cal team, and support personnel during the quarantine period; it also has the contingency capability of housing all personnel that might be exposed to biologic hazards in the sample laboratory by failure in a barrier system. It is anticipated that the period of quarantine for samples will be about 30 days, which period the laboratory is capable of extending if a specific problem of back-contamination emerges.

Conclusion

The Lunar Receiving Laboratory will be the permanent depository of a portion of the collection of lunar samples; it will safeguard the collection, providing continuing security and ensuring scientific integrity. In carrying out the time-dependent experiments and continuing functions of the laboratory, NASA will rely on visiting expert scientists supplementing a relatively small resident staff; outside scientists will be relied upon for most investigations and detailed analyses of samples. It is believed that the designed procedures and facilities provided will ensure the maximum scientific return from the Apollo Program in the way of information from lunar samples.

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Radiation Chimeras and Genetics of Somatic Cells

Tissue cultures in vivo allow selection and detection of genetic variants of somatic cells.

Alena Lengerová

It took geneticists a fairly long time to admit that genetic analysis in mammals might in principle bypass sexual reproduction and take advantage of the processes of genetic recombination which have come to light in bacteria and viruses, or of similar processes (*1*). It may, however, take even longer before what seems theoretically possible will be generally possible. What are actually the prerequisites of success in any experimental system? They seem to be the following:

1) Existence of processes in somatic cells which can result, under appropriate conditions, in gene recombination.

2) Availability of suitable genetic markers—that is, alternative characters detectable at the cellular level.

3) Conditions which favor the variant phenotype on the basis of one or another of the possible principles.

4) Availability of techniques of handling somatic cells according to the requirements of formal genetic analysis.

The first of these four conditions seems to be critical, as it is much more independent of experimental skill, sophistication, or good luck than the other three are. It seems quite clear either that processes possibly resulting in gene recombination are very rare in mammalian somatic cells (if they occur at all) or that the recombinants have a low chance of surviving or of being phenotypically expressed. This might be due to the existence of some mechanism (or mechanisms) which keeps control over the uniformity of somatic cells. The possibility that the somatic recombinants might either come into being or happen to survive and express their phenotypic change just by some sort of accident does not necessarily mean that they could not be exploited in a systematic study. The parasexual cycle in filamentous fungi (*2*) is based on a series of rare and probably accidental events, and nevertheless it proved extremely useful as a means of genetic analysis. The first problem thus is to find whether there are at least some indications that gene recombination can occur, however rarely, in mammalian somatic cells.

A great deal of experimental evidence is based on immunoselection of homozygous (or hemizygous) cell variants arising in mouse tumors heterozygous at the *H*-2 locus (*3*). The results are fully compatible with somatic crossing-over as the underlying mechanism, although some other possibilities cannot as yet be excluded. Additional but still indirect evidence in favor of somatic crossing-over in the same experimental system was provided by the demonstration that the parent compatible variants are due to changes at the chromosomal rather than at the phenotypic level (*4*). Recently, reports of rare instances of animals heterozygous for a recessive coat-color gene but showing patches of fur with the recessive phenotype were extracted from the literature, and an attempt was made to find a common denominator for various peculiarities of such mosaics; in spite of the rather speculative nature of evidence after the event, the reported data seem to make "a strong case for the existence of somat-

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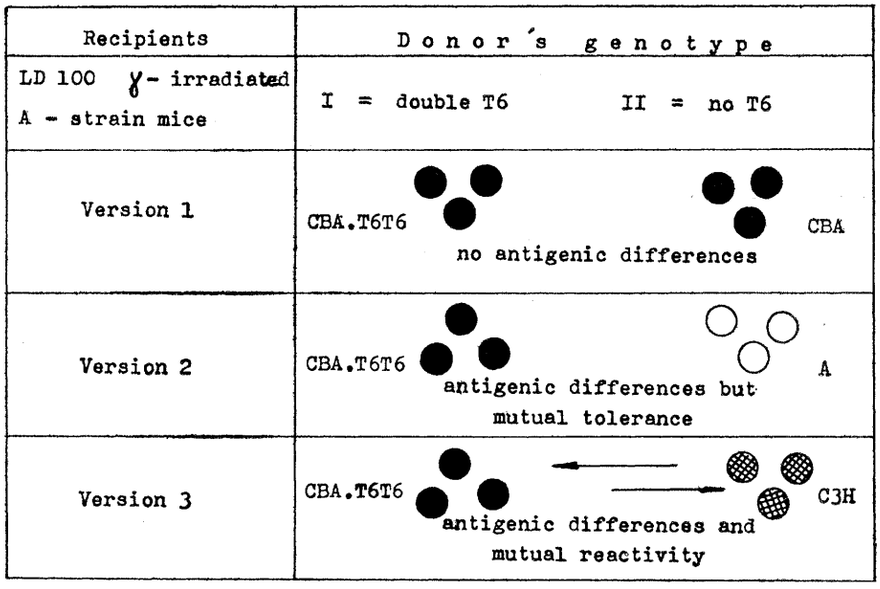

Fig. 1. Three versions of a radiation chimera scheme for detecting heterozygous somatic cell hybrids.

ic crossing over in the laboratory rodents" (*5*). Furthermore, the finding of quadriradical configurations in human nucleated blood cells dividing in vitro (*6*) may be interpreted as cytological evidence of somatic crossing-over; the low incidence of such configurations and the absence of reports of such quadriradicals in dividing cells taken directly from bone marrow seem to suggest that somatic crossing-over, especially in vivo, may be an extremely rare phenomenon in most individuals.

Crossing-over is, however, not the only potential source of gene recombination in mammalian cells. There are now data from several laboratories showing clearly that, under certain conditions, some sort of mating takes place between somatic cells in vitro (*7*). The first direct evidence of this phenomenon was provided by the demonstration of cell fusions in mixed cultures of tumor cells distinguishable by their chromosome number and morphology; subsequent gradual loss of chromosomes can be considered a process of mitotic segregation operating in such systems (*8*).

All these findings are very stimulating, as they clearly demonstrate that the individual steps of the parasexual cycle (or their analogs) in principle also exist in mammalian somatic cells. However, some of the usual features of cells grown in vitro (especially aneuploidy) represent a definite disadvantage for genetic analysis. It is thus desirable to investigate other experimental systems with respect to their suitability for the proliferation and detection of genetic recombinants of normal somatic cells.

The purpose of this article is to show that radiation chimeras might provide an adequate model to meet all the requirements specified above.

Radiation chimeras (*9*) are created by injecting healthy hematopoietic and lymphoid cells into animals previously irradiated with a dose of ionizing radiation which would ordinarily be lethal. The injected cells display a homing instinct and settle down in host tissues corresponding to their histogenetic origin. Since the immune mechanism of the host has been eliminated by prior irradiation, the foreign cells can repopulate the radiation-depleted host tissues. This generally occurs during a relatively short period of extraordinarily active cell proliferation, which might provide an increased chance of some accidental recombination processes' taking place. A radiation chimera can thus be considered a sort of tissue culture in vivo, where the advantage of the more or less normal physiological conditions is not counterbalanced by the disadvantage of tissue incompatibility of the usual living system.

Genetic markers available in this experimental system are different forms of hemoglobin (*10*), antigenic differences [in mice, especially those controlled by the *H*-2 and other *H* loci, where advantage can be taken of the existence of many congenic lines (*11*)], and some chromosomal characteristics.

Experiments on competitive cell repopulation in mice given doses of radiation which would ordinarily be lethal ("lethally irradiated mice") showed that the dynamics of mixed cell populations in radiation chimeras is governed by a series of factors of immune and nonimmune nature which can be used to select for the variant or against the original cell phenotype. As passaging and cloning (at least to some extent) of the chimera's cells is also possible, the basic formal requirements are satisfied. A specific advantage of radiation chimeras seems to be the fairly wide range within which the experimental system can be manipulated in order to increase the incidence of newly formed variants or the chance of their selective proliferation and, consequently, of their detection. Experiments can, for example, be designed to search specifically for cell recombinants due to somatic mating or to somatic crossing-over. The selective capacity of such experimental designs can then be tested by determining the thresholds of detectability for the presumed variant phenotype in artificial mixtures of cells used as curative inocula for lethally irradiated mice.

Somatic Fusion and Segregation

Under normal conditions in vivo, somatic mating followed by segregation can be detected only in heterozygotes, for some cellular markers, when the homozygous cell products are distinguishable. This would be the case, for example, with XX cells arising in male tissues. The known examples of sex chromosome mosaicism (*12*), as well as the incidence of erythrocytes lacking one or the other parental antigen in some individuals of blood group AB (*13*), have been interpreted on the basis of different principles up to this time. One wonders, however, whether one of the factors behind the more general absence from heterozygotes of homozygous somatic recombinants might not be the selective disadvantage of such recombinants in the competition with the heterozygous cells. If this is the case, more favorable growth conditions for the recombinant cell phenotype could be created in radiation chimeras through the use, as curative inoculum, of a mixture of two cell types, homozygous for one or the other of the markers. In that case, the product of somatic mating and segregation would be

heterozygous and should enjoy a selective advantage. Some other factors might, however, play a role even earlier by affecting the frequency with which the recombinants arise. The first step in the process of fusion of two cells is obviously their contact. The chance of contact depends primarily on cell concentration, but it might possibly be increased by means of agents which tend to aggregate the cells. It is not immediately clear whether the tendency for the cells to fuse depends, further, on some sort of polarity or whether it depends simply on some difference between the two cells or on an altered activity of the cell membranes (*14*). But fusion alone might not give rise to a viable product if the two cells were not in a corresponding phase of their mitotic cycle. In forming radiation chimeras, attempts can be made to control these and other factors through choice and preliminary treatment of the curative inoculum.

Some of the possibilities have been tested in this laboratory (*15*). The basic experimental scheme, which is being used in different versions, is illustrated in Fig. 1.

The recipient is a mouse totally irradiated with a dose of gamma rays which is ordinarily absolutely lethal and induced to recover through administration of a mixture of hematopoietic cells (plus lymphoid cells) derived from two types of donors. The origin of the injected cells or their progeny can be recognized by the presence or absence of two chromosome markers, the T6 translocation chromosomes. At different intervals after the formation of chimeras the animals are treated with Colcemid and then killed, and their bone marrow, spleen, and lymph nodes are examined in order to determine the relative incidence of metaphases of the two parental types and eventually of a new recombination type.

In the attempt to affect the mutual affinity of cells of the two parental lines, various modifications of the basic scheme can be tested. The two chromosomally distinguishable cell types can be either syngenic (antigenically similar)—the type used in version 1 of the experimental scheme—or allogeneic (antigenically different)—the type used in versions 2 and 3. (In addition, their antigenic relationship to the host can be varied.) The antigenic difference between the two cell types does not necessarily result in an immune interaction. It was demonstrated earlier (*16*) that when antigenically different cells are taken from immunologically immature donors—that is, from fetuses—they usually acquire tolerance of each other and are capable of coexisting in the chimeric tissues. Nevertheless, such immunologically neutral antigenic differences still represent some sort of structural differences of cell surfaces which might affect, in one way or another, the mutual affinity of the cells and the likelihood that they will come into contact or, eventually, fuse (version 2). There is a further variation: The case when the two antigenically different cell types do not acquire mutual tolerance (version 3). This is usually the case when immunologically competent cells from adult donors of two different genotypes are brought into contact; since they mutually represent both source and target of antigenic stimulation, they have a tendency to kill each other through contact. The possibility remains, however, that there will be survivors, resulting from accidental fusion of the mutually reactive cells.

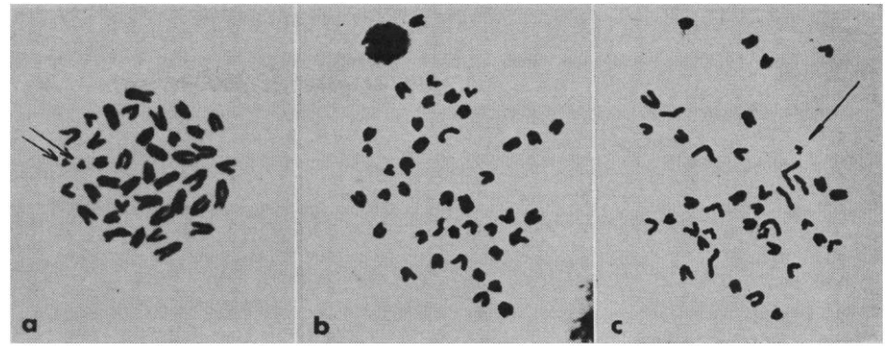

Fig. 2. The metaphases of the parental cell types with (a) two T6 markers and (b) no T6 marker, and of (c) a diploid hybrid cell with one T6 marker.

Among several thousands of scored metaphases in experimental versions 1 and 2, about 100 diploid cells with one T6 marker were found (Fig. 2). (So far, no "hybrid" cells have been found in experimental version 3.) The origin of such cells can scarcely be explained by an accidental loss of the other T6 marker from a diploid cell homozygous for the T6 translocation because these cells have a normal set of 40 chromosomes, and this would require the loss of one T6 marker's being counterbalanced by the gain of one normal chromosome. This might occur through a double (complementary) mitotic nondisjunction, but this is highly unlikely. It is also rather unlikely that the mechanism by which the diploid chromosome number is reestablished after the presumed fusion of two diploid cells is based on gradual accidental loss of chromosomes such as occurs in vitro. The intermediary aneuploid cells are not found, which is not surprising because their chance of surviving in vivo must be very low, as is indicated, for example, by the extremely low incidence of hyperdiploidy in normal mouse tissues (*17*). Somatic cell mating seems to occasionally take place (or be detectable) also in animals with erythrocyte mosaicism due to prenatal vascular anastomosis with another individual, such as may occur spontaneously in cattle twins (*18*) or may be produced experimentally in avian embryos (*19*). In the cases discovered so far of recombination of erythrocyte antigens in natural twin cattle chimeras (*18*), there is only indirect indication of somatic segregation, and thus the mechanism remains obscure.

Some light might be cast on this process by study of somatic hybrids between two parental cell lines distinguishable by more than one chromosome marker. In the mouse radiation-chimera system, the choice of suitable chromosome markers is limited but the sex chromosomes represent at least one set additional to the T6 translocation. The identification of mouse sex chromosomes (*20*) may be considered uncertain, but with some practice such identification can be made with a reasonable degree of confidence. In an experiment of this kind a mixture of two types of fetal cells—XX, two T6 markers, and XY, no T6 markers—was given as a curative inoculum for lethally irradiated A-strain female mice (*15*). If in this case two types of cells heterozygous for the T6 marker were found with about the same frequency (XX and XY), this would indicate either that the segregation mechanism was rather regular or that the

hybrid cells found represented only a selection of viable products of an irregular segregation process. The number of hybrid cells scored so far is still too small to warrant any conclusion.

This type of experiment might also have some bearing on the mechanism of inactivation of one X chromosome in female somatic cells in mammals (*21*). The hybrid cells with one T6 marker and two X chromosomes should fall into three classes according to the origin of their X chromosomes: cells with both X's from the maternal (female) cell (that is, cells with only one active X chromosome) or cells with one active X of paternal (male-cell) origin and the other X of maternal origin and consequently either active or inactive. It would be interesting to see whether the hybrid cells with two active X chromosomes might persist or whether the inactivation mechanism can be triggered at any stage of life when, by some sort of mitotic accident, two active X chromosomes happen to appear in one cell. Cells with two inactive X chromosomes would be even more interesting for what they would reveal of the stability of the X inactivation. The number of cells with two active X chromosomes should represent a certain proportion of all the XX cells. [A recent report on a sex chromosome mosaic seems to point to the possibility of both X chromosomes' being active in human peripheral lymphocytes dividing in vitro (*22*), an occurrence which might, of course, be an exception rather than a rule.]

The technique of scoring genotypes of somatic hybrid cells could, of course, be used only if the observed ratio could be compared with the ratio expected on the basis of a known mechanism of somatic segregation. Indirect evidence for the existence of somatic segregation was accumulated, and the first cytological proof was provided, by Ohno (*23*). The cytological evidence is based on the finding of all three possible combinations of an acrocentric and a subtelocentric chromosome from a heteromorphic pair of autosomes in diploid spleen cells of a deer mouse (*Peromyscus maniculatus*). Ohno also used a short-term culture of mixed spleen cells (a mixture of cells with two T6 markers and cells with no T6 markers, or mixed radioactively labeled and unlabeled cells) from mice previously sensitized with a skin graft from a third strain. He found an incidence of diploid hybrid cells similar to that reported above for the radiation chimeras, and also many examples of fusion of two similar or different cell types. He suggested tetrapolar mitosis or two simultaneous bipolar mitoses partially overlapping in the cytoplasm of the tetraploid cell as the most probable mechanism of somatic segregation. This latter alternative seems to be compatible with the finding that not all autosomes are necessarily involved in the segregation process (*23*).

Selection for Somatic Cell Hybrids

Since the somatic cell hybrids in the radiation-chimera system described (as well as in some other experimental systems) appear much the same as cells from heterozygotes for a given marker (for example, the T6 translocation) obtained by sexual crossing, it is possible to test various conditions with respect to their capacity to stimulate selectively the proliferation of such heterozygous cells artificially added, in small amounts, to a mixed cell population of the two parental cell types. If the selection pressure were strong enough, the obviously low yield of newly arising recombinants in the primary chimeras should not particularly matter. One possibility is to make use of the differential colony-forming capacity of bone marrow cells of different genotypes in the spleen of massively irradiated recipients (*24*). The relative number of macroscopically visible colonies [which have been shown to represent cell clones (*25*)] in the syngenic host-donor combination indicates a characteristic colony-forming capacity of the cell genotype in question; if its performance is known to be reduced in a certain nonsyngenic host, such a host environment can be advantageously used when this cell type, present in a mixed cell population, is to be selected against. In one experiment of this type (Fig. 3), a mixture of equal parts of cells of three different genotypes distinguishable by the presence of two T6 markers, none, or one [genotypes CBAT6T6, A, and (CBAT6T6 × A)F_1, respectively] were injected into three groups of mice of the same three genotypes previously irradiated with 900 roentgens of gamma rays. Ten days later the recipients were killed, after treatment with Colcemid; the nodules in their spleens were counted, and then each nodule was dissected out and the cells were prepared for chromosome analysis, in order to score the genotypes of the individual clones. The result indicated that the effect of the host environment on the colony-forming capacity of cells of different genotypes is more pronounced when the cell types are in competition than when each cell type is tested separately. The syngenic components increased, during the 10-day period, from 33 percent to 75 to 90 percent, while the allogeneic and semisyngenic components decreased correspondingly (*15*). How the competitive advantage of the syngenic component can be expressed under the less favorable initial conditions existing when the syngenic component represents only a small minority of cells in the mixed inoculum deserves further investigation.

The proliferative advantage provided by the syngenic-host environment does not necessarily hold for any genotype. It seems to hold for the C57BL-C3H combination (*24*) and also for the A-CBA combination, as indicated by the test for colony-forming capacity mentioned above and also by an experiment by Ford (*26*): In A-strain mice which were inoculated at birth with CBA spleen cells (chromosomally distinguishable from both A and normal CBA cells) and which consequently tolerated CBA skin grafts, no dividing donor cells could be detected among bone marrow cells until the bone marrow cells had been transferred into lethally irradiated CBA hosts —that is, into a syngenic environment. However, under certain conditions, cells of a certain genotype may have a higher proliferative capacity in a foreign host than cells of another genotype in the syngenic environment. This is clearly demonstrated by the fact that the quantitative composition of an erythrocyte mosaic is often the same in cattle twins of the same pair, indicating that the host type is not necessarily predominant (*18*). The proliferative performance of cells in a foreign host environment seems to be associated with their genotype—in mice, particularly with the alleles of the histocompatibility *H*-2 locus (*27*). This does not automatically indicate an immune background of the host-cell interactions but indicates, rather, the important physiological role of the loci whose products can secondarily also function as cellular antigens. The phenomenon may have a common background with the "syngenic preference" or "allogeneic inhibition" (*28*) which seem to represent two aspects

of the same process of defective growth of cells exposed to cellular antigens absent from their own surface.

Although the syngenic preference, in a broader sense, is greatly dependent on many qualitative and quantitative factors, once the optimum conditions for a given system are found, it can provide a selection force in the search for somatic cell variants. In the radiation-chimera system, the curative inoculum should thus consist of two homozygous cell types which exhibit a defective growth in the respective F_1 hybrids. If the F_1 hybrids are then used as recipients, the rarely arising somatic hybrid cells could enjoy syngenic preference, whereas cells of both the parental cell lines should theoretically suffer from allogeneic inhibition. The difference might become even more pronounced on regrafting of the chimeric bone marrow into secondary irradiated F_1 recipients, where the three different cell types would have to undergo many cell divisions during competitive repopulation of the host's bone marrow spaces. The experimental design should, however, allow one to distinguish (by means of a nonantigenic cell marker) between the cell progeny of the somatic variant and the progeny of an antigenically similar host's cell that had accidentally survived the massive irradiation.

Selection for Homozygous Variants

One may ask whether the possibility of selecting for heterozygous cell variants excludes successful selection for homozygous products of somatic crossing-over arising in a population of heterozygous cells. It does not necessarily do so, and the system of radiation chimeras can be exploited to this end.

Two approaches based on the host-to-graft and the two-way graft-to-graft interactions may be considered.

The first of these approaches is based on the known fact that radiation chimeras can give a host-specific type of immune response provided the recipient of the curative inoculum received the appropriate antigenic stimulus prior to irradiation. Let us assume a congenic system of two lines of mice differing antigenically only at the *H*-2 locus, the respective alleles being, for example, *b* and *d*. Homozygotes *bb* can be immunized against antigens of the *d* allele and then used as irradiated recipients of a *bd* inoculum presumably containing some homozygous cell variants of the *bb* genotype. The *bd* cells would be the target of host-versus-graft immunity leading to their gradual more or less complete elimination, whereas the cells of the *bb* variant, syngenic with the host, would have a selective advantage. In one model experiment of this type an artificial admixture, to the inoculum of 10^7 heterozygous cells, of 10^6 homozygous cells of the presumably variant phenotype was shown to be sufficient to insure survival of about half of a group of mice sensitized to the *d* antigens and then given doses of radiation that would ordinarily be lethal, and in some of the survivors erythrocytes were only of the "variant" phenotype. With a spontaneous variant, the quantitative relationship would, of course, be much less favorable for the homozygous cells; it is thus not clear whether they could induce survival of the recipient without any contribution of heterozygous cells which had escaped destruction by host immunity. Nevertheless, a considerable shift in the composition of the mixed cell population could be achieved on the basis of this principle. The possibility that any *bb* erythrocytes present might be of host origin can be excluded by arranging the experiment so that the *H*-2 compatible variant and the host cells (both *bb*) would have different types of hemoglobin (for example, diffuse versus simple). An obvious disadvantage of this experimental system—and one which cannot be eliminated—is the fact that only one homozygous variant can be detected in one type of chimera, since the complementary type would necessarily share the fate of the heterozygous cells being selected against in the specifically immunized host. This means the sacrifice of one indirect criterion of somatic crossing-over, a mechanism which, unlike other mechanisms of variant formation, might be expected to give about

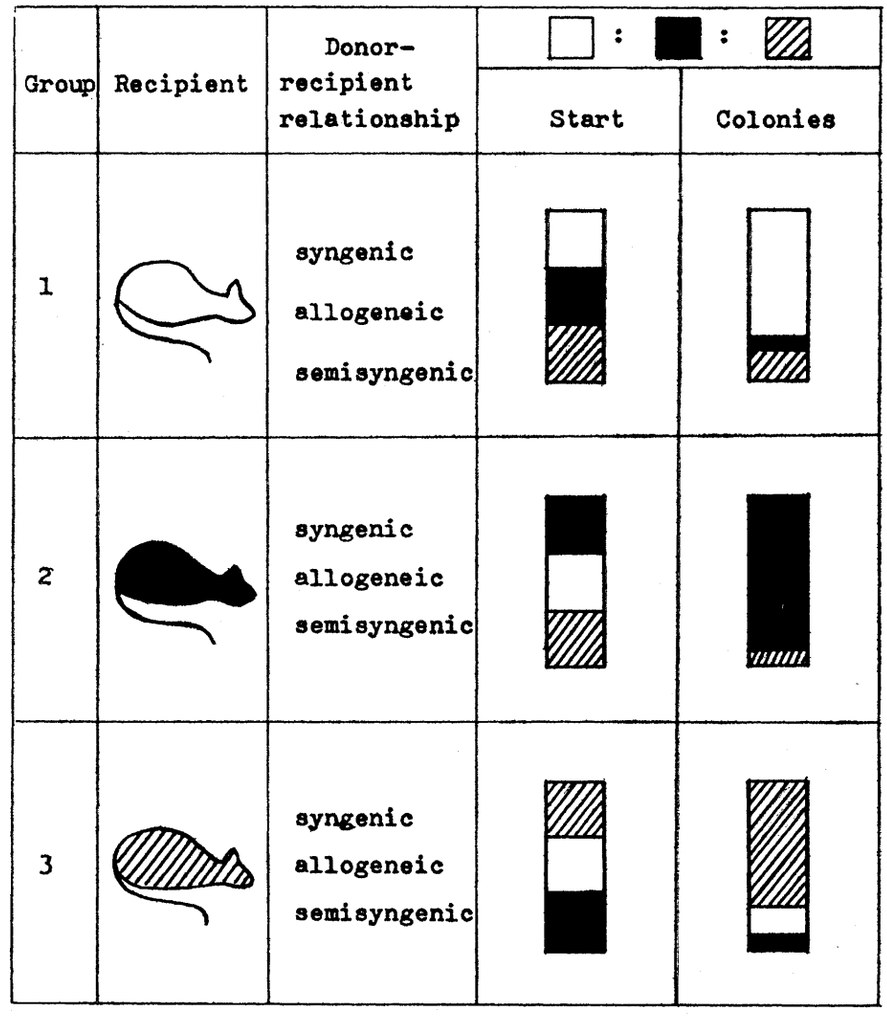

Fig. 3. Competitive colony-forming capacity of cells of three genotypes [A (= white); CBAT6T6 (= black); and (A × CBAT6T6)F_1 (= shaded)] injected simultaneously in equal amounts into mice previously given 900 roentgens of gamma radiation.

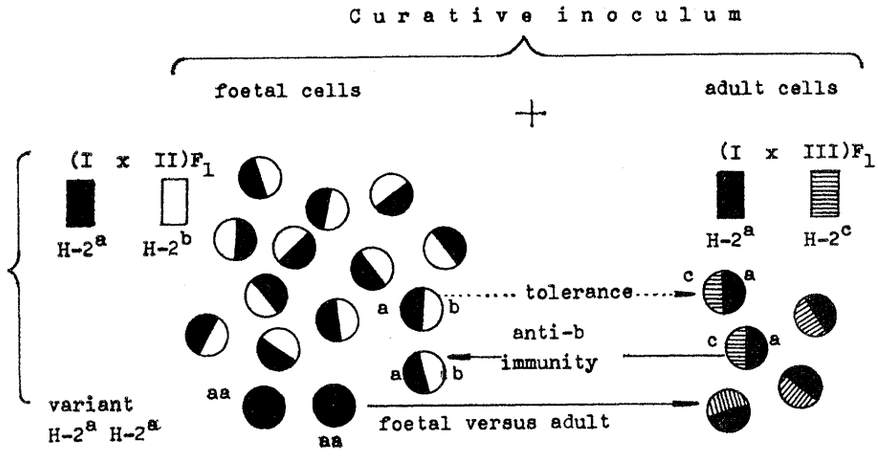

Fig. 4. Selection system for homozygous cell variants (due to mitotic crossing-over) based on graft-to-graft interactions.

equal yields of homozygous variants of the two types. However, since the analytical value of this criterion is rather limited, the price does not seem too high.

Two other types of interactions previously demonstrated (*29*) in a mixed cell population in radiation chimeras might be of interest in selecting spontaneous homozygous variants. (i) When two antigenically different cell grafts are brought into contact, one from fetuses and the other from adult donors, they represent a mutual antigenic stimulation to which the adult cells respond by immunity whereas the fetal cells respond by tolerance. The tolerance response, here, is an obvious proliferative disadvantage, as the tolerant cells are unable to retaliate and are eliminated by an immune reaction of the others. (ii) On the other hand, the fetal cells, even if in a minority at the start, appear physiologically superior and can out-grow the adult cells, which are, for genetical reasons, unable to react against their antigens.

A combination of these two principles might serve as a possible basis for selection for homozygous cell variants provided such variants could arise in heterozygotes during the prenatal period (Fig. 4). Let us assume that the curative inoculum for irradiated mice consists of a population of fetal cells from F_1 hybrids between two congenic lines I and II (*H*-2^a, *H*-2^b) already containing some cells derived from a spontaneous *aa* variant. If some mature lymphoid cells, or even lymphoid cells, sensitized to the *b* antigens, from F_1 hybrids between congenic lines I and III (*H*-2^a, *H*-2^c), are added to this population, the following interactions may be expected to occur. The adult *ac* cells would react against the *b* antigens of the fetal *ab* cells, which would in turn become tolerant of their *c* antigens and consequently would be at a disadvantage in the (two-way) graft-to-graft immune interactions. Sooner or later cells bearing the *b* antigens would be completely eliminated. On the other hand, the *aa* variant has a higher proliferative capacity, because of its fetal origin, and it might out-grow the adult lymphoid cells. This chance could even be increased through reduction of the risk of allogeneic inhibition due to lack of *c* antigens—for example, by coating the *c* antigens on the *ac* cells with a specific antiserum (*30*).

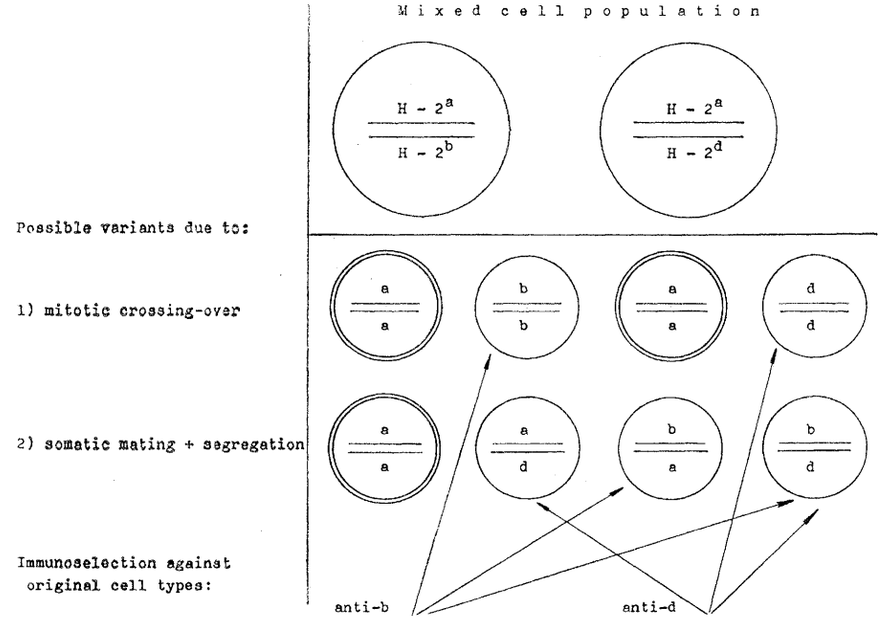

Fig. 5. A possible system for selecting at the same time for cell variants due both to mitotic crossing-over and to somatic mating followed by segregation.

The combination with a third congenic line might also be useful in selecting—in a single experimental system—for homozygous cell variants arising by both mitotic crossing-over and somatic mating followed by segregation. Figure 5 illustrates a possible experimental scheme. The curative inoculum consists of two types of fetal cells denoted by their *H*-2 alleles. Three out of eight possible crossover and hybrid variants would be homozygous for the *a* allele shared by the two original cell lines. A selection pressure against the F_1 cells might be exerted through long-term administration of antisera reacting specifically with the antigenic products of the *b* and *d* alleles. An additional effect of such a treatment might be the abolishing of allogeneic inhibition.

These are some of the well-demonstrated principles, based on antigenic and physiologic disparities, which govern mixed cell populations in mouse radiation chimeras and which may now be exploited in the search for genetic variants of somatic cells. This will be a third phase in the history of radiation chimeras. During the first phase they were just a by-product of research on protection against radiation, while during the second phase they were the focus of many direct experimental attacks. This period seems to be over, but the various mechanisms and relationships which have been discovered can now be exploited in other fields; genetic analysis of somatic cells appears one of the fields in which radiation chimeras provide a promising approach.

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Privacy and Behavioral Research

Preliminary Summary of the Report of the Panel on Privacy and Behavioral Research

In recent years there have been growing threats to the privacy of individuals. Wiretapping, electronic eavesdropping, the use of personality tests in employment, the use of the lie detector in security or criminal investigations, and the detailed scrutiny of the private lives of people receiving public welfare funds all involve invasions of privacy. Although the social purpose is usually clear, the impact on the persons involved may be damaging. Our society has become more and more sensitive to the need to avoid such damage.

This concern has led to extensive discussion about the propriety of certain procedures in behavioral research, by the Congress, by officials in the various agencies of the government, by university officials, by the scientific community generally, and by leaders in professional societies in the behavioral sciences. The Office of Science and Technology appointed a panel*, in January 1966, to examine these issues and to propose guidelines for those who are engaged in behavioral research or associated with its support and management.

The panel has restricted its attention to issues of privacy arising in connection with programs of data collection and study which are intimately associated with behavioral research. For example, it has not reviewed a number of the programs for data collection which are sponsored by the federal government, such as the various censuses, health and welfare statistics, and financial information secured from business and industry. These programs may also encroach upon the privacy of individuals, either through the burden of disclosure which they impose on respondents or through their availability for unintended purposes.

It is our opinion that the principles described in this report for protection of privacy in behavioral research should apply equally to such inquiries. When response is mandatory, as in the case of information that must be furnished to the government, there is an even greater burden on the sponsoring agency to protect the individual against disclosure unless disclosure is specifically sanctioned by statute.

The panel has not reviewed in detail the wide variety of mechanical or electronic devices which make it possible to intrude into private lives. We have become acquainted with a few of the problems in that field, however, and are dismayed to observe the disregard for human values indicated by the advocacy or actual practice of eavesdropping, the use of lie detection without clear justification, and the frequent willingness to institute surveillance procedures to handle the problems of a small proportion of our population at the risk of eroding the

*The Panel on Privacy and Behavioral Research was appointed by the President's Office of Science and Technology. The members of the panel are as follows: Kenneth E. Clark (chairman), dean, College of Arts and Sciences, University of Rochester, Rochester, New York; Bernard Berelson, vice president, Population Council, Inc., New York, N.Y.; Edward J. Bloustein, president, Bennington College, Bennington, Vermont; George E. Pake, provost, Washington University, St. Louis, Missouri; Colin S. Pittendrigh, dean, Graduate School, Princeton University, Princeton, New Jersey; Oscar M. Ruebhausen, Debevoise, Plimpton, Lyons & Gates, New York, N.Y.; Walter S. Salant, Economics Studies Division, Brookings Institution, Washington, D.C.; Robert Sears, dean, School of Humanities and Sciences, Stanford University, Palo Alto, California; Benson R. Snyder, psychiatrist-in-chief, Medical Department, Massachusetts Institute of Technology, Cambridge; Frederick P. Thieme, vice president, University of Washington, Seattle; Lawrence N. Bloomberg, assistant chief, Office of Statistical Standards, Bureau of the Budget, Washington, D.C.; and Colin M. MacLeod, deputy director, Office of Science and Technology (now vice president for medical affairs, The Commonwealth Fund, New York, N.Y.). Consultant to the panel is Richard M. Michaels, technical assistant, Office of Science and Technology, Washington, D.C. The full text of the report will be available about 1 March 1967 from the Superintendent of Documents, Government Printing Office, Washington, D.C. 20402.