ADP causes an ATP formation of 0.029 ± 0.005 µmole/min per mg of mitochondrial protein (experiment A). After the addition of 14.1 nmole of Ap_5A (i.e. 28.2 nmole per mg of mitochondrial protein) no ATP formation occurs (experiment B). It should be mentioned here that, in another set of experiments, we made sure that Ap_5A does not affect the hexokinase/glucose-6-phosphate dehydrogenase system itself. The amount of Ap_5A added in experiment A is more than sufficient to inhibit the adenylate kinase reaction completely. We have found that the addition of only 0.5 nmole of Ap_5A leads to a 60% inhibition of the ATP formation corresponding to a K_i value between 10^{-6} M and 10^{-7} M.

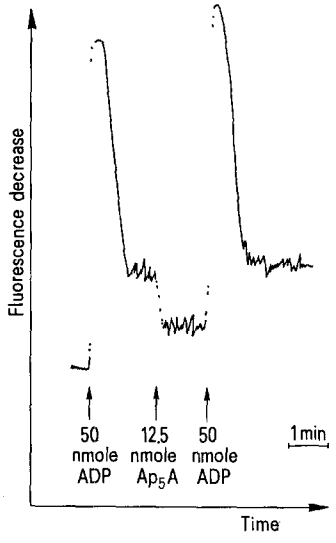

Fig. 3. Redox cycles of the endogenous pyridine nucleotides were carried out in a final volume of 0.5 ml. Excitation filter: 313 + 366 nm, emission filter: 500–3000 nm. The incubation mixture was preincubated with 1 µmole of succinate as an electron donator for 2 min. After the addition of Ap_5A, no alterations of the redox cycles occur.

Looking once more at Figure 1 B, one can see that even high concentrations of Ap_5A do not inhibit the mitochondrial nucleosidediphosphate kinase activity. The addition of 47.2 nmole of UTP leads to a clearly recognizable ATP formation, although Ap_5A is present. Figures 2 and 3 show that even high concentrations of Ap_5A do not at all affect oxidative phosphorylation. In respiratory control experiments (Figure 2), neither state 4, state 3, nor the P/O ratio is altered. These findings are confirmed by assaying the redox cycles of the endogenous pyridine nucleotides (Figure 3). Neither their shape, height, nor basic width is affected by Ap_5A.

Conclusions. 1. Adenylate kinase activity is inhibited by Ap_5A, even when the enzyme is an integral component of the mitochondrial architecture, but its more difficult accessibility leads to a higher K_i value than that established by LIENHARD and SECEMSKI[2] for the pure enzyme. Fortunately the decrease of the inhibitory effect is not very striking, probably due to phenomena similar to the 'intramitochondrial intermembranal large amplitude protein movements' described by WAKSMAN and RENDON[6] for mitochondrial aspartate aminotransferase. 2. The generally accepted view that in intact mitochondria ADP is the primary P_i-acceptor during oxidative phosphorylation has been challenged by OZAWA and MACLENNAN[3,4]. According to their hypothesis, ADP is generated by oxidative phosphorylation of AMP and then converted to ATP and AMP by adenylate kinase. AMP again acts as P_i-acceptor. Our results contradict their conclusions, because Ap_5A inhibits mitochondrial adenylate kinase completely, but does not affect oxidative phosphorylation at all.

[6] A. WAKSMAN and A. RENDON, Biochimie *56*, 907 (1974).

Karyological Patterm of two Chilean Lizards Species of the Genus *Liolaemus* (Sauria; Iguanidae)

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Summary. The karyotypes of Chilean lizards *Liolaemus pictus* and *Liolaemus cyanogaster* is described for the first time. Both species possess 34 chromosomes; 6 pairs of macrochromosomes and 11 pairs of microchromosomes. Karyologically it is possible to differenciate this species because the pair No. 2 is metacentric (*m*) in *L. pictus* and submetacentric (*sm*) in *L. cyanogaster*. It is shortly discussed the signification of formule $2n = 34$ for the species of *Liolaemus* analized karyologically and its possible mechanism of acquisition.

There is little chromosome information concerning the South American lizards of the genus *Liolaemus*. The only known species is *Liolaemus lutzae* from Brazil ($2n = 34$)[1], of which only the chromosomes of one male individual have been reported. In the present paper, new karyological data about lizards of the genus are given. The chromosomes of *Liolaemus pictus* and *Liolaemus cyanogaster*, two species of South Chile, are presented for the first time. Both species overlap geographically (Concepción to Chiloé) and exhibit a spectrum of morphological, ecological and physiological adaptations to certain habitats (forests and meadows). The two species are different in details of scutellation and colour patterm. The classification of *L. pictus* and *L. cyanogaster* follows that of DONOSO-BARROS[2]. Chromosomes were obtained from bone marrow, testes and leukocytes of young animals previously injected with colchicine 0.1%. The chromosome preparations were made according to methods developed by FORD and HAMERTON[3] (bone marrow and testes) and BAKER et al.[4] (culture of whole blood) and stained afterwards with Giemsa solution. Chromosome classification (only macrochromosomes) according

[1] G. C. GORMAN, L. ATKINS and T. HOLZINGER, Cytogenetics *6*, 286 (1957).

[2] R. DONOSO-BARROS, *Reptiles de Chile* (Ediciones de la Universidad de Chile, Santiago de Chile 1966).

[3] C. E. FORD and J. L. HAMERTON, Stain. Techn. *31*, 247 (1956).

[4] R. J. BAKER, J. J. BULL and G. A. MENGDEN, Experientia *27*, 1228 (1971).

to centromeric position was made on basis of arm-radio, and the nomenclature of LEVAN et al.[5] was adopted. A total of 63 metaphases – 28 from *L. pictus* and 35 from *L. cyanogaster* – were analyzed for chromosomic count. Additional 91 primary spermatocytes at diakinesis – 15 from *L. pictus* and 56 *L. cyanogaster* – were examined. The karyotypes of both species were made with chromosome cuts from enlarged photographs of c-metaphase plates. In order to compare the chromosomes of both species, macrochromosomes were measured – microchromosomes are very difficult to measure – and their lengths normalized according to BOGART[6]. The results of the macrochromosome measurements are included in the Table, and the idiograms constructed from the data in the Table are presented in Figure 2. The lizards analyzed were deposited in the collection of reptiles at the Institute of Zoology of the Austral University, Valdivia (IZUA). The following are the register numbers, sex and geographical locality where the lizards were collected: *L. pictus*; 5 males (IZUA-R 529–531, 536, 538) and 2 females (IZUA-R 629–630); near volcán Llaima (Cautín Province). *L. cyanogaster*; 4 males (IZUA-R 463, 469,

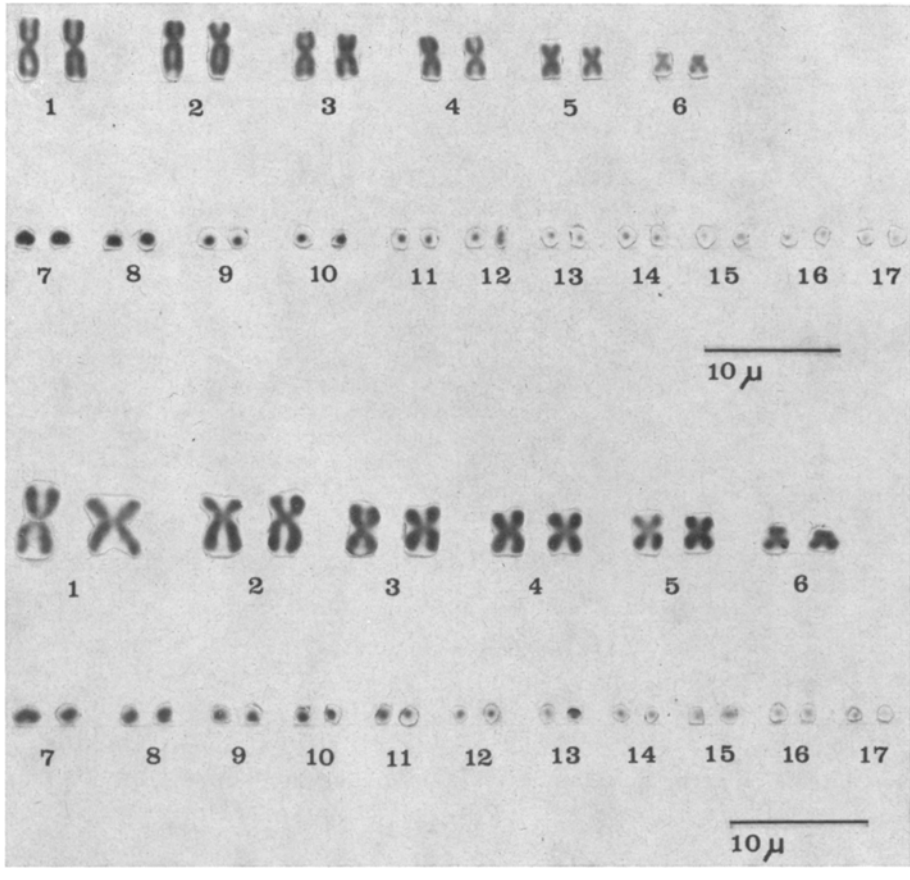

Fig. 1. Karyotypes of *L. pictus* (a) and *L. cyanogaster* (b).

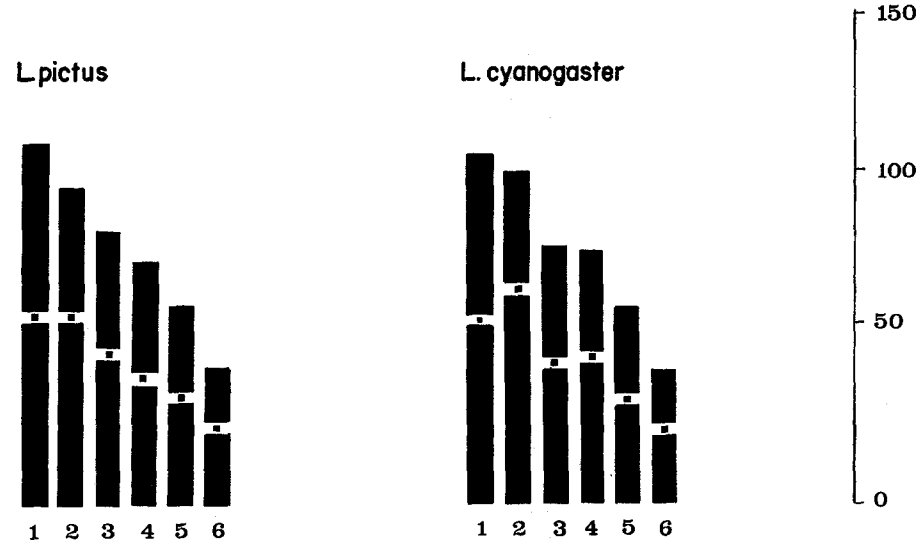

Fig. 2. Idiograms of macrochromosomes of *Liolaemus* species. The scale refers to the normalized length. Idiograms constructed according to BOGART[6].

Analysis of haploid macrochromosome complements

Species of		Macrochromosome No. 1	2	3	4	5	6
Liolaemus pictus	length	117	112	86	79	64	42
	ratio	1.11	1.70	1.21	1.15	1.15	1.49
	type	*m*	*m*	*m*	*m*	*m*	*m*
L. cyanogaster	length	115	109	87	80	64	43
	ratio	1.13	1.86	1.12	1.12	1.15	1.30
	type	*m*	*sm*	*m*	*m*	*m*	*m*

The value for the arm ratio is determined by dividing the long arm by the short arm; type designates the chromosome as determined by the position of centromere (*m*, metacentric; *sm*, submetacentric); length refers to the normalized length of the macrochromosome complement.

664, 790) and 3 females (IZUA-R 464, 637, 638): near Valdivia city (Valdivia Province). *Liolaemus pictus*. This species has a diploid number of 34 chromosomes; these can be arranged in pairs and numbered in order of decreasing length (Figure 1 a). There are 6 pairs of metacentric (*m*) macrochromosomes and 11 pairs of microchromosomes, pair 7 being considerably larger than the others. In the testicular cells (diakinesis) we found 6 large bivalents and 11 microbivalents.

Liolaemus cyanogaster. The karyotype (Figure 1 b) is essentially the same as *L. pictus*, except that in *cyanogaster* chromosome pair number 2 (macrochromosome) is submetacentric (*sm*). In diakinesis there are 6 macrobivalents and 11 microbivalents. Thus, besides known morphological differences in both species, karyologically they are distinct at the level of No. 2 chromosome (metacentric in *L. pictus* and submetacentric in *L. cyanogaster*).

The karyological pattern of the lizards of the genus *Liolaemus* examined (*L. lutzae*[1], *L. pictus* and *L. cyanogaster*) is very similar. The three species present the formula $2n = 34$ and the karyotypes show 6 pairs of macrochromosomes and 11 pairs of microchromosomes, of which the first pair is very 'large'. Within the family Iguanidae, a similar formula ($2n = 34$) and karyotype (6 macrochromosomes and 11 microchromosomes) is found also in some species of *Anolis* (*r. roquet, r. extremus, aeneus*[7], *vermiculatus*[8]) and the lizards of the genera *Uma* (*notata*[1]) and *Scelophorus* (*orcutti*[9], *chrysostictus, maculatus, utiformis, gadavidae*[10,11], *nelsoni, pyrocephalus*[11]). According to GORMAN et al.[1], a karyotype consisting of 6 pairs of macrochromosomes and 12 pairs of microchromosomes ($2n = 36$) is considered primitive for the family Iguanidae. The species of *Liolaemus* (*lutzae*[1], *pictus, cyanogaster*) differ from this karyological patterns by having only 11 pairs of microchromosomes. Reduction in chromosome number appears to be the evolutionary trend in iguanids[7]. If this is true, *Liolaemus* species would have derived karyological forms of the primitive formula ($2n = 36$). The presence of a pair of 'large' microchromosomes (pair 7) in *L. lutzae* has made GORMAN[1] believe in the existence of centric fusion among two of these pairs. We are in agreement with this hypothesis in order to explain the occurrence of formula $2n = 34$ in *L. pictus* and *L. cyanogaster*, since in these two species also the chromosomic pairs No. 7 are very 'large'.

[5] A. LEVAN, K. FREDGA and A. SANDBERG, Hereditas *52*, 201 (1964).
[6] J. P. BOGART, Copeia *3*, 728 (1974).
[7] G. C. GORMAN and L. ATKINS, Syst. Zool. *16*, 137 (1967).
[8] G. C. GORMAN and L. ATKINS, Herpetologica *24*, 13 (1968).
[9] CH. J. COLE, Am. Mus. Novitates *2431*, 1 (1970).
[10] CH. J. COLE, Am. Mus. Novitates *2450*, 1 (1971).
[11] CH. J. COLE, Herpetologica *27*, 1 (1971).

A Possibility to Achieve Genetic Transformation in the Platyfish-Swordtail System *(Platypoecilus maculatus – Xiphophorus helleri)*[1]

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Summary. In order to determine how informative homologous donor DNA might be made available to propigment cells of the recipient *Xiphophorus helleri* for transformation, labelled heterologous DNA from *E. coli* was injected into the neural crest region or the yolk sac of embryos of the recipient. On the basis of the degradation rate of the donor DNA and the incorporation rate of radioactivity into the recipient DNA, it is concluded that injection into the neural crest region may be a suitable method to make available informative homologous donor DNA for transformation.

Since AVERY, MACLEOD and MCCARTY[2] originally observed genetic transformation after treatment of pneumococcal recipient strains with DNA from donor strains, many attempts have been made to achieve genetic transformation also in higher organisms. A variety of genetic alterations has been obtained[3]; it has been argued, however, that they might not be due to transformation.

One objection against interpreting these alterations as the result of a transformational event has been that DNA mediated mutations or conversions[4] of the already present allele[5] might have pretended genetic transformation. This objection cannot be brought against the transformation system we have chosen, in which the platyfish (*Platypoecilus maculatus*) is the *donor*, carrying up to 12 different luxurious loci for distinct pigment cell patterns, and the swordtail (*Xiphophorus helleri*) is the

[1] Supported by DFG through SFB No. 103, and by Stiftung Volkswagenwerk.
[2] O. T. AVERY, C. M. MACLEOD and M. MCCARTY, J. exp. Med. *79*, 137 (1944).
[3] J. M. OLENOV, Int. Rev. Cytol. *23*, 1 (1968). – L. LEDOUX, *Informative Molecules in Biological Systems* (North-Holland Publ. Co., Amsterdam/London 1971); *Uptake of Informative Molecules by Living Cells* (North-Holland Publ. Co., Amsterdam/London 1972). – D. HESS, Naturwissenschaften *59*, 348 (1972); Umschau *75*, 501 (1975).
[4] Mutations could be demonstrated in *Drosophila* after treatment with DNA (O. G. FAHMY and M. J. FAHMY, Nature, Lond. *196*, 873 1962), and are known to be induced by degradation products of DNA (J. J. BIESELE, R. F. BERGER, M. CLARKE and L. WEISS, Expl Cell Res., Suppl. *2*, 279 (1952). – B. A. KIHLMAN, Expl Cell Res. *25*, 694 (1961); J. cell. comp. Physiol. *62*, 267 (1963).
[5] All transformation experiments were done so far on the basis of the replacement of one allele by another.