peptides, either alone or in combination, are necessary for the adrenal response to sodium depletion. The results of pituitary gland injections suggest that ACTH and possibly thyrotropin are not necessary for a significant response to sodium depletion.

The pituitary factor is not the primary stimulator of aldosterone secretion, since sodium depletion is also required for a marked effect of the injections of pituitary glands. Furthermore, the width of the zona glomerulosa, the site of aldosterone biosynthesis, is increased in sodium-depleted hypophysectomized rats not receiving pituitary gland injections.

The pituitary gland injections did not stimulate the secretion of aldosterone in sodium-depleted hypophysectomized rats to the same level as in sodium-depleted intact rats. This may indicate that there was an inadequate dose of the pituitary gland injected or that labile pituitary factors are also necessary for the maximum response.

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"Intrinsic" Immunological Tolerance in Allophenic Mice

Abstract. *Mice experimentally derived from pairs of conjoined, undifferentiated, cleavage-stage embryos of different histocompatibility genotypes can retain cells of each strain, which still produce their characteristic antigenic products. The animals are permanently tolerant of cells of both original types, remain free of runt disease, and display a normal and specific immune response to introduction of a foreign antigen. Absence of autoimmunity in development of ordinary animals is explainable by the "intrinsic" kind of tolerance found here.*

The immune rejection of antigenically foreign cells which ordinarily follows their introduction into an organism can be circumvented in at least two ways: by introducing the graft prenatally or early postnatally before immunological maturity (*1*), or by depressing the adult host's immune system [for example, by irradiation (*2*)] before introducing the antigen. Acceptance of grafts in both cases illustrates the phenomenon of acquired tolerance (*1*).

The earliest developmental origins of the immune system are still largely obscure, despite interesting recent investigations of the problem. It is therefore difficult to assess the precise immune status of embryos at any of the stages involved in previous prenatal inductions of tolerance, whether by way of natural vascular anastomoses, transplantation, or parabiosis (*1*, *3*). In no instance is it certain that the host completely lacked primordial cells of the immune system at the time the foreign component was first presented.

The *allophenic* mice experimentally produced by Mintz (*4*) are the first animals in which homologous cell association within the embryo unquestionably predates any immunological differentiation. These single individuals are each of multi-embryo origin. Their precocious genotypic multiplicity is established during the cleavage period, when blastomeres have been demonstrated still to be developmentally entirely labile (*4*). As a consequence, *any* tissue can ultimately consist of different genotypes of cells, related as coevals rather than as host and donor.

The animals are formed by first assembling all the blastomeres from two (or more) genetically distinctive embryos into one composite group in vitro; this is later transferred surgically to the uterus of a pseudopregnant female. Here regulation from double to single embryo size occurs during implantation. Normal development to birth frequently follows and approximately 500 healthy adults, comprising many pairs of genotypes, have been obtained (*4*, *5*). Mice arising in this manner are called *allophenic* because of the orderly coexistence in them of cells with different phenotypes ascribable to known allelic genotypic differences. The two cell populations in a tissue strikingly and invariably occupy nonrandom positions with respect to each other and therefore form consistent patterns. From analyses of such distributions it has been possible to deduce complete clonal ontogenies of several kinds of cells, including melanocytes (*5*, *6*).

Many of the animals are derived from two genetic sources differing at one or more histocompatibility loci, including the *histocompatibility-2(H-2)*-locus which exerts the strongest influence over graft acceptance. Mintz and Palm (*7*) have in fact identified in some allophenics the simultaneous presence of $H\text{-}2^k$ and $H\text{-}2^b$ erythrocytes, by agglutination and absorption methods. In the present study, grafting procedures were used to extend to skin the tests for production of diverse antigens, and also to examine for tolerance and immunity. Three kinds of experiments were conducted: (i) Some allophenic mice were grafted with skin from the original (or "parental") strains, to ascertain whether tolerance existed (Table 1). (ii) Some of the tolerant animals were challenged with skin whose histocompatibility genotype differed from either of the constituent strains, to observe whether such recipients were capable of a normal immune response. (iii) Some allophenics served as donors of skin grafts to the "parental" strains, as a direct measure of formation of the respective antigenic products (Table 2).

In certain of the strain pairs, coat color markers are also included along with *H-2* differences. The four inbred strains involved were C3Hf [genotypically $H\text{-}2^kH\text{-}2^k$ and *AA* (agouti)]; C57BL/6 [$H\text{-}2^bH\text{-}2^b$ and *aa* (*nonagouti*, or in this case black)]; CBA-*T6T6* ($H\text{-}2^kH\text{-}2^k$ and *AA*; also homozygous for the *T6* translocation); and BALB/c [$H\text{-}2^dH\text{-}2^d$ and *cc* (*albino*, which masks other color factors, in contrast to *colored*, or *CC*, in the three preceding strains)]. *AA* mice all have some black hairs and *aa* have

scattered agouti hairs in certain body regions, but the prevailing colors are unmistakable. The pure-strain experimental combinations tested were C3Hf↔C57BL/6, CBA-*T6T6*↔C57BL/6, C3Hf↔BALB/c, and C57BL/6↔BALB/c. In the case of C3Hf↔F_1 (C57BL/6 ♀ × BALB/c ♂), one embryo of the pair was an F_1 hybrid and three *H-2* alleles can thus be present concurrently; pigmentation is not a differential since BALB/c carries *AA* and both constituents would therefore be agouti.

When blastomeres from any two genetic sources are experimentally aggregated, the resultant animals generally fall into three classes: those which resemble only one or the other of the genotypes used, and a group with cells of each of the two kinds. It must be emphasized that elimination of one of two original genotypes clearly bears no relation to immune rejections. A number of survivors with only one detectable genotype have been obtained even when both members of the pair were immunogenetically identical: for example, coisogenic strains differing only because of a mutation at one coat color locus (*4–6*). The process of embryo size reduction in vivo may contribute heavily to such unilateral exclusions, but selection undoubtedly takes place at later times as well. The mosaic class itself consists of a quantitative spectrum of individuals with varying proportions of the two genotypes of cells. The frequency distribution within the total span is often significantly skewed in a direction characteristic of the genotypic combination. As suggested by Mintz elsewhere (*5*), the facts strongly imply that specific selective advantages commonly exist between cells with alternative gene expressions at virtually any locus, and that selection at the cell level plays an orderly and major role in ontogeny.

The availability of a quantitative color series within an allophenic combination provides opportunity for comparisons between color markers and the strain-associated *H-2* variants. Coat color genes are known to act directly in melanocytes (for example, black or albino) or indirectly on melanocytes through effects in hair follicles [for example, agouti or non-agouti (*8*)]. The *H-2* genes, on the other hand, lead to antigen formation in a wide variety of tissues, including skin. It is therefore reasonable to expect that two-color animals might show *H-2* mosaicism as well as tolerance for component genotypes. The tolerance situation in one-color cases is less apparent because of possible internal mosaicism, and could be clarified by appropriate graft tests. (The term *allophenic* is retained for all mice from multiple embryos, since total loss of one strain may not become ascertainable definitively in a given case.)

Table 1. Grafts from "parental" strains to 45 allophenic recipients in tests for tolerance. Symbols: + indicates graft acceptance; 0 indicates graft rejection.

Allophenic recipient	Color of recipient	Cases (No.)	"Parental" strain donors	
C3Hf↔C57BL/6	Two-color*	9	C3Hf +	C57BL/6 +
	Agouti†	3	C3Hf +	C57BL/6 +
	Agouti	10	C3Hf +	C57BL/6 0
	Black	6	C3Hf 0	C57BL/6 +
CBA-*T6T6*↔C57BL/6	Two-color	1	CBA-*T6T6* +	C57BL/6 +
	Agouti	1	CBA-*T6T6* +	C57BL/6 +
	Agouti	1	CBA-*T6T6* +	C57BL/6 0
	Black	2	CBA-*T6T6* 0	C57BL/6 +
C3Hf↔F_1(C57BL/6 ♀ × BALB/c ♂)		5	C3Hf +	BALB/c +
		1	C3Hf 0	BALB/c +
C3Hf↔BALB/c	Agouti	2		BALB/c 0
C57BL/6↔BALB/c	Black	1		BALB/c 0
	White	3	C57BL/6 0	

* The tolerant allophenic recipient in Fig. 1 is from this group. † The tolerant allophenic recipient in Fig. 2 is from this group.

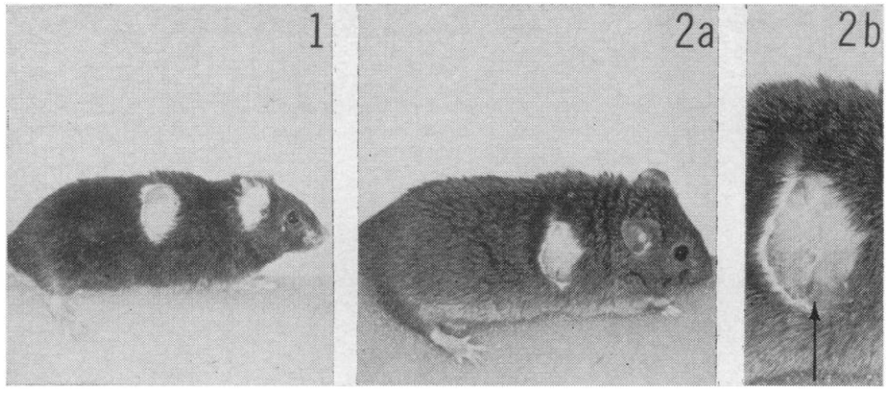
The initial aggregation of embryos in random pairs could lead to XX/XY sex chromosomal mosaicism in as many as 50 percent of allophenic mice. Some individuals of this sort have in fact been identified through karyotype analyses, and their external sex phenotype can evidently be either male or female (*9*). Therefore, grafting experiments were arranged so as to avoid the immunological complication that a Y-linked histocompatibility factor (*10*) would cause if an ostensible female contained XY cells. Grafts from allophenics went only to male recipients; only female donors were used in grafting to allophenics of either external sex.

Ear skin grafts to the side of the body were employed because of the ease with which they remain distinguishable from surrounding host skin. (A report describing the fate of multicolor body skin from allophenics after grafting to the pure strains is in preparation.) Grafts were prepared by cutting off the ear at its base, peeling the two flat surfaces apart, and removing the cartilage by scraping on a support of saline-saturated filter paper. Each ear thus yielded two thin half-moon shaped pieces. Surgical procedures for application of graft to host have been described elsewhere (*11*). When one animal received two grafts, they were placed side by side in the same bed. Where an allophenic acted as graft donor, half of each ear went to each of the two "parental" strain recipients. Control grafts were also exchanged between the original strain pairs. Dressings were removed and operative sites inspected on the eighth to tenth postsurgical days. Grafts from allophenics were examined daily for the first 3 weeks and then at least weekly for 7 weeks. Grafts to allophenics were also observed frequently, and persistent ones were followed for the entire lives of the animals.

Table 2. Grafts from 21 C3Hf↔C57BL/6 allophenic donors to "parental" strains, in tests for mosaicism. Two-color donors are listed in order of progressively decreasing proportion of agouti and increasing black in overall coat color. "T" animals were also used as graft recipients and found tolerant to grafts of both original strains; "t" animals were not tolerant of the opposite-color strain (see Table 1).

Donors (No.)	Percentages of grafts surviving in recipients:			
	C3Hf		C57BL/6	
	Left ear	Right ear	Left ear	Right ear
Agouti-colored donors				
2, t	100	100	0	0
1, T	100	100	0	0
Black donors				
4, t	0	0	100	100
Two-color donors				
1	65	100	0	10
1	100	75	0	0
1, T	95	60	10	35
1	60	35	0	25
1, T	100	90	0	10
1	20	20	100	100
1	50	50	95	100
1	50	25	100	50
1, T	40	25	100	90
1, T	0	20	100	100
1, T	†	†	100	100
1, T	0	0	50	100
1, T	0	10	40	100
1, T*	0	0	95	95

* This donor is shown in Fig. 1. † Technical failure.



Figs. 1 and 2. Tolerant C3Hf↔C57BL/6 allophenic recipients, each bearing a C3Hf and a C57BL/6 ear skin graft side by side on the right flank. The recipient in Fig. 1 has both agouti and black coat colors; the animal's own ears were removed and grafted to the "parental" stains. The recipient in Fig. 2a is all agouti; an enlargement of the graft, in Fig. 2b, shows donor pieces on either side of a fine vertical scar (arrow).

The tests for tolerance (Table 1) show that all ten two-color animals (of C3Hf↔C57BL/6 and CBA-*T6T6*↔C57BL/6 types) were indeed tolerant of grafts from both original strains (Fig. 1). Of 15 randomly chosen all-agoutis from the same two allophenic pairs, four were tolerant of both parental grafts (Fig. 2) and therefore must have contained some C57BL/6 cells elsewhere than in their hair follicles; the remaining 11 were not. Of eight all-blacks from these strains, however, none accepted grafts from the agouti-colored source. The rate of rejection of incompatible grafts followed the same strain-specific schedule as in controls (for example, rejection of C57BL/6 by C3Hf by day 12, and the reciprocal by day 9). A small sample of six one-color individuals from other strain combinations (C3Hf↔BALB/c and C57BL/6↔BALB/c) were similarly found to lack tolerance to the strain of opposite color. The C3Hf↔F_1 (C57BL/6 ♀ × BALB/c ♂) hosts were challenged with C3Hf and with BALB/c in lieu of the F_1 constituent. Tests here also revealed some tolerant animals and one that accepted skin from only one donor.

Tolerance was permanent in all cases. Some animals were killed (for other studies) at ages ranging from 8½ to 26 months; some are still alive at 30 months with grafts still intact. No evidence of runt disease has appeared in these or in any of the total population of 500; the spleen, lymph nodes, and other organs of many have nevertheless been found to be a mixture of two unrelated cell strains (*6*, *12*).

Tolerance here can be designated natural or "intrinsic," rather than "acquired," because of its known basis in early development, and it therefore differs from all previous experimental examples of tolerance. Burnet and Fenner (*13*) first postulated a model of absence of autoimmunity in normal ontogeny. The present experiments serve to establish the validity of that model and to show that this kind of "intrinsic" tolerance is indeed characteristic of development. Association of genetically unlike cells prior to differentiation precludes any subsequent appraisal by either type of the other as foreign. Presumably, an *active* process of self-recognition (here multiple "selves") is involved. The lack of tolerance in some one-color animals, which assuredly had the "lost" strain at least through the blastocyst stage, suggests that capacity for immunological self-recognition does not arise in embryonic life until post-blastocyst.

Nontolerant allophenics might conceivably harbor some cells of the graft-rejected type, since analogous situations have been reported. These include slow rejection of skin grafts between dizygotic cattle co-twins with red cell mosaicism (*14*), and homograft rejection by mouse radiation chimeras with donor-type γ-globulin in their serum (*15*). Many tissues as well as blood from our nontolerant allophenics are being analyzed for mosaicism with various markers, and the results, although incomplete, suggest that under these conditions nontolerance may usually accurately reflect nonmosaicism (see *6*).

In order to be sure that tolerance in allophenic mice is not due simply to immunological inadequacy, two grafted individuals from the CBA-*T6T6*↔C57BL/6 group were challenged with skin from the unrelated BALB/c strain. As the good health of the animals would lead one to expect, the normal immune response was made and the grafts were rejected.

All graft tests for mosaicism (Table 2) were done with C3Hf↔C57BL/6 animals. Two-color donors were color-graded as coded unknowns and listed in order of progressively decreasing proportion of agouti and increasing black in overall coat color. The first five were more than half agouti; the last nine, more than half black. Results observed independently in pure-strain hosts bearing grafts from these animals were then added. Most grafts were partially rejected so that necrotic and intact patches of varying sizes were intermingled. The rejection pattern was evident by the 13th day, when the total area of healthy foci was estimated. Intact sites often later became contiguous, but the final graft size was sometimes much smaller than the original. In other respects, they were indistinguishable from completely compatible ear skin transplants.

There is considerable agreement between color ranking and graft scores of donors. As black becomes more prominent, graft acceptance by C3Hf diminishes, and acceptance of C57BL/6 increases, as would be expected from the fact that the *A* and *a* genes responsible for this color differential act in components of skin. Despite some complete rejections in one strain, the partial rejection of skin from the same donor in the other strain shows that all two-color donors (apart from one technical failure) possessed both genotypes of cells. Local skin differences are evident, even in the same ear, and zero scores could be due to these or to difficulty in detecting a very small surviving patch. The results demonstrate production of different allele-specific histocompatibility antigens in skin of these animals, as has also been seen (*7*) in the erythrocyte population. The grafts from seven one-color allophenics were accepted by the same color strain only.

Fifteen allophenic donors were also checked for tolerance, and the results were concordant (Table 2). The one tolerant agouti without C57BL/6 in its ear skin is no exception. Other analyses of that individual and of the other tolerant all-agoutis prove that mosaicism exists in a number of their internal tissues (*6*).

The results of the present investigations are directly relevant for an assessment of the hypothesis of "allogeneic

inhibition" advanced by the Hellströms and the Möllers (*16*). According to this view, based on complex in vitro and transplant experiments, cells of different histocompatibility types can destroy or inhibit each other on direct contact, without antibody formation or immune-system intervention, and can thereby act as a surveillance system in the organism by eliminating variant cells. While the observations are of interest, they appear to be strictly *sui generis* and to apply to the unusual conditions of those experiments rather than to the biological situation more characteristic of an organism. From allophenic mice, it is clear that in an intact, unirradiated, and healthy animal, cells of diverse histocompatibility types can coexist in any tissue for an entire lifetime with complete impunity.

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Radio Reflection by Free Radicals in Earth's Atmosphere

Barry, Coleman, Libby, and Libby (*1*) suggest that signals recorded by the Canadian satellite Alouette II are due to the stimulated emission of magnetic-dipole radiation from Zeeman transitions of free radicals in the earth's magnetic field. The main evidence presented in favor of this hypothesis is that the signals appear to track with the value of the magnetic field at the satellite and that the observed frequencies give Landé g values (proportional to the ratio of signal frequency to local magnetic field) between 0.6 and 1.8, which are in the same range as some known ground- or metastable-state g values of free radicals. The ionograms shown were taken over the Antarctic with the satellite at altitudes of 1000 to 1300 km where the earth's field is about 0.3 gauss. It was tacitly assumed that the radiation propagates at the free-space velocity, and the same assumption is used in most of the following.

Unfortunately, the calculation given in (*1*) for the signal due to induced magnetic dipole transitions is incorrect. The authors assume that the energy lost by those free radicals which were initially in an upper Zeeman sublevel is radiated isotropically, that those in the lowest sublevel do not take part in the radiation process, and that the amplitudes back at the satellite antenna add incoherently. Actually, only spontaneous emission together with the difference between the stimulated emission and the absorption play a role. Also, any excess stimulated emission energy will travel away from the satellite, in the same direction as the transmitted pulse, owing to a net constructive interference between the electromagnetic wavelets radiated by the free radicals and the very much stronger stimulating wave (*2*).

We can find the power radiated back to the satellite by quantum mechanical calculation of the expectation value of the oscillating magnetic dipole moment, and by then putting this value into the standard magnetic dipole radiation formula derived from Maxwell's equations. For completely incoherent addition of the radiation from each free radical as assumed in (*1*), the actual power returned to the antenna would be weaker by a factor of about 10^{20} than that calculated in (*1*) and would just correspond to normal spontaneous emission. This is in agreement with the standard result obtained if the radiation field is quantized also: namely that the stimulated emission due to a given mode of the radiation field goes into the same mode provided that coupling between the modes can be neglected.

To calculate the actual power that would be returned to the Alouette II satellite, still assuming free-space propagation, we must take account of the relative phases of the wavelets radiated by the free radicals. We assume a satellite dipole antenna along the the earth's field direction and take this to be the axis. For resonance at $\nu = 10^6$ cy/sec, $J = \frac{1}{2}$, and $g = 2$ and for distances of roughly a wavelength or greater from the satellite, the oscillating magnetization per unit volume in the azimuthal direction is given approximately by

$$M = 4.5 \times 10^{-14}\left(\frac{\sin\theta}{r}\right) \times$$

$$\tau(N_+ - N_-)\, e^{ikr} e^{-i\omega t} \quad [\text{erg gauss}^{-1}\ (\text{cm}^3)^{-1}]$$

Here r is the distance from the antenna, θ is the angle between r and the axis, $k = 2\pi/\lambda$, λ is the wavelength, $\omega = 2\pi\nu$, $\tau \leq 10^{-4}$ second is the time the satellite pulse has been on at the point in question, and $N_+ - N_-$ is the difference between the number of radicals per cubic centimeter initially in the upper and the lower Zeeman sublevels. After adding up the contributions to the electric field along the satellite antenna which were due to the magnetization in each element of volume and estimating the contributions from closer than a wavelength to the antenna, we find that a value of $N_+ - N_-$ of about 10^7/cm would be needed in order to give observable signals. The contributions from the near-field and far-field regions are comparable. The power returned to the satellite should be regarded as enhanced spontaneous emission from a highly radiating state set up by the satellite pulse.

From the above result and the normally assumed total atmospheric density of $N < 10^6/\text{cm}^3$ at the altitudes of interest, it seems highly unlikely that the Alouette II signals discussed (*1*) are due to induced magnetic dipole radiation from free radicals. If the radicals had a thermal equilibrium distribution over the Zeeman levels, a density of about $10^{14}/\text{cm}^3$ would be required for observable signals, since $(h\nu/kT) \sim 10^{-7}$. This agrees with the estimates made by Hodges and Colegrove (*3*) in refuting an earlier proposal for finding the density of free radicals at