Chez les animaux préalablement immatures, au bout de 5 semaines à 33 °C, l'observation histologique révèle que l'ovogenèse n'est pas sensiblement modifiée par rapport aux témoins.
On note l'abondance à 33 °C de gros ovocytes en dégénérescence. Pour la spermatogenèse, les stades postérieurs à la prophase méïotique sont peu abondants. La vésicule séminale ne contient pas de spermatozoïdes. Au bout de 14 semaines on trouve toujours des spermatides âgées et des spermatozoïdes dans la gonade.
Les animaux de la 3e série d'expériences, sacrifiés au bout de 5 mois à 33 °C montrent la totalité des stades des 2 gamétogenèses. La vésicule séminale contient des spermatozoïdes.

Tractus génitaux. L'étude pondérale de la gonade et des tractus génitaux à différentes températures donne les résultats suivants:
La dissection comme l'étude pondérale ne montre pas de réduction de la glande de l'albumine; au contraire, on constate qu'elle est significativement plus importante chez les adultes à 33 °C que chez les témoins. A 30 °C et à 33 °C il en est également de même chez les juvéniles. Chez ces derniers, de plus, la partie femelle a un poids supérieur à celui des témoins.

Discussion. Une température constante de 33 °C stérilise totalement les *Biomphalaria glabrata* adultes ou immatures. Ce blocage de la reproduction dure tant que la température reste élevée. Quelques jours après le retour à une température plus basse, les planorbes deviennent ou redeviennent fertiles.
La température élevée n'empêche pas le développement de l'appareil génital. Les différentes catégories de cellules sexuelles apparaissent; les diverses parties des tractus génitaux se différencient et se développent mais elles ne fonctionnent pas.

Quels que soient les animaux utilisés, contrairement aux observations de Michelson[3], il n'y a aucune régression de la glande de l'albumine; au contraire, son poids relatif par rapport au corps est plus élevé à haute température. Chez les immatures, on observe aussi un plus grand développement de la partie femelle.
Contrairement à Michelson[3], il apparait que l'ovogenèse n'est pas notoirement affectée par une élévation de la température, tout au plus peut on noter l'abondance des gros ovocytes en dégénérescence.
En revanche, chez les immatures et les adultes, les stades postérieurs à la prophase méïotique de la spermatogenèse semblent affectés par une haute température et sont peu abondants. Cependant des spermatozoïdes se différencient si l'animal est soumis à 33 °C dès la fécondation.
Des individus dont l'ovotestis renferme tous les stades des gamétogenèses et dont les tractus sont normalement développés peuvent rester stériles plusieurs mois. Une température élevée n'empêche donc pas la différenciation de l'appareil génital mais elle en pertube cependant le fonctionnement jusqu'à stériliser totalement les animaux.
Il est probable que l'action de la température ne s'effectue pas directement sur les gamétogenèses et les tractus. On ne peut écarter l'hypothèse de l'existence d'un relais au niveau du système nerveux central mais ceci reste encore actuellement à démontrer.

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Determination of adhesive rate constant in normal and neoplastic homogeneic cells[1]

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Summary. The adhesive rate constant (ARC) of neoplastic SGS-2 cells which have a low contact inhibition, is remarkably higher than that of normal homogeneic fibroblasts. This is in contrast with the mutual adhesion theory which states that the loss of contact inhibition is strictly related to the loss of cell recognition and consequently to the loss of cell adhesion capacity.

Cell recognition and cell-cell adhesion are important phenomena involving certain components of the cell membrane. Intercellular contact induces a series of events which lead to specific cellular differentiation by regulating mitosis and gene expression. Thereby histo- and organogenesis are under control. In a normal tissue, contact inhibition, with its manifold aspects like inhibition of movement, mitosis, growth and density dependence, hinders an uncontrolled cellular proliferation and guarantees a physiological and harmonious development of tissues and organs. This control mechanism is lacking or is defective in most neoplastic cells. The consequence is an anomalous cell proliferation which determines the invasive properties of neoplasias in loco and at a distance. One possible explanation[3,4] is that the loss of contact inhibition observed in a number of neoplastic cells is caused by the loss of mutual recognition capacity and consequently of their adhesive properties. According to the mutual adhesion theory of Gail and Boone[5] the molecular mechanism of cell-cell adhesion is very similar if not identical to that which regulates contact inhibition. Irrespective of this theory, there are several experimental observations which clearly show that the adhesion capacity of certain neoplastic cells, either derived from spontaneous tumors or transformed by oncogenic viruses, is very low. Conversely, we found that SGS-2 cells, a neoplastic cell strain actually investigated in our laboratories[6], have a remarkably higher adhesion than that reported for other neoplastic cells in non-specific cell-substrate ad-

hesion experiments[5,7-11], and in specific cell-cell adhesion experiments[12-15]. Since it was impossible to establish a priori wether the high ARC value was due to an unusually high adhesive capacity of our cells or rather to the method employed, we performed a study based on a direct comparison between the ARC of neoplastic cells and normal fibroblasts having a common origin.

Materials and methods. A neoplastic cell strain (SGS-2: sarcoma galliera strain-explant 2)[16] is derived from a solid sarcoma of the rat (sarcoma galliera)[17]. This neoplasia, first observed in 1920[18], has been maintained until now by means of homoplastic implants of the sarcomatous tissue into the same animal strain (*Rattus norvegicus*, var. *albus*, galliera strain) in which the tumour originally developed[18]. Also the SGS-2 cells can induce such neoplasia into the above mentioned rat strain[19]. The fibroblast cell strain (FG: fibroblasts galliera) is derived from embryos of the same rat strain. Cells were grown in Dulbecco's modified Minimum Essential Medium supplemented with 10% newborn calf serum (Gibco). Adhesion tests were performed by the method of Walther et al.[12] modified according to the properties of SGS-2 cells[6]. This method belongs to the category in which cell-cell adhesion is measured and consists of determining the percent of single cells, labeled with ^{3}H-leucine, adhering to a confluent monolayer. Normal and transformed single labeled cells were released from the monolayer with 0.25% trypsin in solution A (Gibco) and incubated at 37 °C for 7 min.

Results and discussion. The results obtained are summarized in the table and figure 1. The time-dependence of a typical cell-cell adhesion test with neoplastic cells and normal fibroblasts is shown in figure 2. The fluctuation on the data is rather high; however, it lies in the range observed with normal and neoplastic cell lines by Walther et al.[12]. But the difference between the ARC values of the 2 homogeneic strains is highly significant.

Most of the data reported in the literature show that neoplastic and transformed cells have e markedly reduced capacity for specific and non-specific adhesion when compared to normal cells. It has been suggested that this behavior is due either to a lower biosynthesis[20] or to an increased degradation[21] of fibronectin. The fact that a poor adhesive capacity is accompanied by a reduced contact inhibition (particularly of mitosis and cell movements) is tentatively explained by the mutual adhesion theory of Gail and Boone[5]. This theory assumes that there is a close relationship between cell adhesion and contact inhibition. However, our results are in complete contrast with this theory by showing a higher adhesion in tumoral cells than in homogeneic normal cells. In fact SGS-2 cells, in spite of a low contact inhibition, have a strong mutual adhesion capacity, whereas homogeneic fibroblasts, though they have a marked contact inhibition, show normal adhesiveness (figs 1 and 2). On the other hand, our observations agree with the data of Walther et al.[12] which show higher ARC in SV 3T3 than in normal 3T3 cells. At present we have no explanation for the discrepancies discussed above; however, there are at least 2 possibilities. Adhesion is measured with essentially different methods, some are based on specific cell-cell adhesion tests, whereas the others are based on non-specific cell-substrate adhesion measurement, and it is likely that each group of methods detects different molecular mechanisms on the cell surface. Alternatively, the differences observed may be due to intrinsically different properties of the cells investigated.

Adhesive rate constant (ARC) of normal (FG) and neoplastic cells (SGS-2)

Cells	Experiment number	ARC	
FG	9	0.145	±0.098*
SGS-2	20	0.545	±0.150

* Values are means ± SD.

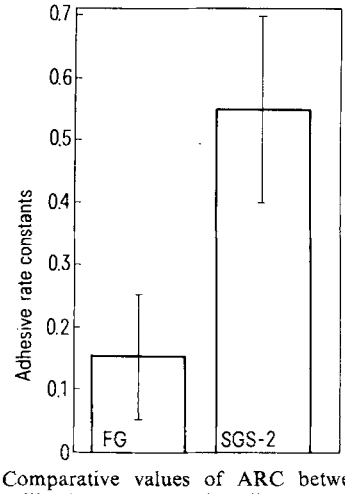

Figure 1. Comparative values of ARC between normal (FG, fibroblasts galliera) and neoplastic cells (SGS-2, sarcoma galliera strain). ARC values were calculated from the linear portion of the cell-adhesion versus time curve. In each experiment 0.33 ml of single cell suspension (8×10^4-1.2×10^5 cells/ml) were added to 1 cm^2 of homologous cell monolayer and incubated at 37 °C.

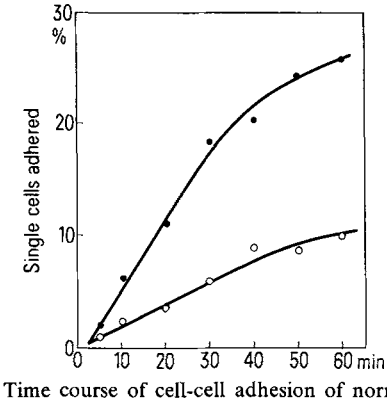

Figure 2. Time course of cell-cell adhesion of normal fibroblasts (FG, ○) and neoplastic cells (SGS-2, ●). The adhesion is expressed as percent of single labeled cells adhering to a homologous monolayer. Experimental conditions were as described in figure 1.

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A novel biochemical systematic technique for herpetology based on epidermal keratin[1]

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Summary. Using green geckos *(Phelsuma)* the molecular weights and isoelectric points of S-carboxymethylated keratin monomers from fresh and preserved reptile epidermis were investigated. Isoelectric focussing reveals that several of the circa 35 components differ between genera, species and races and that the relative similarity of the SCMK pattern is directly related to evolutionary similarity and is of considerable taxonomic value.

Biochemical systematics of reptiles and other animal groups depends largely on proteins obtained from live specimens to supply 'genetic' or molecular information. This has several disadvantages in that it is often impracticable to adequately study widely distributed species because of the difficulties in obtaining geographically comprehensive samples of live animals. It can also exclude the study of endangered, extinct and, in the case of reptiles, the many dangerously venomous species. Keratin, a structural protein, is an extremely stable polymer which can be obtained from the epidermis of preserved museum specimens as well as from freshly shed skin of reptiles. Using green geckos *(Phelsuma)* as a model, this paper shows how S-carboxymethylated components of epidermal keratin (both fresh and preserved) can be characterized by isoelectric focusing on specially formulated gels to give useful taxonomic information, particularly at the interspecific level. The use of keratin from museum specimens may overcome many of the above problems associated with the use of live specimens and thus be of considerable value to herpetological taxonomy.

The S-carboxymethylated keratin (SCMK) components are prepared in the following way. Shed skin is thoroughly washed in detergents and organic solvents and is then reduced by magnetic stirring for 12 h in thioglycollic acid, EDTA, and 8 M urea in a nitrogen atmosphere. The samples are alkylated with iodoacetic acid at pH 9.6[2]. The SCMK sample is then dialyzed against distilled water and concentrated with an Amicon multimicrofiltration unit using a Diaflo YM5 ultrafiltration membrane.

The SCMK sample could be split (before concentration) into high and low sulphur fractions by precipitating out the low sulphur fraction with zinc acetate and redissolving with sodium citrate[3].

The molecular weights of the SCMK components were determined by comparing their mobility with that of standard markers (BDH No.44262L) when run on SDS 11% polyacrylamide disc gels following the Laemmli procedure[4]. The high sulphur SCMK components show (fig. 1) 3 bands of low mol.wt (16–20 kdaltons) whilst the low sulphur components are revealed as being of more diverse mol.wt (one group at 11–20 kdaltons and another group at 32–69 kdaltons).

These SCMK components can be characterized by PAG or gradient PAG electrophoresis[5–9] but we obtained far better results for reptile keratin with isoelectric focusing (IEF).

The IEF gels were prepared from 5% acrylamide, 0.15% NN - methylene-bisacrylamide, and 8 M urea as well as 2% pH 2.5–4, 2% pH 4.0–6.0, and 1.7% pH 5.0–8.0 LKB Ampholines. The SCMK sample and both the anolyte and

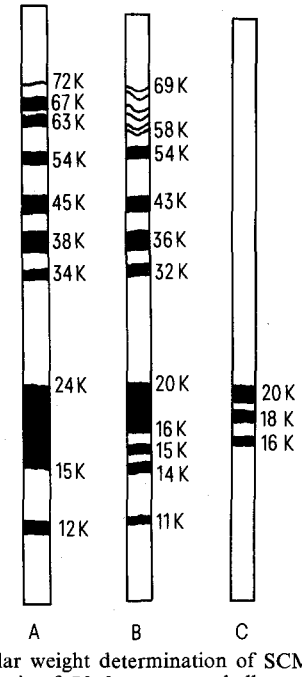

Figure 1. Molecular weight determination of SCMK components from fresh epidermis of *Phelsuma a* seychelles species. The SDS gels are drawn to scale with the computed[13] mol.wts indicated in daltons (taking into account marker dye position and gel shrinkage etc.). A, Unfractionated sample; B, low sulphur fraction; C, high sulphur fraction. Wyld and Brush[14] investigated molecular heterogeneity of keratin in a range of reptiles but do not give mol.wts for epidermal keratin of geckos or other lizards.