This is in contrast with the lack of considerable alterations in osmotic fragility of bovine erythrocytes in hypotonic NaCl solutions[10]. This is understandable, since ILT_{50} may depend on several parameters including permeability of the cell membrane for a given solute, relative critical hemolytic volume of a cell and mechanical properties of the membrane. As a result, red cells of various ages can differ in the time at which hemolysis occurs[15], though showing no difference in the final fraction of cells lysed in a hypotonic solution. Therefore, although fractionation of bovine erythrocytes according to age was impossible using graded osmotic hemolysis in hypotonic NaCl solutions[10], isoosmotic lysis stopped after various time intervals might provide a general basis for such a procedure. It was observed that the fraction of 1% of bovine erythrocytes most resistant to glycerol lysis had about 2-fold higher activity of a cell-age dependent enzyme, glucose-6-phosphate dehydrogenase, than the whole red cell population, in agreement with this prediction.

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Chromosomal homology in southern Akodon[1]

M.H. Gallardo[2]

Instituto de Ecologia y Evolucion, Universidad Austral de Chile, Casilla 567, Valdivia (Chile), 12 February 1981

Summary. Differential staining (G and C) of southern South American *Akodon* are presented. *A. olivaceus, A. longipilis* and *A. sanborni* all have the same karyotype (2n = 52, NF = 58). A virtually identical band sequence is observed. This situation is interpreted using the canalization model of chromosomal evolution which stresses an optimum karyotype for each adaptive zone. Despite the high degree of conservation of the chromosome structures, the specific status of these species is supported by maintenance of distinctness when they occur in areas of sympatry.

The genus *Akodon* (Rodentia, Cricetidae) is the largest among akodontine sigmodontine rodents and is one of the most confusing and complex taxa of South American rodents. It currently contains 7 subgenera that perhaps deserve the status of full genera[3]. Karyotypically *Akodon* is characterized by chromosome multiformity (2n = 14–52)[3–6] with a wide array of intraspecific and intrapopulational polymorphisms. Such extensive chromosomal diversity indicates that chromosome rearrangements must have occurred quite frequently during the evolution and diversification of the genus. Nevertheless, when bands are analyzed, this variation is shown to be more illusory than real and hence, consistent stability of banding patterns seems to be a rather common situation in the genus.

This paper presents for the first time the banded karyotypes (G and C) of *Akodon (Akodon) olivaceus olivaceus, A. (Akodon) olivaceus brachiotis, A. (Abrothrix) longipilis* and *A. (Abrothrix) sanborni.*

Live trapped animals were collected from the following places in Chile: *A. o. olivaceus* (2 males and 2 females) from Santiago and La Serena, *A. o. brachiotis* (3 males and 4 females) from Valdivia, *A. longipilis* (3 males and 3 females) from Valdivia and Osorno Mountain, *A. sanborni* (1 male and 4 females) from Osorno Mountain.

Voucher specimens in accordance with Osgood[7] were deposited in the Collection of Mammals, Institute of Ecology and Evolution, U. Austral of Chile (IEEUACH). Mitotic plates were obtained by the standard air dried technique described elsewhere[8]. G-bands were obtained by using the trypsin method of Seabright[9] and C-bands as described by Schnedl[10]. Induction of the C-banding pattern was difficult, probably because of the minimal amount of heterochromatin present. A total of 190 representative spreads were photographed and 50 were selected for interspecific comparisons.

The diploid complements of these species, characterized by the same diploid numbers and overall karyotypic morphology, present 52 chromosomes (NF = 58) with 22 pairs of acrocentric and 3 pairs of submetacentric chromosomes (pairs 15, 22 and 25). Sex chromosomes are formed by a medium sized subtelocentric X and a small acrocentric Y. 90 specimens from 14 localities were previously karyotyped by standard technique and no evidence of intra or interpopulation variation in chromosome complement was observed. Their banded karyotypes show a virtually identical G-banding pattern in both autosomes and sex chromosomes, indicating a high degree of homology (fig. 1). The same remarkable correspondence is observed in the C-banding patterns which are characterized by low amounts of centromeric heterochromatin (fig. 2).

A. olivaceus, A. longipilis and *A. sanborni* together with *A. lanosus, A. xanthorhinus* and *A. markhami* occupy the southern-most peripheral distribution of the genus[3]. In contrast with vole mice from the Central Andean region (presumed geographical area of differentiation), these southern species are characterized by chromosome stability and high diploid number[3]. More recently, the same karyotype and chromosome stability described here, has been found in *A. xanthorhinus*[11].

Homology in banding patterns among recognized species is not an unusual phenomenon in mammals. It has been demonstrated in Primates[12], Cetacea[13], Pinnipedia[14], Carnivora[15], Chiroptera[16] and also in rodents as *Neotoma*[17], *Tylomys*[18], *Rattus*[19], and *Mus*[20]. Bianchi et al.[21] have described G-banding homology in Argentinian *A. obscurus* (2n = 34), *A. molinae* (2n = 42, 43, 44) and *A. azarae*

(2n = 38), all of which share up to $\frac{1}{3}$ of the chromosomal complements. However, the sex chromosomes show great differences in spite of morphological similarities. *A. olivaceus, A. longipilis* and *A. sanborni* share close banding in only 3 or 4 chromosomes when compared with those described by Bianchi, indicating more remote phylogenetic relationships.

It has been postulated that chromosome uniformity in Carnivora might be the result of low amounts of heterochromatin[22]. However, a multiform genus (i.e. existence of different chromosome complements within that particular taxon) which has been shown to have only small amounts of constitutive heterochromatin[23] indicates that stability cannot be explained by that hypothesis.

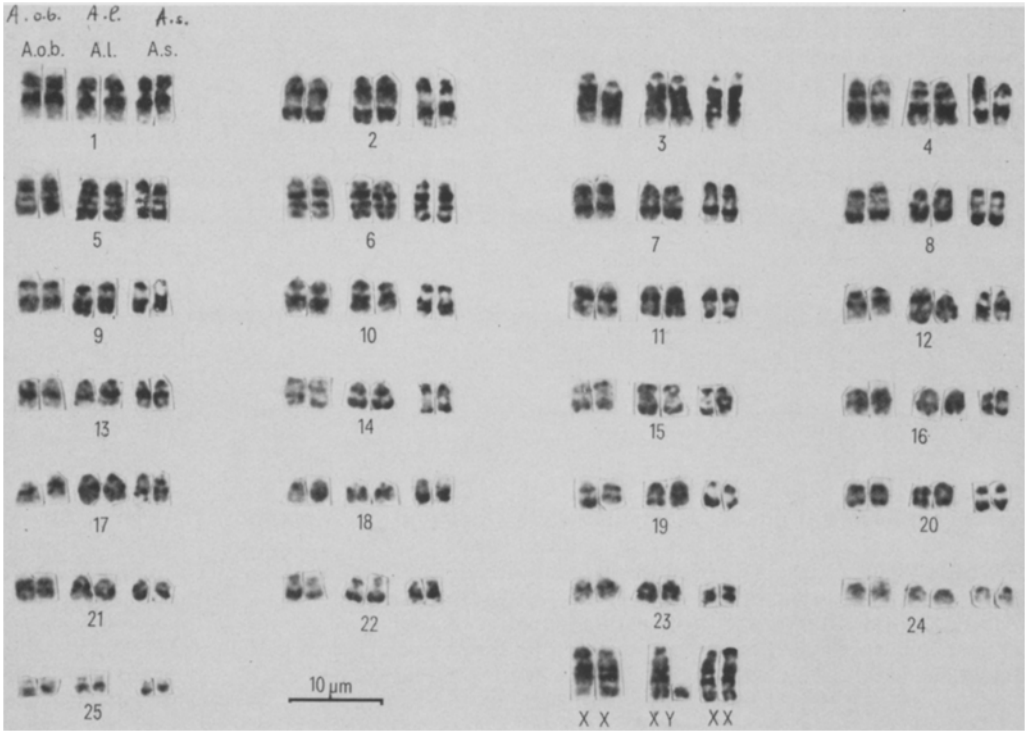

Figure 1. G-banding pattern of *A. o. brachiotis, A. longipilis* and *A. sanborni.*

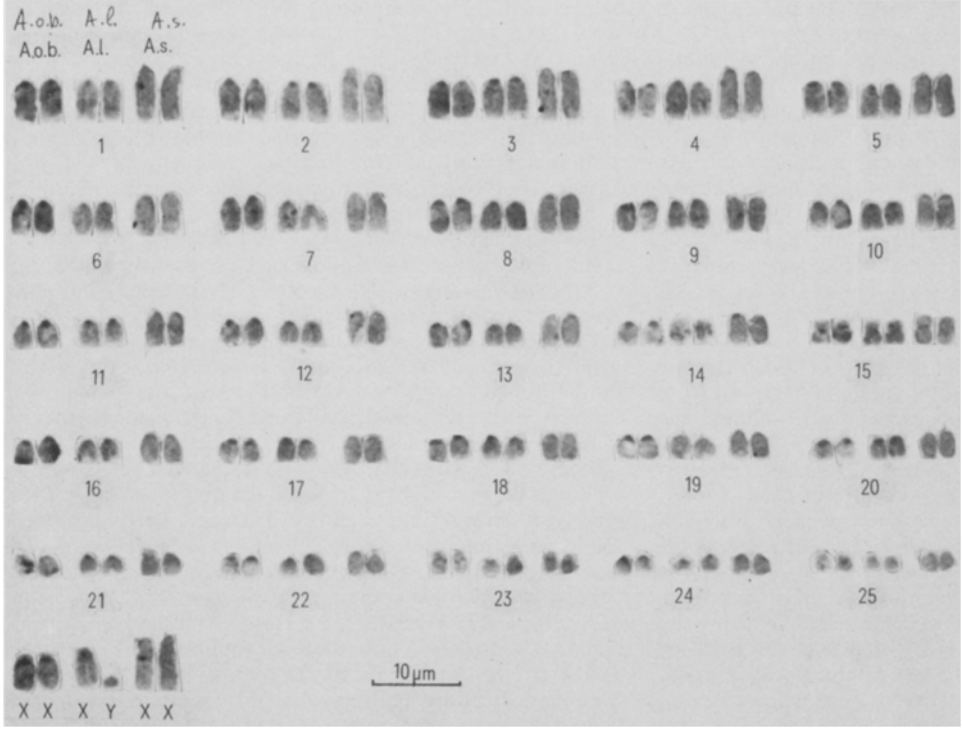

Figure 2. C-banding pattern of *A. o. brachiotis, A. longipilis* and *A. sanborni.*

The adaptive role of the karyotype has been demonstrated in rodents as *Thomomys*[24] and *Spalax*[25]. Bickham and Baker[26], stressing this karyotypical adaptiveness, proposed the canalization model of chromosomal evolution in which 'the karyotype contributes significantly to the fitness of the individual, and that for a given set of biological parameters faced by an evolving lineage, there is an optimal karyotype. Thus, those lineages which have reached that optimum adaptive karyotype would show chromosomal stability; such is the case with these species of *Akodon* and, as they point out, also the situation of *Lasiurus* (i.e. virtually identical karyotypes and morphological distinctness). The 2 subspecies of *A. olivaceus* illustrate chromosomal adaptiveness since animals trapped in 4 localities covering 1400 km latitudinally showed an unvarying karyotype in spite of strong biotic and abiotic differences. In addition, consistent stability of banding patterns has been retained in species belonging to the different subgenera *Abrothrix* and *Akodon* and thus reveals a close genetic relationship between them, which strongly favors the interpretation of descent from a single common ancestor.

Based on morphological grounds, the systematic relationships of these species are clear. It follows that morphological diversification was not accompanied by a significant amount of chromosomal disruption which would markedly alter banding patterns. The biological significance of these morphological differences rests upon the maintenance of distrinctness when the species occur together. Consequently, areas of sympatry provide a test of species status supporting previous assignments.

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2 Present address: Department of Biology, Box 3AF, New Mexico State University, Las Cruces (New Mexico 88003, USA).
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Dystrophic mutation (dy^{2J}) affecting regulation of lactate dehydrogenase (LDH) and pyruvate kinase (PK) in C57BL/6J mice[1]

Shiva M. Singh, Caroline H. Wang and Ann Phillips

Department of Zoology, University of Western Ontario, London (Ontario, Canada N6A 5B7), 7 June 1982

Summary. The genotype difference (dystrophic vs nondystrophic) in the LDH isozymes is observed in kidney. These differences are evident only at birth and at early developmental stages (before the expression of dystrophic symptoms). The tissue specific genotype differences for PK are limited to the thigh muscle (M form) and heart (L form), after the onset of the condition. These differences may reflect the pleiotropic effect of the dy^{2J} locus during the temporal regulation of these and other enzymes implicated in muscular dystrophy (MD).

The phenomenon of enzyme heterogeneity is a general aspect of metabolic structure and function of cells. In general it is characterized by tissue specificity and developmental profiles. The understanding of this general pattern of heterogeneity is particularly desirable for evaluating the role of enzyme(s) in an organ specific genetic defect. Animal models of muscular dystrophy (MD), which is genetically heterogeneous in humans, are a muscle specific genetic defect. In mice the dy^{2J} dystrophic mutation is expressed as the degeneration of thigh muscles, around 3 weeks after birth[2]. Although several enzymes, including lactate dehydrogenase (LDH, E.C. 1.1.1.27), and pyruvate kinase (PK, E.C. 2.7.1.40) have been suggested to be involved in MD[3–6], the basic defect and how it affects specific tissues is not understood[6].

LDH and PK are key enzymes of carbohydrate metabolism (L. lactate $\xrightarrow{\text{LDH}}$ pyruvate $\xrightarrow{\text{PK}}$ P enol pyruvate) with a wide distribution. The genetics, subunit structure and biochemistry of these enzymes are partly understood. Although attempts to utilize these enzymes in Duchenne MD have yielded encouraging results[3], their role in producing dystrophy remains unclear. Most enzyme studies in MD have concentrated on levels of enzyme activity and little attempt has been made in understanding the ontogenic pattern and tissue distribution of different subunits. Such studies are useful in providing insight into the genetic regulation of enzymes with altered activity, which may in fact form the basis for MD and other inborn errors of metabolism for which no basic enzyme defect has been demonstrated. This report deals with the tissue distribution