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Interfacial damage in ion-irradiated GaAs/AlAs superlattices

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Damage creation at GaAs/AlAs/GaAs interfaces during irradiation with MeV Kr and Ar ions was investigated by ion channeling experiments. The GaAs layers became amorphous while the AlAs layers showed unusual damage behavior. At one interface, AlAs on GaAs, an amorphous phase grows into the AlAs while at the opposite interface, GaAs on AlAs, the AlAs remains crystalline. The asymmetry is also observed in samples with five alternating layers; amorphization occurs at the two AlAs on GaAs interfaces but not the GaAs on AlAs interfaces. The rate at which the amorphous layer grows does not depend on the deposited damage energy alone, but rather depends on the ratio of the ionization to damage energies, demonstrating the importance of ionization in the damage process. At large ratios of ionization to damage energies, the growth rate can be zero or even negative.

Compositional disordering of GaAs/AlAs superlattices has been extensively investigated in recent years owing to its significance for fabricating highly structured quantum-well laser devices.[1] One method to disorder superlattices is ion irradiation, either directly by ion beam mixing[2,3] or coupled with thermal annealing.[4,5] Irradiation of GaAs/AlAs with heavy ions is particularly interesting because the GaAs layers become amorphous while the AlAs layers are resistant to damage, even at irradiation temperatures as low as ≈ 80 K.[6] This system is additionally interesting because the interfaces influence the response of the superlattice to ion irradiation. In previous studies, for example, it was reported that there is an asymmetry in the interdiffusion at AlAs on GaAs interfaces or GaAs on AlAs interfaces[7,8] and that the AlAs delays amorphization in the GaAs at both interfaces.[9] Despite these interesting results regarding interfacial effects and the resistance of AlAs to radiation damage, no clear picture of the underlying physical mechanisms has emerged. In the present study we show first that damage production at the interfaces, like interdiffusion, is asymmetric, in that an amorphous layer grows at one interface but not at the other. More importantly, we demonstrate that the growth rate of this layer depends on the ratio of the electronic to nuclear stopping powers. For too high ratios, the growth of the amorphous layer can be suppressed or even made negative. Thus, it is shown that ionization plays a clear and important role in the damage formation at interfaces in GaAs/AlAs superlattices during ion implantation. This had not been considered in previous investigations. The reversible growth of an amorphous layer, moreover, makes it possible to systematically investigate the effects of ionization on damage at interfaces.

Damaged GaAs/AlAs/GaAs specimens, containing three or five layers, were examined by ion channeling measurements. Because the depth resolution of Rutherford backscattering is $\approx 5-10$ nm, the specimens were fabricated with the thicknesses of the AlAs layers being ≈ 200 nm. The specimens were grown by atmospheric pressure metalorganic chemical-vapor deposition (MOCVD) in a vertical geometry, rotating disk reactor on a (001) Si-doped GaAs substrate. The growth temperature was 800 °C for which the background *n*-type carrier concentration is 10^{15} cm^{-3} for the GaAs layers and the background *p*-type concentration is $\approx 10^{18}$ cm^{-3} for the AlAs layers. Details of the MOCVD facility have been published elsewhere.[10]

The specimens were irradiated at 80 or 110 K, with either Ar or Kr ions in the energy range 0.50–2.7 MeV, and to doses between $1-4\times10^{16}$ cm^{-2}. An aperture with diameter 3.2 mm defined the beam on the specimens. Typical ion fluxes were ≈ 500 nA cm^{-2}; they were determined with a precision of better than 3% by measuring the rate at which the irradiation ions backscattered from a rotating tungsten wire which periodically intercepted the beam. The scattering rate was calibrated with a Faraday cup. Other fluxes were employed to test for effects of the defect production rate and possible experimental artifacts such as beam heating. The depth distributions of damage and ionization energies were obtained using TRIM.[11] Channeling measurements were performed with 2-MeV He ions using the ⟨100⟩ channel normal to the specimen surface.

The asymmetry of the damage at the two interfaces is illustrated in Fig. 1. Here spectra acquired in random and channeling directions are shown, both before and after irradiation at 110 K with 1-MeV Kr to doses of 1×10^{16} and 2×10^{16} cm^2. At the top interface, GaAs on AlAs, no evidence for damage is found, while at the bot-

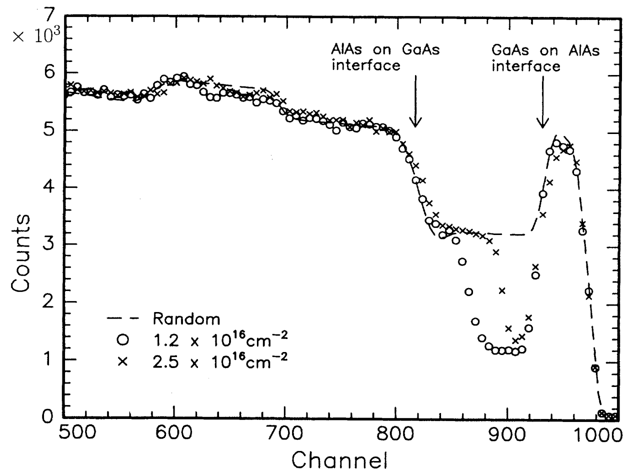

FIG. 1. Backscattering spectra of a 1.0-MeV Kr irradiated GaAs/AlAs/GaAs trilayer sample in the ⟨100⟩ channeling direction after fluences of 1.2×10^{16} cm^{-2} (○), and 2.5×10^{16} cm^{-2} (×). A random spectrum (— — —) is included for comparison.

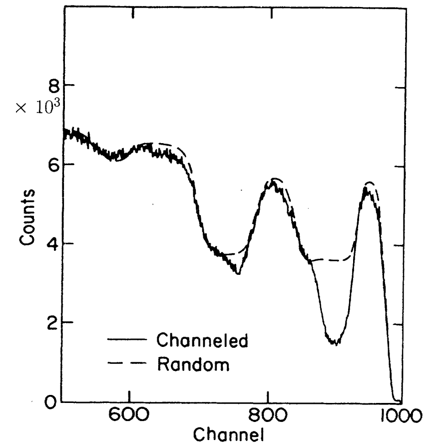

FIG. 2. Backscattering spectrum of a 1.0-MeV Kr irradiated GaAs/AlAs/GaAs/AlAs/GaAs pentalayer sample in the ⟨100⟩ channeling direction after a fluence of 1×10^{16} cm^{-2}. A random spectrum (— — —) is included for comparison.

tom interface, an amorphous layer grows from the interface into the AlAs, approximately linearly with fluence. The greater amount of damage revealed at the lower interface cannot be attributed to larger amounts of damage energy deposited at this position. For 1-MeV Kr irradiation, the damage is nearly the same at the two interfaces, yet amorphization occurs at the bottom interface after a dose of 1×10^{16} cm^{-2} while no amorphization is observed at the top interface, even at twice this dose. Table I lists the calculated damage energies at the relevant interfaces. The asymmetric damage is again illustrated in Fig. 2, but here in a five-layer specimen. Amorphization is observed at both bottom interfaces (AlAs on GaAs) but not at either top interface (GaAs on AlAs), demonstrating that the phenomenon is, indeed, an asymmetry with respect to the type of interface and not a consequence of depth of the interface.

The more fundamental observation of this work is the influence of ionization on the growth of the amorphous layer. Table I lists the growth rate of the amorphous phase, $d\Delta x/d\Phi F_D$, for Ar and Kr ion irradiations at various energies. Δx is the layer thickness, Φ is the ion fluence, and F_D, which is used for normalization, is the damage energy deposition per unit length x normal to the surface. The growth rates do not scale with damage energy. This is shown explicitly in Fig. 3 for the Kr irradiations, and shown in the table for the Ar irradiations. This is rather unusual radiation damage behavior, particularly for irradiations with MeV heavy ions since the damage is created in subcascades, i.e., energetic recoils are spaced far apart. Nevertheless, the normalized growth rates of the amorphous AlAs phase decrease dramatically with increasing energy for both Ar and Kr ions, and for a fixed energy, the normalized growth rates decrease with decreasing ion mass. Varying the ion flux by an order of magnitude, on the other hand, had no measurable effect on the normalized growth rates.

Since the integral damage energy ΦF_D does not alone control the growth rate of the amorphous phase, we consider the instantaneous damage energy and ionization energy densities, F_D and F_I, respectively, along each ion track; $F_i = dE_i/dx$. These quantities are listed in Table I for the various Kr and Ar irradiations. While F_D is a decreasing function of energy for a given ion, F_I increases with energy. For example, F_D decreases by a factor of ≈ 3 while F_I increases by a factor of ≈ 2 as the Kr energy is increased from 0.75 to 2.7 MeV. We thus consider the following possibilities for the reduced growth rates of the amorphous AlAs layer with increasing ion energy and de-

TABLE I. Normalized growth rate of the amorphous AlAs layer during irradiations with Ar or Kr ions at various energies.

Ion	Energy (MeV)	F_D (eV/Å) (back)	F_D (eV/Å) (front)	F_I (eV/Å) (back)	F_I (eV/Å) (front)	$\Delta X/\Phi F_D$ (Å^4/eV)
Kr	0.75	135	128	92	111	3.63
Kr	1.0	126	110	110	121	2.2
Kr	1.5	97	93	126	130	1.16
Kr	2.0	81	74	138	141	0.69
Kr	2.7	64	61	152	156	0.45
Ar	0.75	34	27	95	105	2.5
Ar	1.0	29	22	112	126	0.69
Ar	1.5	19	17	145	154	0
Ar	2.5	13	11	190	200	−6.0

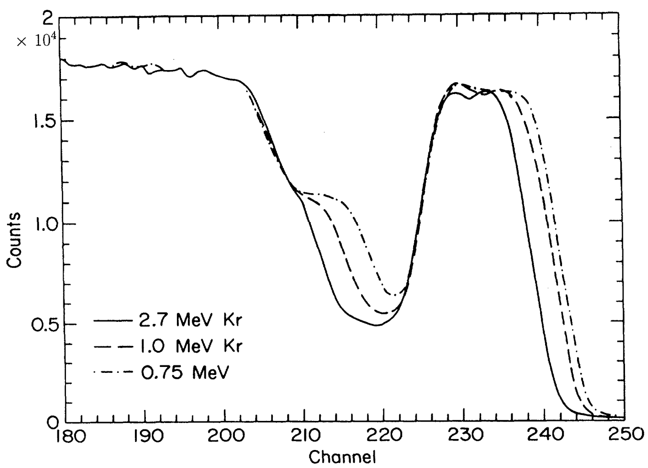

FIG. 3. Backscattering spectra of a GaAs/AlAs/GaAs trilayer sample in the ⟨100⟩ channeling direction after irradiation with 0.75-, 1.0-, or 2.7-MeV Kr ions. The doses are normalized to produce the same damage energy at the back interface.

creasing ion mass: (i) the decrease of F_D (this is contrary to our remarks about subcascades, above, but nevertheless, a possibility); (ii) the increase in F_I; or (iii) some combination of (i) and (ii). Possibility (i) is negated by the observation that the amorphization rate for the 0.75-MeV Ar irradiation is twice that for the 2.0-MeV Kr irradiation, since F_D for the former is half that for the latter. Similarly, values of F_I for 1.0- and 1.5-MeV Ar are smaller than those for 2.0- and 2.7-MeV Kr, respectively, while the amorphization rates are higher for the respective Kr irradiations; thus possibility (ii) is also negated. The growth rates of the amorphous phase must therefore depend on both damage and ionization deposition rates, increasing with F_D and decreasing with F_I. Note, for the 1.5-MeV Ar irradiation, the relative values of F_D and F_I are such that amorphization is completely suppressed. Whether large ratios of F_I/F_D can lead to negative growth rates of the amorphous layer was examined by first irradiating with 1-MeV Kr, to grow an amorphous AlAs layer, and subsequently irradiating with 2.5-MeV Ar for which the ratio of ionization to damage energies is high. A negative growth rate was, indeed, observed, suggesting that damage energy promotes the growth of the amorphous layer and that ionization energy leads to epitaxial regrowth of amorphous AlAs layers. In summary, these experiments have demonstrated that damage in GaAs/AlAs is strongly affected by ionization during ion implantation. It was further shown that damage in this superlattice, like interdiffusion, is asymmetric with respect to the interface. It is also shown that AlAs is not completely resistant to damage but can be rendered amorphous by an interfacial reaction. Amorphous AlAs is stable at temperatures at least as high as 300 K.

Although a complete model for understanding radiation damage and interdiffusion in GaAs/AlAs is not available, the important role of ionization reported here provides a firm basis for understanding these effects. We first note that ionization is known to stimulate defect processes in some semiconductors.[12] In GaAs, there is evidence that the underlying process is "recombination enhancement" of defect motion, whereby a majority carrier trapped at a point defect site captures a minority carrier. The pair recombines by a radiationless transition,[13] and the electronic energy is transferred to a phonon mode localized on the defect site, stimulating defect motion. Defect recovery induced by the minority carrier injection[14] and radiation-enhanced dislocation glide in a transmission electron microscope[15] have been tentatively explained on this basis. Maeda *et al.* pointed out that dislocations were efficient sites for nonradiative recombination in GaAs,[15] indicating the likelihood of a similar situation for amorphous-crystalline interfaces. We suggest, therefore, that the mechanism of recombination enhancement can stimulate crystallization, and that it explains why increased ionization during irradiation induces crystallization at amorphous-crystalline interfaces. This mechanism might also explain why defects do not build up in the AlAs. Why the amorphous phase forms at just one interface requires an asymmetry in the radiation geometry. Bode, Ourmazd, and Cunningham assumed that an internal electric field originating at the surface provided this asymmetry.[7] If we adopt this view, we can understand the asymmetric damage as follows. The irradiation produces many electron-hole pairs. The minority carriers drift to the top interface where they find majority carriers and stimulate defect motion and crystallization. Majority carriers drift to the opposite interface. Thus, amorphization is suppressed at one interface, but not the other. Only when much higher ionization levels are produced are a sufficiently large number of minority carriers available at the bottom interface to induce crystallization. Other mechanisms for the asymmetry may be possible, but they must include the effects of ionization. We currently prefer the recombination enhancement mechanism described above, since it provides a common basis for all three phenomena reported here. Although additional work will be required before a comprehensive damage model can be completed, we wish to point out that even before this is achieved, the results are important in themselves. First, they illustrate that heterogeneous interfaces, and possibly grain boundaries and dislocations, can strongly influence damage production and interdiffusion in electronic materials due to effects of ionization. These effects were readily observed in the present work because of the amorphous to crystalline transition, but other effects may occur in other materials. The experiments also show that comparing implantation results in heterostructural materials, or generalizing results from a single experiment, even on a qualitative basis, may be misleading, owing to the sensitivity of the radiation effects to the type of implantation ion, the ion energy, the depth of the interface, and its relative location with respect to the surface for the reasons cited here.

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