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DOSE RATE EFFECTS ON DAMAGE FORMATION IN ION-IMPLANTED GALLIUM ARSENIDE

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ABSTRACT

The residual damage in GaAs was measured by ion channeling following implantation of either 100 keV $^{30}\text{Si}^+$ at temperatures of 300K or 77K, or 360 keV $^{120}\text{Sn}^+$ at 300K. For room-temperature Si implants and fluences between 1 and 10×10^{14} Si/cm², the amount of damage created was strongly dependent upon the ion current density, which was varied between 0.05 and 12 $\mu\text{A}/\text{cm}^2$. Two different stages of damage growth were identified by an abrupt increase in the damage growth rate as a function of fluence, and the threshold fluence for the onset of the second stage was found to be dependent on the dose rate. The dose rate effect on damage was substantially weaker for $^{120}\text{Sn}^+$ implants and was negligible for Si implants at 77K. The damage was found to be most sensitive to the average current density, demonstrating that the defects which are the precursors to the residual dose-rate dependent damage have active lifetimes of at least 3×10^{-4} s. The dose rate effect and its variation with ion mass and temperature are discussed in the context of homogeneous nucleation and growth of damage during ion irradiation.

INTRODUCTION

A persistent difficulty with ion implantation doping of GaAs is the inability to achieve high carrier concentrations for n-type dopants such as Si. Recently, the electrical activation of Si-implanted GaAs was shown to be dose-rate dependent, and a significant increase in activation was reported when the implantation rate was kept below $0.05 \mu\text{A}/\text{cm}^2$. [1,2] A subsequent investigation showed that the electrical activation of implanted Si can be correlated with dose-rate dependent residual damage in the GaAs lattice. [3] Dose rate effects on damage production in GaAs have also been reported for various other ions. [4-6] Due to the importance of obtaining high electrical activation for Si-implanted GaAs, it is important to understand the relationship between the ion implantation rate, residual damage, and electrical activation. Therefore, further investigation of the parameters which affect the dose rate dependence of the ion-induced damage is required. This contribution describes the influence of ion mass, implant temperature, and raster conditions on the dose rate dependence of damage in ion-implanted GaAs.

EXPERIMENTAL PROCEDURES

Substrates for ion implantation were undoped, n-type GaAs(100) wafers. In order to minimize channeling effects, samples were tilted 7° with respect to the beam direction during implantation. Samples were implanted with either 100 keV $^{30}\text{Si}^+$ or 360 keV $^{120}\text{Sn}^+$. These energies were chosen to produce equal projected ranges of 100 nm, as determined from computer simulations using the TRIM code. [7] Samples implanted at room temperature (RT) were simply clamped onto the nickel target holder. For implants at liquid-nitrogen temperature (LNT), samples were mounted on the liquid-nitrogen cooled sample holder with a colloidal silver glue to improve the heat transfer, in addition to being clamped. The beam was rastered electrostatically in two dimensions to obtain uniform implants over the sample area. The ion-induced damage was determined from ion channeling spectra taken along the $\langle 100 \rangle$ direction with 2 MeV He^+ and a scattering

angle of 160° . Ion scattering analysis was performed at RT. Samples implanted at LNT were stored in liquid nitrogen prior to analysis, and those implanted at RT were analyzed as soon as possible after implantation. Measurements of the annealing rate at RT indicated that with these precautions, systematic errors due to RT annealing are minor and do not affect the conclusions presented here.

RESULTS

Figure 1 summarizes measurements of the residual damage following implantation of 100 keV $^{30}\text{Si}^+$ into GaAs at both RT and LNT, and of 360 keV $^{120}\text{Sn}^+$ at RT. The integrated total damage N_d (scattering centers per cm^2) was determined from ion channeling spectra by subtraction of an appropriate dechanneling background, as indicated in the inset of Fig. 1. For RT Si implants at a fixed fluence of 2×10^{14} Si/ cm^2 , the damage increased dramatically with current density J , varying as $N_d \propto J^m$ where $m=0.37$ over a range of current density between 0.15 and 12 $\mu\text{A}/\text{cm}^2$. At a current density of 0.05 $\mu\text{A}/\text{cm}^2$, the damage deviated from this power-law dependence as a consequence of a change in the mode of damage growth at low damage levels which is described more fully below (Fig. 2). For the heavier Sn $^+$ ions, a dose of $\phi=3 \times 10^{13}/\text{cm}^2$ produced a damage level comparable to that created by Si implants of $\phi=2 \times 10^{14}/\text{cm}^2$. At this dose, there is also a dose rate effect for implantation of Sn $^+$ which can again be described by a single power law. However, the exponent m was found to be 0.20, indicating that the dose rate effect is considerably weaker for ^{120}Sn implants than for ^{30}Si at comparable damage levels.

The temperature dependence of the dose rate effect was examined for $^{30}\text{Si}^+$ implants at LNT. In this case, intermediate damage levels of approximately $1 \times 10^{17}/\text{cm}^2$ were obtained with $\phi=3 \times 10^{13}$ Si/ cm^2 . Interestingly, the measured damage was found to decrease slightly as J was increased. However, this inverse sensitivity to dose rate was strongly enhanced when the heat-conducting glue was not used to mount the samples, indicating

that this inverse effect is primarily the result of in situ annealing due to a dose-rate dependent temperature increase in the samples. Consequently, there is no evidence for a positive dose rate effect at LNT.

The dependence of damage on fluence is shown in Fig. 2 for selected implant conditions. In the case of RT implants (Fig. 2(a) and (b)), the curve of damage vs. fluence clearly exhibits two stages of damage growth. In the initial stage the damage grows sublinearly with fluence. The damage accumulation rate increases abruptly at a fluence of ϕ_c , and subsequently the damage grows superlinearly with fluence up to saturation. Damage saturation, which occurs at a limiting defect concentration $n_0 = 4.4 \times 10^{22} \text{ cm}^{-3}$ (the atomic density of GaAs), may be considered as a third stage of damage growth. In this stage, atomic displacements are created predominantly in predamaged or amorphous material and ion channeling is not sensitive to any additional changes which these displacements may produce. The rate of damage growth also increases with fluence for LNT implants. However, a sublinear growth stage (region of negative curvature) cannot be clearly identified in this case. Rather, these data are consistent with superlinear damage accumulation throughout the measured range.

The fluence ϕ_c , which marks the onset of superlinear growth, is dependent on current density and temperature. ϕ_c tends to decrease at high dose rates or low temperatures. If ϕ_c is defined as the intersection of the tangents to the two segments of each curve, then $\phi_c = 5.8, 0.9$, and $0.2 \times 10^{14} \text{ Si/cm}^2$ in Figs. 2 (a), (b), and (c) respectively. Since no sublinear region could be clearly identified for the LNT implants, assignment of the corresponding transition point is somewhat arbitrary and the quoted value should be regarded as a conservative upper bound. As a result of the multiple stages of damage growth, the measured value of the exponent m , which relates N_d to J , depends on the fluence used for the measurement. Values ranging from $m = 0.20$ to 0.67 were obtained for arbitrary choices of ϕ between 1 and $10 \times 10^{14} \text{ cm}^{-2}$ respectively. The exponent tended to increase at higher fluences and decrease at lower fluences. Therefore, meaningful

comparisons of the strength of the dose rate effect for different implant conditions can only be made at comparable damage levels. Accordingly, the data shown in Fig. 1 are representative of the intermediate levels of damage, from approximately 1 to $4 \times 10^{17} \text{ cm}^{-2}$, within the second stage of damage growth.

The dose rate effect is also sensitive to the manner in which the beam is rastered across the sample. As the beam is rastered, ions strike the surface in a series of periodic pulses. In order to investigate the effect of rastering, samples were implanted with the same fluence ($2 \times 10^{14} \text{ Si/cm}^2$) but with the raster parameters varied to produce three different pulse profiles as shown schematically in Fig. 3(a). In case A, the total source current was set at $2.4 \text{ } \mu\text{A}$, and the raster amplitude was adjusted to give a time-averaged current density of $0.05 \text{ } \mu\text{A/cm}^2$. Starting from this condition, case B was obtained by reducing the raster amplitude (which increases the pulse duration) to increase the average current density by a factor of 3 to $0.15 \text{ } \mu\text{A/cm}^2$. Similarly, C was obtained from A by increasing the source current by a factor of 3 to $7.2 \text{ } \mu\text{A}$ and increasing the raster amplitude to maintain the average current density at $0.05 \text{ } \mu\text{A/cm}^2$. Ion channeling spectra obtained from these three samples are shown in Fig. 3(b). The integrated damage in B was found to be four times greater than in A, while the damage in C was approximately equal to that in A. Therefore, the damage level is more sensitive to the average current density than to the instantaneous current density under these raster conditions.

DISCUSSION

Models for damage growth during ion implantation can be conveniently divided into two classes according to whether the damage is formed within or outside of individual collision cascades. In the first class, referred to as homogeneous, the residual damage is assumed to be nucleated from simple precursor defects which survive the cascade quench and diffuse, becoming more uniformly distributed throughout the irradiated volume. Nucleation of damage in the form of more stable defects, such as clusters or dislocation

loops, occurs as the density of these precursor defects increases with implantation dose. Due to the possibility of alternative processes such as recombination and defect trapping, the number of precursor defects originating from a given cascade may decay with time after the cascade quench. In such a case, homogeneous models will predict dose-rate dependent damage growth.[8,9] The alternative class of damage accumulation models (heterogeneous) assumes that damage is nucleated during the quenching of individual collision cascades. Due to the short time scales ($<10^{-11}$ s) for cascade quenching, dose rate effects cannot occur except at extremely high current densities ($J > 100$ A/cm² for typical cascade dimensions of 10^{-5} cm). Generally, both homogeneous and heterogeneous mechanisms are operative during ion irradiation. However, certain characteristics of the resulting damage growth kinetics, such as dose rate effects, can be used to identify the dominant mechanism.

It has been shown that the dose rate effect at RT is stronger in GaAs than in either Si or Ge for similar levels of damage.[3] Therefore, homogeneous damage nucleation and growth, if not dominant, is at least an important mechanism for damage accumulation in GaAs. The dose rate effect in GaAs becomes weaker as the ion mass is increased. For comparable levels of residual damage, the dose rate effect parameter m is approximately half as large for implantation of ^{120}Sn as for implantation of ^{30}Si . This is to be expected since heavier ions produce more dense cascades, which promote heterogeneous damage nucleation.[10] Another consequence of increasing the ion mass is to increase the damage efficiency, defined as the ratio of residual defects to atomic displacements. The damage per ion increased by about a factor of 15 for the Sn implants relative to the Si implants, while the number of atomic displacements per ion increased by a factor of four, as estimated from TRIM simulations. This is also consistent with the increased importance of heterogeneous damage nucleation. At cryogenic temperatures, the lifetimes of the primary defects are extended due to the lower mobilities, and as a result, dose rate effects

should become much weaker. This is exactly what was observed for Si-implantation of GaAs at LNT.

According to the ion channeling spectra in Fig. 3, the damage depends directly on the average current density J , and not on the instantaneous peak flux J_0 . Moore has similarly found the electrical activation efficiency for 100 keV Si implants to be more sensitive to J . [1] These results can be used to estimate a lower limit for the precursor lifetime τ . This lifetime corresponds to the rate of some unspecified process, such as annealing, recombination, or trapping, which removes active precursor defects from the irradiated volume in competition with damage nucleation. In the homogeneous model, dose-rate dependent damage results from the interaction of precursor defects created by separate ion impacts, and the dose-rate dependent damage level is controlled by the number $\sigma(\tau)$ of ions which arrive within a time interval τ at a particular point on the sample. It is convenient to distinguish two cases depending on the relationship between τ and the time interval between successive pulses t_2 ($t_2=10$ ms in the present experiments). First, suppose $\tau > t_2$, so that precursors created during one pulse can interact with those from one or more succeeding pulses. In this case, there is no significant reduction in the precursor population over time scales of the order of t_1 , which is much smaller than t_2 , and so the damage yield will be sensitive to the average rather than to the instantaneous current density, consistent with the observations. However, a dependence on average current density can also be obtained under certain circumstances in the case $\tau < t_2$. In the present experiments, interaction between precursor defects resulting from separate ion impacts within a single pulse is thought to be likely [11] and it is appropriate to consider this alternative case. In this case, $\sigma(\tau) = \int_{t=0}^{\tau} j(t) dt$, where $j(t)$ is the instantaneous local flux illustrated in Fig. 3(a), and $t=0$ refers to the leading edge of the ion pulse. If $\tau < t_1$, then for the given experimental conditions $\sigma^C(\tau) > \sigma^A(\tau)$ and the damage would be greater in C than in A, contrary to the observed result. Instead, it was found that $N_d^C \approx N_d^A$, therefore $\sigma^C(\tau) \approx \sigma^A(\tau)$ which is satisfied only for $\tau \geq t_1 = 3 \times 10^{-4}$ s (beam diameter divided

by sweep velocity). Using similar arguments, the same conclusion may be drawn from the increase of the damage observed in B. Thus, a lower limit for the precursor lifetime τ is 3×10^{-4} s. At the other extreme, τ must be smaller than the minimum implant time for which a dose rate effect was observed. This time was ≈ 3 s for $\phi = 2 \times 10^{14}$ /cm² at $12 \mu\text{A}/\text{cm}^2$.

CONCLUSIONS

Dose rate effects on damage growth have been observed for implantation of Si and Sn into GaAs at RT, but not at LNT. The strength of the dose rate effect implies that homogeneous nucleation of damage is an important mechanism for damage accumulation in GaAs. The damage was shown to increase with dose rate as $N_d \propto J^m$ over a large range of current densities. For intermediate damage levels between 1 and 4×10^{17} cm⁻², the exponent m was found to be approximately 0.37 for 100 keV Si implants and 0.20 for 360 keV Sn implants. As expected, the dose rate effect was weaker for Sn implants because of the larger average cascade energy density produced by these heavier ions. Due to the correlation between ion-induced damage and electrical activation, these results imply that the activation of heavier dopants can be improved through the use of low dose rates, although the benefit may not be as great as for lighter dopants such as Si. In these experiments, the damage was dependent on the average current density rather than the peak instantaneous current density. Accordingly, limits on the lifetime of the dominant precursor defect were estimated to be 3×10^{-4} and 3 s.

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FIGURE CAPTIONS

Figure 1. The depth-integrated damage is shown as a function of dose rate for implantation of 100 keV Si and 360 keV Sn at constant fluences. For RT implants, the damage obeys a power-law $N_d \propto J^m$ with the indicated value of m . The inset illustrates the determination of damage from a typical ion channeling spectrum.

Figure 2. The peak concentration of damage is shown as a function of fluence for 100 keV Si implanted with various dose rates at (a), (b) RT, and (c) LNT.

Figure 3. (a) A single pulse of the time-dependent ion flux which results from beam rastering is illustrated schematically for each of three experimental arrangements. The peak instantaneous flux J_0 and the pulse duration t_1 were varied as described in the text for cases A, B, and C. The mean time between pulses was 0.01 s in each case. (b) Ion channeling spectra for samples implanted under the flux conditions represented in (a). The integrated damage in cases A and C was similar, but was four times greater in case B.

Figure 1

Figure 2

Figure 3

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