that the transition occurs by pieces or bits at these low temperatures. It has been observed by us that frequently the bismuth 1–2 transition at −80°C occurs in steps. Sometimes three pressure increments are needed before the transition is complete. One would expect that the sluggishness of a transition would increase as the pressure is increased and the temperature is lowered.

Of greater interest is the nonmetallic behavior shown in Fig. 3. The maximum observed resistivity in the system was 0.013 ohm cm, a value much too high for any metal, and characteristic of a very heavily doped sample of germanium. The temperature coefficient supports the view that the material has become a semiconductor. If the energy gap (Eg) is computed in the usual manner,

$$R = Ae^{(Eg/2kT)}$$

the gap is found to be a strong function of the pressure. The results of the calculations are shown in Fig. 5. Since the purity of this material is only 99.8 percent, it is evident that this gap is not the intrinsic value but rather attributable to impurities. On a highly purified sample, it would be expected that these gaps would be higher than that reported here.

Evidently there are some unexpected peculiarities in the electron structure of ytterbium. First, let us consider the values of the resistances. The rare earths have unusual magnetic properties. A perusal of the literature shows that no known magnetic change can account for the magnitude of the change observed here, a factor of nearly 800. Resonance scattering (*9*) of the electron between the 4*f* and 5*d* levels could not account for this very large increase.

The electron configuration of ytterbium in the gas phase is $4f^{14}5s^25p^66s^2$. The magnetic susceptibility data of Lock indicate that in the solid only 1/250 of the 4*f* electrons are in the 5*d* state (*8*). This implies that the binding is through the $6s^2$ electrons, a binding similar to that of the alkaline earth metals. This latter agrees with the data on the compressibility of ytterbium. Ytterbium, according to Bridgman's measurements, is much more compressible than the other rare earths he measured; in fact the compressibility is very close to that of barium. This means that the original conductivity is the result of the overlap of the 6*p* and 6*s* bands.

If it is assumed that the band structure of ytterbium is represented by the scheme shown in Fig. 6, it is possible to account for the observed electrical behavior.

After a small compression, the 6*s* and 6*p* bands no longer overlap, and the valence band is now full, and the 6*p* is the conduction band. The gap increases, until the 5*d* intersects the 6*p* band, at which time the 5*d* becomes the conduction band. The gap increases as long as the conduction band is 6*p*, and decreases when the 5*d* becomes the conduction band. When the 5*d* band intersects the 6*s* band, the metallic properties would reappear.

An apparent problem with the simple picture that has been presented is the fact that the resistivity continues to rise after the gap begins to decrease. This can be accounted for in the following manner. The multiplicity in the *d*-band is higher than in the *p*-band. Consequently, the effective mass of the electron will be higher than in the *p*-band. Because the effective mass appears to a high power in the mobility of the electron, it is not unreasonable that the resistivity increases even though the gap has become smaller.

This picture of ytterbium in no way negates Hall's (*3*) proposal of the electronic transition to account for the crystallographic transition. All of the semiconducting phenomena occur in the α phase. The β phase is a normal metal, even when the pressure is reduced below the transition point.

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Homograft Tolerance in Mice: Use of Urethan and Sublethal Irradiation

Abstract. *Adult mice [CBA/J (H-2^k)], which received either a single sublethal dose of x-radiation (500 rad) or urethan plus 500 rad, were given intravenous injections of C3H/HeJ (H-2^k) spleen or bone marrow cells (18 to 42 × 10^6 cells per mouse) or both, for 3 days. C3H/HeJ tail-skin homografts were retained (over 130 days) by these mice, whereas BALB/cJ (H-2^d) homografts all were rejected within 33 days. Similarly irradiated or urethan-treated controls (or controls treated with a combination of both), which did not receive C3H cells, rejected both homografts. Specific homograft tolerance is induced in adult mice by this procedure.*

Induced tolerance to homografts, after parenteral administration of allogenic cells into newborn mice, is well-established (*1*), but similar attempts to produce this tolerance in mature recipients, fully competent immunologically, have proved more difficult.

To produce tolerance across the H-2 histocompatibility barrier in adult mice, the recipients must be subjected to lethal whole-body x-irradiation and then to infusion of allogenic bone marrow cells. Long-term survivors from this treatment are tolerant of skin homografts (*2–4*) and contain donor cells specifically tolerant of the host (*2*). In both these situations, specific unresponsiveness to skin homografts is associated with cellular chimerism. Homograft tolerance in nonirradiated adult recipients has been achieved, but only with mouse strains differing unilaterally at the H-2 locus—for example, parental strain recipients (C3H) and (A × C3H)F_1 hybrid cell donors (*5, 6*). This was accomplished by repeated intravenous injections of numerous viable F_1 hybrid spleen cells (up to 1.5 × 10^9 cells) into the parental strain recipients. Cells similarly injected in the strain combinations (C57B1 × C3H)F_1 hybrid----→ C3H and A----→ C3H failed to produce skin homograft tolerance (*6*).

Very recently, Michie and Woodruff (*7*) reported specific homograft tolerance in sublethally x-irradiated (500 r) parental strain mice (A-strain) which received multiple massive intravenous and intraperitoneal injections of (CBA × A)F_1 hybrid spleen cells. To study the induction of specific tolerance to

Table 1. Specific skin homograft tolerance in sublethally x-irradiated urethan-treated CBA mice injected with C3H spleen-marrow cells.

Treatment of recipients	C3H cells injected	Tolerant recipients (No.)		Skin homograft survival time (days)			
				C3H		BALB/c	
		C3H grafts	BALB/c grafts	Mean	Maximum	Mean	Maximum
None	None	0/5	0/5	16	17	12	12
500 rad	None	0/4	0/4	52	59	28	36
500 rad	Spleen*	5/5	0/5			22	28
Urethan + 500 rad	None	0/5	0/5	61	70	20	38
Urethan + 500 rad	Spleen*	5/5	0/5			22	33
Urethan + 500 rad	Marrow†	5/5	0/5			31	33
Urethan + 500 rad	Spleen plus marrow‡	4/5	0/5			15	19

* A single intravenous dose of 18×10^6 cells was injected 2 hours after irradiation. † A total of 42×10^6 cells was injected intravenously daily for 3 days after irradiation. ‡ The C3H graft on one mouse was rejected at 59 days.

allogenic skin grafts from donors sharing the same H-2 locus ($H\text{-}2^k$) as the recipients, we injected relatively small numbers of donor hemopoietic and lymphoid (C3H) cells into sublethally irradiated CBA mice and into irradiated mice which had been treated with urethan. The use of urethan with sublethal irradiation to enhance suppression of the homograft response in mice has been described (*8*).

Several groups of 3-month old male CBA/J mice were injected intraperitoneally with 20 mg of urethan daily for 2 days prior to irradiation. These mice, together with untreated controls, were exposed in a single sitting to 500 rad of 250 KVP x-rays (30 rad/min). The mice then received intravenous injections of either C3H/HeJ spleen cells (18×10^6 nucleated cells within 2 hours of irradiation), or bone marrow cells (a total of 42×10^6 cells daily for 3 days after irradiation), or both spleen and marrow cells. The cell donors were normal adults, 3 months old. On the day of the last marrow injection, all mice were tail-skin grafted with normal 3-month old male CBA/J ($H\text{-}2^k$), C3H/HeJ ($H\text{-}2^k$), and BALB/cJ ($H\text{-}2^d$) skin, and the grafts were observed for signs of rejection (*9*). No further treatment was given. The results are tabulated in Table 1.

In five nonirradiated, untreated controls, grafts had a mean survival time (MST), in days, of 16 for C3H and 12 for BALB/c. Grafts in four mice, treated with x-irradiation only (500 rad) showed mean survival time values of 52 and 28 for the C3H and BALB/c. A third group of five mice, which had received urethan prior to irradiation, showed a mean survival time of 61 for the C3H, and 20 for the BALB/c skin grafts. By contrast, the mice which received C3H marrow and spleen cells showed no response toward the C3H skin grafts but still rejected the BALB/c grafts in a manner no less vigorous than that of the 500-rad control group. All the mice in the irradiated group which received C3H spleen cells have retained their C3H grafts for over 130 days (as of the time of this writing) and have rejected the BALB/c grafts (MST 22 days). Similarly, all the mice in the groups treated with urethan and irradiation (500 rad) which received C3H spleen or C3H bone marrow cells have retained their C3H grafts (>130 days) and rejected the BALB/c grafts (MST 22 and 31). Finally, in a group of five mice which had received urethan, radiation, and both C3H spleen and bone marrow cells, four have retained the C3H grafts (the fifth lost its C3H graft after 59 days) and have rejected the BALB/c grafts in an essentially *normal* manner (MST 15 days). As expected, all the CBA recipients have retained syngeneic CBA grafts. None of the mice in the recipient groups died.

Procedures, as described here, have been used to develop tolerance in mice toward homografts in allogenic combinations differing at the H-2 locus, but with only rare success (*8*). One difficulty is that the injection of allogenic immunocompetent cells (C3H spleen cells, for example) into urethan-treated, sublethally irradiated LAF_1 recipients often resulted in death, presumably through graft-versus-host reaction (*7, 8*). Addition of hematopoietic cells did prolong homograft survival but, thus far, long-lasting tolerance has not been produced at will.

Our results show again (*8*) that urethan, with x-irradiation, suppresses the homograft response to a greater extent than does the radiation alone. They also show that skin homograft tolerance can be achieved in adult mice with sublethal radiation and injection of allogenic spleen or marrow cells (donor and host sharing the same H-2 locus). The unresponsive state produced is apparently *specific*, since under these conditions the skin grafts from a third strain (BALB/c) unrelated to the spleen-marrow donors were rejected. Furthermore, the rejection time of the BALB/c skin grafts was definitely accelerated where C3H spleen plus marrow cells were administered. This acceleration suggests that the injected donor cells (or their progeny) contributed to the rejection of the BALB/c skin grafts.

The mechanisms involved in induction of specific homograft tolerance under these conditions are a matter of conjecture. They presumably involve persistence of isoantigens as a result of the continued presence of replicating donor spleen-marrow cells. It is conceivable also that a limited graft-versus-host immunological reaction, which would act to suppress the host's own homograft response and yet not kill the animal, may too be involved (*10*).

Note added in proof: At this time, 165 days after treatment of the CBA recipients, the original C3H homografts are still intact and viable. Both tolerant and nontolerant CBA mice have since been regrafted with a second set of grafts 123 days after treatment. The tolerant mice again accepted the second C3H grafts and they rejected the second BALB/c grafts (MST = 14). This is further evidence for the existence of a state of specific homograft tolerance in these mice.

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