the addition of one microchromosomal pair, and morphological differences in chromosomes 5, 7, and 9. The Northern and Southern races of *L. m. monticola* differ from each other by centric fissions at chromosomes 3 (polymorphic in 50% of the individuals examined) and 4, and the presence of an additional pair of microchromosomes (see descriptions by Lamborot, 1991). Populations of the Northern and Southern races are separated by natural barriers formed by the Maipo River and its tributaries near Santiago, but they form a secondary zone of contact originating from the construction of a bridge over the Yeso River and a subsequent migration by the Northern race across the river to contact the Southern race. Meiotic data from hybrids (Lamborot, 1991) indicated the presence of pairing irregularities at diakinesis, suggesting the chromosomal differences may result in decreased fitness in heterozygotes. The phylogenetic relationships of the hybridizing populations are unknown, but strong geographic concordance between patterns of morphological and chromosomal divergence strongly implicate the major rivers as barriers to gene flow, suggesting that the overall genetic divergence between the Northern and Southern races may be substantial (Lamborot and Eaton, 1992). This species complex and the dynamics of the hybrid zone await more detailed analyses.

Utility of Chromosomal Rearrangements as Phylogenetic Markers

We have not yet considered issues regarding the identification of independent characters in the karyotype and coding them for phylogenetic analysis. We agree with Kluge (1994) that the information content is maximized when chromosome pairs are treated as separate characters and that between-species chromosome arm homologies should be inferred on the basis of tests of conjunction, similarity, and congruence (Patterson, 1982). Treatment of chromosome pairs as independent characters permits numerical coding and the formation of a character-state matrix amenable to phylogenetic analysis by any of a number of numerical algorithms (see Frost and Timm, 1992; Modi, 1987; Smith, 1990; for recent examples).

When evaluated for phylogenetic content in this manner, chromosomal characters possess the same strengths and limitations as do morphological or molecular characters, and they must be evaluated within the context of a sampling design that will maximize the information available for the reconstruction of evolutionary history. For example, rates of autosomal rearrangements may vary from extremely slow (in the case of cryptodiran turtles, some apparent homologs have remained

unmodified in several families for about 200 million years: Bickham, 1981) to extremely rapid (some populations of tropical rodents of the genus *Oryzomys* segregate high frequencies of multiple fission/fusion combinations: Koop et al., 1983). Not surprisingly, the latter kinds of rearrangements are likely to undergo recurrent origins and are therefore subject to multiple independent fixations (see examples for mammals in Qumsiyeh et al., 1987; Rogers et al., 1984; Stangl and Baker, 1984). Sometimes the genomes of closely related taxa are so drastically altered that the number and exact nature of the rearrangements cannot be identified (Baker and Bickham, 1980). All of these observations have important implications for how variant chromosomes are coded and utilized in phylogenetic studies.

As we have described in the *S. grammicus* and *L. monticola* complexes, some rearrangements are maintained at high frequencies as within-population polymorphisms, and they will complicate character coding unless sampling is adequate to identify within- versus between-population variation. Lastly, different classes of chromosomal rearrangements may be positively or negatively selected, or approximately neutral, in their fitness effects, and so become established at different rates within any given clade. This implies that different weighting options should be incorporated into numerical phylogenetic analysis, perhaps analogous to those currently being explored for different kinds of nucleotide substitutions in DNA sequences (see Knight and Mindell, 1993; Moritz et al., 1992; for recent examples). Testing and extending these ideas, in combination with other kinds of data, will maximize the utility of chromosomal characters in phylogenetic inference.

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