Effects of low-dose Si implantation damage on diffusion of phosphorus and arsenic in Si

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The effects of low-dose Si implantation damage on diffusion of low-concentration P and As in Si wafers are investigated. Dopants are implanted at a low dose and subsequently preannealed to remove any self-damage. An enhanced diffusion of P is observed by directly comparing dopant profiles in damaged and undamaged regions. Monitoring effective diffusivity of P at various annealing temperatures and times reveals that the enhanced diffusion is a transient process with a time constant which is larger at lower temperature. This enhancement is larger and of longer duration the lower the annealing temperature is. In contrast to P, As diffusion in the damaged region does not show any enhancement. This implies that the defects induced by the Si implants have separate mechanisms for interaction with each type of dopant.

Extensive knowledge of transient diffusion of dopants due to ion implantation damage in silicon during annealing has been required for designing shallow junctions in submicron devices. The transient diffusion is thought to be caused by the complex interactions of dopants, point defects, and extended defects whose concentrations are changing with time and location during thermal annealing cycle. Recent studies have reported transient enhanced diffusion of implanted boron during furnace annealing and rapid thermal annealing (RTA).6-10 Most of these experiments showed anomalous movement of high-concentration boron profiles on substrates amorphized by high-dose Si implantation. Packan and Plummer6 observed substantially enhanced diffusion of low-concentration boron in deep regions of silicon wafers implanted with low doses of silicon and showed its dependence on the implant dose and annealing temperature.

Enhanced diffusion was also observed during RTA for phosphorus implanted at a dose below the amorphization threshold.7,8 Additional studies have looked at enhanced diffusion of phosphorus produced by annealing the samples amorphized with high-dose silicon implants.9,11 Servidori et al.12 reported enhanced diffusion of predeposited phosphorus due to low-dose silicon implants during furnace annealing at 600–750 °C and its dependence on relative depth position of defects. In the case of arsenic, previous results contradict each other. Some researchers showed initial transient enhanced diffusion of arsenic during RTA,13,14 while others observed no initial transient,15 or only very small enhancement of arsenic profiles with Si damage implants.9,10,16

In contrast to previous work, this study looks at the effects of low-dose silicon implantation damage on lightly doped phosphorus and arsenic layers. To build physical models for the implantation damage, it is necessary to separate the effects of high-concentration diffusion, extended defects, and point defects. The experimental procedure used for this study was designed to diminish any possible complexity of transient diffusion mechanisms due to evolution of extended defects so that only point defects injected directly by implantation could contribute the diffusion kinetics. A 250 Å oxide layer was grown on (100) oriented, 80 Ω cm, n-type silicon wafers. Half of the wafers received a phosphorus implant at 60 keV with a dose of 2 × 10^{13}/cm². The remainder of the wafers received an arsenic implant with the same implantation conditions. The implantation energy and dose are low enough to produce low-concentration dopant profiles, which eliminates high-concentration diffusion effects. The subsequent procedure is common to both phosphorus and arsenic wafers. All the wafers were annealed for 10 min at 900 °C in order to remove any point defects induced by implanted dopant itself and to activate the dopant. The doping profiles at this step were measured through spreading resistance profiling (SRP). The wafers were shadow masked, such that half of each wafer was implanted with 28Si at 60 keV to a dose of 1 × 10^{14}/cm², which is below that necessary to amorphize the surface. This low-dose silicon implant produces a limited amount of damage which results in point defects with very few extended defects.17 The samples were annealed for several different times at various temperatures ranging from 800 to 1100 °C using both RTA and furnace annealing. The dopant profiles were then measured from these annealed samples by using SRP.

The results from phosphorus wafers are described first. Figure 1 shows the phosphorus dopant profiles of undamaged and damaged regions on a sample which was annealed at 800 °C for 30 min. Large enhancement of phosphorus diffusion due to the low-dose Si implant damage can be seen directly compared with the diffusion without implant damage. The peak concentration of the profile at this lowest temperature is about 1 × 10^{18}/cm³, which is below the intrinsic carrier concentration at all anneal temperatures, and thus precludes any extrinsic diffusion.

Figure 2 shows the behavior of phosphorus profiles on damaged regions for increasing annealing times at 900 °C. Here the enhanced motion of dopants saturates after the prolonged furnace annealing times longer than 15 min. This implies the transient damage enhancement must be considerably shorter than the anneal time. Since the measured
profiles are nearly Gaussian, a depth-independent effective diffusivity can be applied to the entire dopant profile.

In Fig. 3, the temperature and time dependence of the ratio of the phosphorus damage-enhanced effective diffusivity \( D \) to normal diffusivity \( D^* \) is shown. This ratio increases abruptly as annealing temperature decreases, which means the amount of transient enhancement is the largest at the lowest temperature. This is consistent with a previous report on boron transient damage-enhanced diffusion due to low-dose silicon implants. The ratio at 15 s ranges from about 300 at 900 °C to 3 at 1100 °C. As time increases, the time-averaged effective diffusivity decreases to the default value \( D^* \), and the rate of this decrease is lower for lower temperature. This indicates that the transient enhancement occurs in a short time interval which is different at different anneal temperatures.

The absolute movement of profiles on damaged regions can be represented as square root of two times the product of effective diffusivity and anneal time \( \sqrt{2D_i} \). The difference of these values for each pair of regions with and without silicon implants, \( \Delta \sqrt{2D_i} = \sqrt{2D_i} - \sqrt{2D^*i} \), quantifies the net enhanced movement of each profile as shown in Fig. 4. At times during which there is a damage enhancement, this quantity will be increasing. After the damage enhancement is gone, the value will saturate and then decay. As the anneal temperature increases, the net enhancement of diffusion is seen to decrease generally, which is consistent with the case of phosphorus diffusion on amorphized silicon in previous studies. At 800 °C, \( \Delta \sqrt{2D_i} \) is nearly constant from 15 to 30 min as was observed by Angelucci et al. in boron enhanced diffusion behavior over the same time range. At 900 °C, the value initially increases during the short time RTA anneals, and then begins to decline during the longer time furnace anneals. The time constant of the enhancement at 900 °C is estimated to be 15 to 30 s. At 1000 °C the time constant must be less than 15 s, as \( \Delta \sqrt{2D_i} \) becomes already constant at that time. 1100 °C RTA data show decrease of \( \Delta \sqrt{2D_i} \), which corresponds to its behavior of large time anneal at 900 or 1000 °C. This implies that the time constant of tran-
sient diffusion at 1100 °C is far less than that at lower temperature. The overall trend of $\Delta \sqrt{2D_i}$ variation shows that the time constant of transient enhanced diffusion decreases with increasing anneal temperature, which is in agreement with the previous observation on phosphorus and boron diffusion under amorphizing implant conditions.4,10

Arsenic diffusion, however, did not show any enhancement effect from the low-dose silicon implants. There was no discernible difference between the arsenic profiles on the undamaged and the damaged regions. This result agrees with some of the previous results of arsenic.9,15 There are several possible reasons for this difference. First, phosphorus is known to diffuse mainly by interstitialcy mechanism, and arsenic by a vacancy mechanism.18 However, Monte Carlo calculations indicate that the defects are created in roughly equal numbers. Second, the arsenic enhancement may be less than our measurement error. Because arsenic diffuses slower than phosphorus, a moderate enhancement of the diffusivity would have been undetectable.

In summary, a marked enhanced diffusion of phosphorus is caused by nonamorphizing silicon implants. This dopant profile redistribution is basically a transient, short-time process which occurs due to the interaction of dopants with damage-induced excessive amount of point defects. The resultant effect of this enhanced diffusion is larger and of longer duration at lower annealing temperature. Arsenic under the same experimental conditions showed no transient enhanced diffusion due to damage. The experiment was designed to minimize high-concentration diffusion effects and the creation of extended defects.

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