

THE INFLUENCE OF DOSE RATE AND ANALYSIS PROCEDURES ON MEASURED DAMAGE IN
 P^+ ION IMPLANTED GaAs

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Abstract It is shown that the damage measured following 40 keV P^+ implantation into GaAs at room temperature, depends significantly upon the ion dose rate as well as ion dose. Substantial post-implantation annealing of the damage at room temperature was also observed. The importance of these results in making intercomparisons between different ion species implantation in GaAs is discussed.

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INTRODUCTION

Earlier studies by one of the present authors and colleagues^{1,2} at McMaster University on P^+ and As^+ implantation of GaAs at low temperature (≈ 40 K) and room temperature suggested that only minor differences occurred in the damage produced by both species for low temperature conditions but substantial differences occurred between the two species in the damage measured for room temperature. These differences were small for high dose implants where the surface region was amorphised but increased with decreasing dose and damage fraction.

In related studies by another of the present authors and colleagues at Salford and at McMaster^{3,4,5} Universities it was found that (1) the disorder produced by a light ion, N^+ , implantation of GaAs at room temperature^{3,4} increased with increasing dose rate for given ion dose, the effect of dose rate becoming of diminishing importance with increasing dose and (2) for identical 40 keV Sb^+ implantations into GaAs at room temperature in both laboratories, the damage

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measured at McMaster exceeded that measured at Salford except at high doses where the results were very similar.

It was shown⁵ that this difference could be ascribed to the different analysis procedures at Salford and McMaster in which the Rutherford Backscattering/Channelling of 2 MeV He⁺ ions was used for disorder assessment but where, at McMaster, analysis directly succeeded implantation in the same substrate system whereas, at Salford, there was a variable time delay between implantation and analysis in two separate systems. Since in the P⁺ and Al⁺ implant studies^{1,2} the dose rates of the two species were not maintained identical it was felt possible, in view of the N⁺ and Sb⁺ (lighter and heavier ions than P⁺ and Al⁺) behaviour, that at least part of the apparently different damaging propensity could result from differences in dose rate. The present study therefore was aimed at observing any dose rate behaviour and this communication reports the results of 40 keV P⁺ implantation into GaAs. The investigation was also extended to assess whether time delay between implantation and analysis resulted in disorder annealing and the results of this work are also reported here.

(100 orientation single crystal GaAs was implanted with 40 keV P⁺ ions to doses of 10¹⁴, 10¹⁵ and 10¹⁶ cm⁻² at ion beam current densities of 0.5 μA/cm² and 5.0 μA/cm² (with an accuracy and stability over the implantation period of ± 5%) at room temperature. The GaAs substrates were tilted ~ 7° from the (100) axis in order to minimise channelling effects.

Analysis of the disorder resulting from these implantations was effected using RBS-channelling of 2 MeV He⁺ ions. The backscattered energy spectra were measured with a surface barrier detector employing 168° or 99° (to improve depth resolution) scattering angles, to determine total disorder and disorder-depth profiles. These spectra were recorded, for each implant condition, at fixed times after the completion of implantation.

A linear dechannelling correction procedure, subtraction of the unimplanted aligned spectrum and correction for the depth (energy) dependence of the backscattering cross section were employed to deduce, from the backscattering spectra, the numbers of displaced atoms and their depth profile.

Fig. 1 displays the backscattering data for the implant conditions described earlier and with a one hour delay between implantation and analysis. These raw data illustrate very clearly that, for each implant dose, the resulting disorder

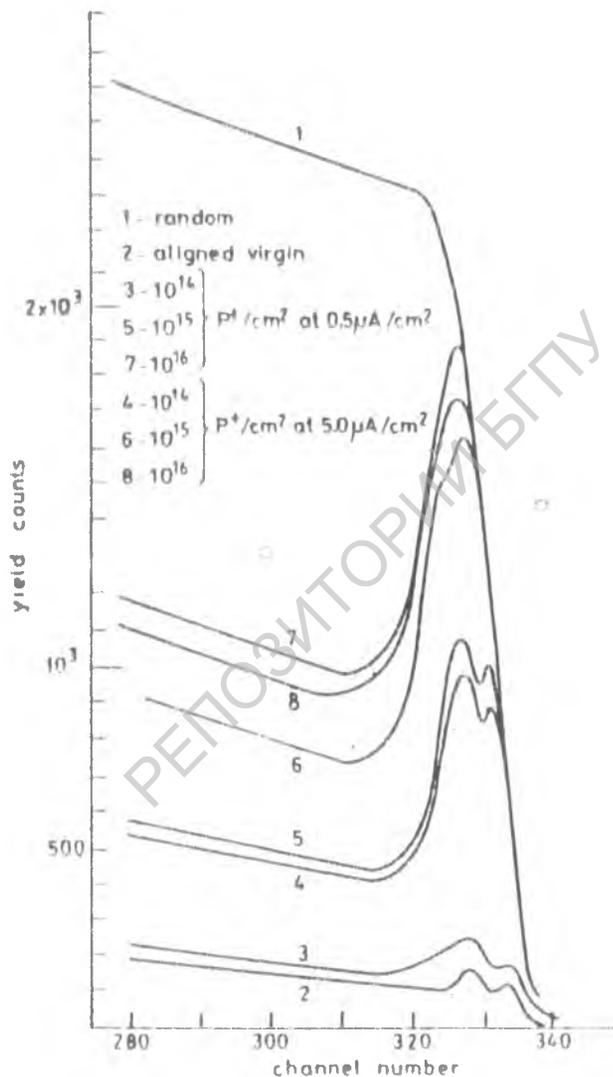


Fig 1 2MeV He⁺ ion RBS spectra from GaAs following 40keV P⁺ implantation at room temperature for different implant conditions. Scattering angle = 168°

increases with increasing dose rate but the relative increase is less at the higher doses. This result is totally equivalent to the earlier studies with $N^{+3,4}$ implants and clearly indicates that the differences do not arise because of annealing effects resulting from substrate temperature rise which would increase with increasing ion dose rate (beam power).

The damage/dose relationships deduced from Fig. 1 are shown in Fig. 2 for both implant dose rates and, for comparison, the equivalent study at McMaster^{1,2} with a substantially lower dose rate but with 60 keV P^{+} ions and more rapid analysis. It is clear that, for the higher doses in the present study, the quasi-saturation levels of damage, are rather independent of dose rate, quite identical to the earlier studies with $N^{+3,4}$ and Sb^{+5} implants. The 60 keV McMaster data^{1,2} reaches a higher quasi-saturation levels as anticipated from the increased depth over which disorder is created by the more energetic implants. At lower doses the present data 'brackets' the McMaster data, suggesting that the different parameters of ion energy, ion dose rate and analysis delay time are mutually involved.

The explanation of the form of the damage (N_D) / dose (Φ) behaviour and the dependence on ion dose rate (J) is probably closely analogous to that proposed earlier for N^{+} implantation^{3,4,5}. In this model the light ions produce simple defects and defect clusters, some of which are stable and some of which may migrate thermally and anneal or agglomerate at room temperature. As dose increases the defect density increases locally to form unstable zones which collapse to amorphousness and these amorphous zones increase in density until they overlap to produce a continuous amorphous layer. As ion dose rate is increased the defect generation, agglomeration and amorphous zone production processes compete more favourably with defect migration and annealing so that the resulting disorder is larger for a given ion dose. At high doses, where continuous amorphous layers develop, simple defect annealing, except for deeper in the solid beyond the amorphous layer, becomes unimportant and the disorder produced and measured becomes insensitive to ion dose rate.

Since such a model suggests defect migration and annealing during implantation it might be anticipated that further annealing could occur after annealing and so the implantation - analysis delay time studies were conducted with the results shown in Fig. 3 for three initial implant conditions. This figure indicates that for all implant conditions, including the quasi-saturation damage regime, there is

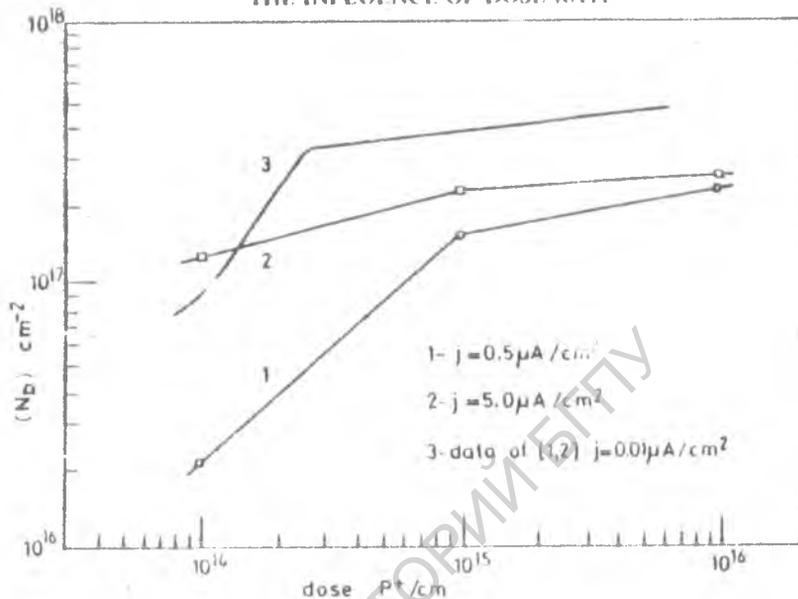


Fig. 2 Damage (N_D) as a function of P^+ ion dose (\int)

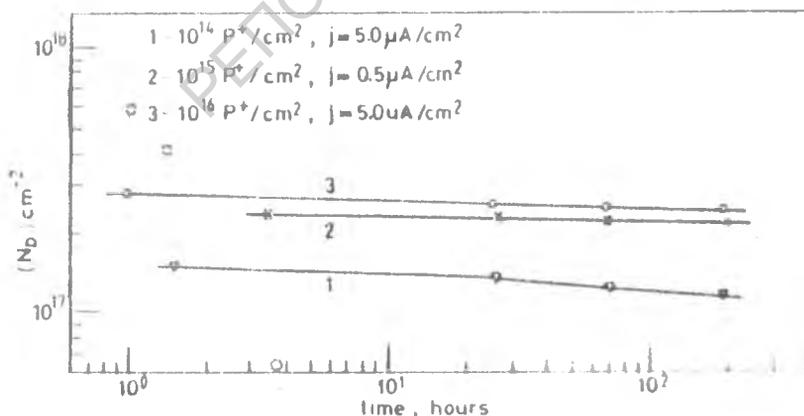


Fig. 3 Residual damage recorded as a function of delay time between implantation and analysis for 3 initial damage conditions

a measurable post implant annealing at room temperature. Each analysis was performed on a different area of the implanted substrate so that problems arising from additional disorder creation by the He^+ ions was minimised⁶. It is clear that the annealing is largest for the low dose, low dose rate (and thus low initial damage) condition and amounts to $\approx 20\%$ over a period of 100 hours. For higher initial disorder concentrations the annealing is substantially reduced. These results are similar to room temperature annealing studies^{7,8} in ion implanted InP where damage recovery continues for months after implantation and in which similar dependences of measured damage upon ion dose rate have been observed.

Since annealing occurs following implantation it is quite reasonable to expect it to have occurred during implantation also and this supports the model of damage generation and annealing discussed earlier and accounts, at least in part, for the observed dependence of measured damage upon dose rate. Whether this dependence of observed damage upon dose rate, clarified here for P^+ implants, can fully explain the earlier measured^{1,2} differences between P^+ and Al^+ implants is uncertain since the Al^+ ion dose rates were uncertain. It was argued¹ that this discrepancy resulted from different interactions of the implant species which either enhanced defect stabilisation or diminished defect annealing. The differences were, however, noted at implant and defect concentrations $< 1\%$ of the GaAs atomic density where it would seem unlikely that the implants would exert an influence over a substantial lattice distance. Moreover the low temperature studies, where defects are substantially immobilised in GaAs⁵, revealed little difference in damage production and stability for the two ion species. This would be entirely expected since the kinematics of collisions in the cases of the similar mass P^+ and Al^+ would be almost identical.

Consequently although a "chemical" inhibition difference in disorder stabilisation for P^+ and Al^+ implants in GaAs cannot be ruled out by the present studies with P^+ implants alone, caution in such interpretation is advocated since the effects of ion dose rate are quite extreme. This study serves as a further reminder^{3,4,5} that in both research and commercial implantation of GaAs not only must ion dose be carefully controlled but so also must ion dose rate and substrate temperature. Care and precision in analysis procedures must also be exercised.

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