Infolge einer Aktivierung der Nebenschilddrüsen durch die experimentelle Calcitoninzufuhr[11,12] dürfte auch das Parathormon bei der Aktivitätsverminderung der Isocitrat-Dehydrogenase[13] in den Pinealzellen eine Rolle spielen. Somit schliessen wir aus unseren Befunden, dass das Calcitonin[14] nicht nur direkt, sondern auch indirekt eine hemmende Wirkung auf die Zirbeldrüse hat.

Résumé. Après injection, pendant 2, 4 et 8 semaines, de 300 MRC microunités de Calcitonine porcine à des rats Wistar mâles de 250 g, on a constaté, par des méthodes histoenzymologiques, une diminution de l'activité des glandes pinéales.

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The Electrophoretic Mobility of Somatic Cell Hybrids

Alterations of the cell surface electric charge arizing from the removal or addition of ionogenic groups can be detected in a quite satisfactory manner by utilizing the technique of cell electrophoresis. Changes of this nature are usually expressed as changes in the cell electrophoretic mobility and surface charge density. Since the electrophoretic mobility and surface charge density reflect net changes at the surface (the quantity and quality of which may be controlled by the cell genome), it was of interest to explore the surfaces of interspecific somatic cell hybrids and their parental lines. It should be noted that hybrids are particularly suitable for these type of studies since 1. both parental sets of chromosomes are functional in the hybrid, and the hybrid, therefore, exhibits the hereditary characteristics of both parent cells, and 2. the hybrids lose some of their chromosomes, which permits assignment of single gene-products to specific chromosomes.

The aim of the present study was to compare the electrokinetic behavior of hybrids between human diploid skin fibroblasts and aneuploid mouse, or hamster cells, with the electrokinetic behavior of their parental lines. Since chromosomal studies have revealed that most of the human chromosomes in these hybrids have been lost, it was of interest to determine whether an interspecific hybrid carrying only a few human chromosomes would exhibit altered electrokinetic behavior.

Materials and methods. The electrokinetic behavior of hybrids between human diploid skin fibroblasts (strains: Ms58, Ms63, and Ms64) and aneuploid mouse (cell line A 9), or Syrian hamster (cell line TG 2, kindly provided by Dr. Littlefield) cells (Table), was compared with the electrokinetic behavior of either of their parents. The cells were hybridized by Drs. M. Siniscalco and K. H. Grzeschik utilizing inactivated Sendai virus. The hybrids were selected in medium containing hypoxanthine, aminopterin, thymidine, and glycine (HAT)[1] and maintained in the same medium for 3–7 months post-hybridization. The parental cell lines were grown in minimal essential medium. Cells were prepared for karyotiping according to conventional methods.

Prior to electrophoresis, confluent cell cultures were rapidly washed in Ca- and Mg-free MEM, and trypsinized for 60–90 sec in 0.25% trypsin at 37° C. The cells were prepared for electrophoretic mobility measurements by washing and resuspending in Earle's balanced salt solution (ionic strength 0.160) at pH 7.2. The cell electrophoretic mobility was measured at 37°C in a cylindrical tube apparatus[2]. The cells were timed across a distance of 13.3 μm in a 3.22 V/cm field gradient when 1.95 mA of current was flowing. The reliability of the instrument was ascertained by calibrating against the constant electrophoretic mobility values for human red blood cells reported in the literature[3].

Results. The results from the electrophoretic mobility measurements (Table) have indicated that the electrokinetic properties of our hybrids were similar ($P > 0.02$), or identical ($P <$, > 0.05) to the electrokinetic properties of their mouse or hamster parental line. In all instances, however, the electrophoretic mobility values for the hybrid cells were significantly different ($P < 0.01$, or 0.001) from the values determined for their human parental lines.

Discussion. Karyotype analyses have indicated that the interspecific hybrids utilized in this study have lost most of their human chromosomes and have kept virtually the entire or the double genome of the mouse or hamster (Table). This spontaneous elimination of human chromosomes is a well established trend affecting interspecific hybrids between human and mouse, or hamster cell populations[4,5].

The true nature of our hybrids, however, was indicated by the fact that the cells were able to grow in HAT medium, for which the presence of the human *X*-chromosome is necessary[1]. In this connection, the enzyme analyses performed on all hybrid types indicated the presence of glucose 6-phosphate-dehydrogenase and 3-phosphosylcerate kinase of the human type. This confirmed the presence of the human *X*-chromosome in the hybrids, to which the genes associated with the production of the above enzymes are linked. Furthermore, upon karyotype analysis, hybrids B82/Ms2 and TG 2/Ms63 revealed the presence of 18 and 2 autosomes, respectively, in addition to the mouse or hamster genomes (Table).

Our preliminary results (Table) tend to indicate that significant changes in the electrokinetic properties of the interspecific hybrids between human and mouse, or hamster, cell populations may not be brought about by the presence of a few human chromosomes in the hybrid genome (although in some instances $P > 0.02$). Studies on somatic

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Cell types	Cell origin	Genetic deficiency	Electrophoretic mobility (μm/sec/V/cm) (No. of observations) ± standard error	*P* value	Mean number of chromosomes
MS2	Human skin fibroblast	HGPRT-	−1.39 (76) ± 0.015	< 0.01	46
B82/MS2	Clonal hybrid line		−1.25 (71) ± 0.014		2×53+19
B82	L929 mouse fibroblast	TK-	−1.15 (63) ± 0.018	< 0.05	53
MS58	Human skin fibroblast	balanced translocation *X*/chr 14	−1.13 (57) ± 0.019	< 0.001	46
TG2/MS58	Clonal hybrid line		−1.29 (88) ± 0.020	> 0.05	2×48+1
TG2	Syrian hamster BHK fibroblast	HGPRT-	−1.24 (83) ± 0.014		48
MS64	Human skin fibroblast	Hydrocephalus	−1.38 (67) ± 0.012	< 0.01	46
A9/MS64	Clonal hybrid line		−1.52 (75) ± 0.013	< 0.05	57+1
A9	L929 mouse fibroblast	HGPRT-	−1.63 (59) ± 0.024		56
MS63	Human skin fibroblast	G6PD- mediter. variant	−1.37 (36) ± 0.017	0.02	46
TG2/MS63	Mixed hybrid population		−1.28 (68) ± 0.023		2×48+3
TG2	Syrian hamster BHK fibroblast	HGPRT-	−1.24 (83) ± 0.014	$0.02 > P < 0.05$	48
MS58	Human skin fibroblast	Balanced translocation *X*/chr 14	−1.13 (57) ± 0.019	< 0.001	46
A9/MS58	Clonal hybrid line		−1.57 (65) ± 0.016		56+1
A9	L929 mouse fibroblast	HGPRT-	−1.63 (59) ± 0.024	> 0.02	56

HGPRT-, hypoxanthine guanine phophoribosil transferase deficient; TK-, thymidine kinase deficient; G6PD-, glucose-6-phosphatedehydrogenase deficient

cell hybrids, however, have indicated that surface antigens and T-antigens are expressed in the hybrids when carried by one of the parents[6], and their synthesis in the hybrids depended on the number of chromosomes from the parental line carrying the marker[4]. In this connection, if the electrokinetic properties of the hybrids studied can be associated with products of genes on a certain chromosome or chromosomes, our results indicate that such chromosomes have probably been eliminated. Studies on early cell hybrid populations, which carry a larger number of human chromosomes but are low in cell numbers, were not made, since the electrophoretic mobility measurements required a larger number of cells than available.

Résumé. Nous avons comparé e comportement électrocinétique des cellules hybrides des fibroblastes diploïdes de la peau humaine et aneuploïde d'une souris ou d'un hamster avec celui des cellules de leurs lignées. Les résultats montrent que les changements significatifs observés dans les propriétés électrocinétiques des hybrides interspécifiques ne peuvent pas être dus à la présence de quelques chromosomes humains dans le génôme hybride.

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Chromosomal Pattern of Asparaginase Sensitive Leukemia and its Resistant Variant

The asparaginase resistant line of EARAD1 leukemia which is originally sensitive to L-asparaginase therapy, was produced by repeated passages in hosts treated with suboptimal doses of asparaginase and subsequently carried out in untreated hosts[1]. The resistant line shows a high degree of resistance to L-asparaginase. Asparagine synthetase activity, which is supposed to be responsible for the development of resistance, is slightly higher in the resistant tumour[1–6]. The present report deals with the gross chromosomal alteration which may be a reflection of genetic change from asparaginase sensitivity to asparaginase resistance, in a asparaginase sensitive leukemia and its resistant variant.

Materials and method. The studies were carried out with a radiation induced and asparaginase sensitive transplantable ascites leukemic tumour (EARAD1) and its resistant variant (EARAD1-Res) in both sexes of isogenic hybrid mice (C57BL/6×A)F1. The size of inoculum is 1×10^6 leukemic cells in 0.5 ml per mouse. We

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