"developed" silver chloride electrode of similar properties. But recent tests have shown it to have an impedance range several times higher and a "coulomb range" considerably smaller than the platinized product has. Also in one case the layer of reduced silver fell off after electrolytic removal of the underlying chloride.

No specific explanations of the structure or operation of this hybrid electrode are offered, and no more detailed description or further investigations of its properties are planned.

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Immunological Competence of Placenta

Abstract. The presence of immunologically competent cells in placenta has been investigated by use of the experimental model of runt disease in inbred strains of mice. The injection of placental cells from C57/Bl mice into newborn Balb/C mice resulted in death in the 2nd to 4th week. Evidence is presented that death was caused by immunologically competent fetal cells.

Interest in the mechanisms of transplantation rejection has recently intensified. This has focused attention on what appears to be a remarkably successful homograft, the placenta.

In considering the immunological relations about the placental site, we first investigated whether placental tissue possesses antigens that stimulate tissue rejection. Using inbred strains of mice, we found that the injection of placenta cells from one strain into another induced accelerated rejection of a subsequent homologous skin graft and also the formation of cytotoxic antibodies (*1*). Uhr and Anderson, using a similar approach, reached the same conclusion (*2*).

The next question to be investigated was the presence of immunologically competent cells in placenta. This was undertaken by using the recently described "runt syndrome" as a model. The runt syndrome, characterized by slow growth and death, is produced by the injection of immunologically competent cells from one strain of mice into homologous newborn mice (*3*). The generally accepted explanation is that the newborn mouse is incapable of rejecting the transplanted cells, which survive and in effect "reject" the host, producing an immunological disease. The subject of this report is the results of experiments in which transplants of placenta cells were used.

Mice (C57/Bl and Balb/C) were obtained from the Jackson Memorial Laboratories. A breeding colony of each strain was maintained in our laboratories. Within 24 hours of the birth of a Balb/C litter, a C57 mouse estimated by size to be 17 to 20 days pregnant was selected, and an experiment was performed.

Cell suspensions were prepared in the customary manner, and 10 million cells of various tissues were injected intraperitoneally into newborn Balb/C mice. In preparing placenta cell suspensions, the maternal decidua was first carefully dissected away under a magnifying lens. The viability of the cell suspensions was checked with 0.1 percent eosin Y.

Each Balb/C litter was divided into two to four groups of two to three animals each, depending on the size of the litter; each group received a different type of cell suspension. This permitted a controlled comparison of results within each experiment. For example, in a litter of nine, three animals received C57 spleen cells, three received C57 liver cells and three received C57 placenta cells. The results of all experiments were so consistent that they have been pooled for presentation (Table 1).

A few deaths occurred in all experimental animals during the first few days. The deaths were undoubtedly related to the problems of birth and possibly to the trauma of the experimental procedure. Thereafter, no deaths occurred until after the first week—the latent period common in primary immunological reactions and in runt disease. After the first week, the deaths were limited almost entirely to those animals that had received injections of C57 placenta or spleen cells.

The results obtained with C57 placenta cells closely paralleled those obtained with C57 spleen cells, suggesting a mechanism common to both. However, the results with placenta were sufficiently unexpected to warrant a series of control experiments to exclude other possible explanations. Three major possibilities were considered other than immunological disease resulting from competent placenta cells. These were infection, the release of a lethal pharmacological agent by surviving cells, and the contamination of the cell suspension by competent maternal cells. The design of many of these experiments was suggested by previous work on runt disease (*3, 4*).

The possibility of infection is rendered unlikely by the survival of Balb/C newborn mice following the injection of C57 liver cells, placenta homogenate, or the combined injection of adult Balb/C spleen cells and C57 placenta cells. As a further check, a series of newborn Balb/C mice that had received intact placenta cells were sacrificed a week later, and suspensions of their livers and spleens were injected into another group of Balb/C animals without ill effect. It would be anticipated that an infectious agent would be detected by such serial transfer.

The second possibility, that of release of a lethal toxin by injected placenta cells, is excluded by the survival of Balb/C newborn mice receiving F1 pla-

Table 1. Effect of various cell suspensions in producing runt disease and death in newborn Balb/C mice. The number of animals that died are listed by age in days at the time of death. There were no deaths during the period 4 to 9 days. Fourteen newborn Balb/C mice received F1 placental suspensions (4 from C57 ♂ × Balb/C ♀ matings, 10 from Balb/C ♂ × C57 ♀), and all survived.

Age at death (days)	No. of deaths with suspension indicated					
	C57 spleen	C57 placenta	Hank's sol.	C57 placenta homogenate	C57 liver	Balb/C spleen + C57 placenta
0–3	3	4	1	1	2	
10–12	6	5				1
13–15	2	6				
16–18	1	5				
19–21	4	6				
22–24	1	2				
Living at 25 days	4*	0	6	11	7	9

* All were much smaller (runts) than controls.

centas derived from matings of either C57 males with Balb/C females or Balb/C males with C57 females. On the other hand, their survival is compatible with immunological theory. The injection of F1 cells into the newborn of either parental strain should result in surviving cells that do not cause runting (*3*).

A final consideration was the possibility that the lethal effect of C57 placental suspensions was produced by small numbers of immunologically competent maternal cells present in the suspensions. Although the maternal tissues had been carefully dissected away before preparing the placental suspensions, it was of course impossible to exclude all maternal cells. To clarify this situation, Balb/C males were mated with C57/B1 females. The placenta was therefore F1 and incapable of causing runting; however, the maternal tissue was pure C57 in origin. If the lethal results of the previous experiments with placenta cell suspensions had been caused only by contaminating maternal cells, this preparation should also cause death. In fact, omitting deaths occurring during the first 3 days of life, in 10 of 10 experimental animals receiving placental suspensions all survived. However, the injection of spleen cells from the mothers bearing these placentas resulted in death in 12 of 13 animals, and the surviving animal was obviously runted. This demonstrated that if a sufficient number of immunologically competent *maternal* cells had been present in the placental suspensions, death should have resulted. The only reasonable explanation of the previous experiments is that placenta does contain immunologically competent fetal cells. It is evident that this makes even more remarkable the success of the placenta as a homograft (*5*).

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Dispersal Patterns of Pleistocene Sands on the North Atlantic Deep-Sea Floor

Abstract. Glauconitic, quartzose sands previously modified on the continental shelf from feldspathic glacial detritus were transported through submarine canyons onto the Hudson deep-sea fan, the Hatteras abyssal plain, and the western and central Sohm abyssal plain. These feldspar-poor, quartzose sands contrast with highly feldspathic sands derived directly from a glacial source and probably transported through the Newfoundland abyssal gap onto the eastern and southern Sohm abyssal plain.

The discovery of sand layers of Pleistocene and Recent age interbedded with deep-sea clays and oozes is one of the surprising and important discoveries of recent oceanographic research. On the physiographic map of the floor of the northwestern North Atlantic (*1*) is shown the location of 50 long piston cores containing Pleistocene (Wisconsin) sand layers which have been analyzed for their mineral composition and texture (Fig. 1). The Wisconsin-Recent boundary in the cores had been previously delineated on the basis of the ratio of cold- to warm-water planktonic foraminifera (*2*). The quantitative mineral composition of the sands was determined in impregnated petrographic thin sections by the point-counter technique. The heavy mineral assemblages (specific gravity over 2.9) were measured quantitatively by the line-count method in separate mounts. Graphic summary statistics (Folk) for mean size, standard deviation, sorting, skewness, and kurtosis were calculated from cumulative grain-size distributions based on sieving and pipette analysis.

Most of the deep-sea Wisconsin sands in the western North Atlantic (Fig. 1) are relatively quartzose, having quartz-to-feldspar ratios often exceeding 90 : 10, omitting calcareous fossils, clayey matrix smaller than 0.03 mm, and miscellaneous grains. These sands are largely very fine-grained to fine-grained, and moderately sorted to moderately poorly sorted (standard deviations of 0.60 to 0.90 predominate). The derivation of these quartzose sands from originally feldspathic glacial detritus is shown by the consistent ratios throughout this large area of various varieties of minerals, and the typical glacial assemblage of many heavy mineral species. Potassium feldspar consistently predominates over plagioclase, and metamorphic quartz (schistose and highly strained quartz plus metaquartzite rock fragments) forms about a third of the total quartz. The transparent, nonmicaceous heavy mineral assemblage is dominated by 20 to 60 percent amphiboles (about one-third blue sodium-bearing metamorphic amphiboles), lesser amounts of garnet, staurolite, tourmaline, hypersthene, sillimanite, and apatite, and traces of many other minerals.

The occurrence of marine sandstones in the Sangamon or Wisconsin inter-

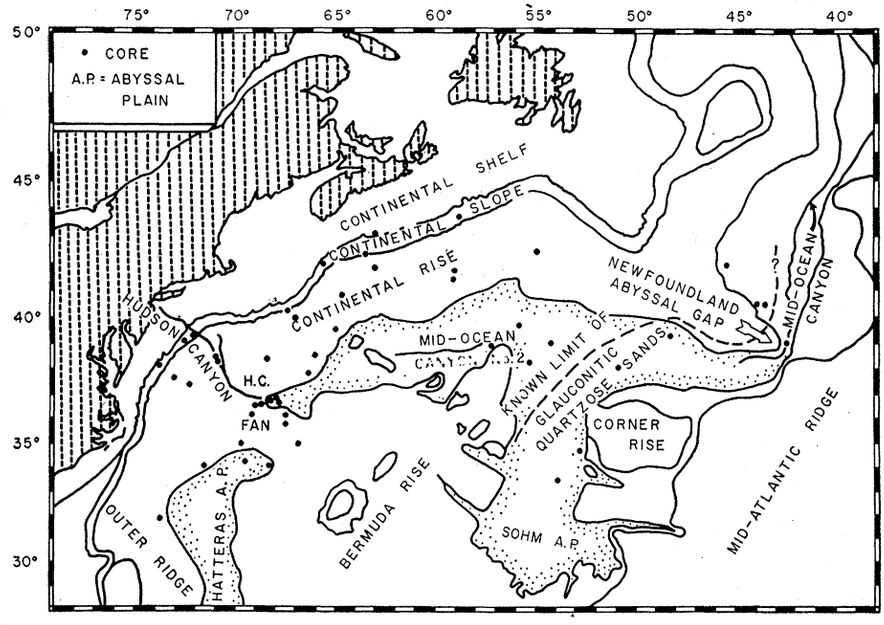

Fig. 1. Location of long piston cores containing Pleistocene (Wisconsin) sand layers which have been analyzed for their mineral composition and texture.