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Evidence for ion irradiation induced dissociation and reconstruction of Si-H bonds in hydrogen-implanted silicon

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We observe that H-related chemical bonds formed in H-implanted Si will evolve under subsequent ion irradiation. During ion irradiation hydrogen is inclined to dissociate from simple H-related defect complexes (i.e., VH_x and IH_x), diffuse, and attach to vacancy-type defects resulting in new platelet formation, which facilitate surface blistering after annealing, a process completely inhibited in the absence of ion irradiation. The understanding of our results provides insight into the structure and stability of hydrogen-related defects in silicon. © 2008 American Institute of Physics.

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Hydrogen in Si has been studied extensively for its scientific and technological importance in silicon based electronic devices. The major role of hydrogen is the passivation of both shallow donor states and deep impurities or other defects through the formation of complexes. During device operation, electronic defects are created that limit device lifetimes, and hydrogen has been observed to be involved in this degradation process. In fact, nonthermal dissociation of Si-H bonds through radiation, which often occurs in outer space, is found to be responsible for hot-electron degradation in silicon based transistors,² as well as light-induced degradation in α -Si:H solar cells (Staebler–Wronski effect).³ Furthermore, modifications and structural transformations accompanied by the loss of hydrogen are often observed in hydrogen composed materials subjected to irradiations.⁴ Current models^{5,6} for the depletion of hydrogen imply that H₂ molecules are formed from two diffusing H radicals, following the breaking of H related bonds by the passage of high-energy ions. However, experimental observation has not been presented so far.

A detailed study on the effects of energetic ions on the stability of Si–H bonds is performed using Fourier transform infrared (FTIR) spectrometry. We observed Si–H bond breaking in monovacancies or interstitials and the formation of new Si–H bonds and reconstruction within vacancy clusters, which leads to the nucleation and growth of "seed" platelets. The newly formed platelets are associated with blister formation in the sample after annealing, an event that does not occur in samples without irradiation. In addition, our experimental results show conclusively the origins of the *reverse annealing* effect observed in thermally annealed H implanted Si.

Hydrogen implantation and subsequent ion irradiation were performed at room temperature on 450-nm-thick silicon-on-insulator (SOI) wafers with a 1 μ m thick SiO₂ buried layer. 30 keV protons at a dose of 3×10^{16} cm⁻² were

implanted into the top Si layer of a SOI wafer to create Si–H complexes. This dose was chosen to avoid blister formation after thermal annealing. 110 keV protons at a dose of 5×10^{16} cm⁻² were subsequently used as an irradiating species to introduce electronic excitations in the vicinity of the Si–H complexes, which would stop in the buried SiO₂ layer⁷ and would not contribute to the study of hydrogen-related defects in the top Si layer by FTIR. All the implanted specimens, with or without irradiation, were annealed in vacuum at 500 °C for 30 min.

Following annealing, scanning electron microscopy (SEM) analysis shows that surface blistering occurs for the implanted-plus-irradiated sample and not for the implanted-only sample [insets of Figs. 1(e) and 1(f)]. For the as-implanted sample, transmission electron microscopy (TEM) shows that defects in the form of platelets⁸ are present [Figs. 1(a) and 1(b)]. However, the density of plate-

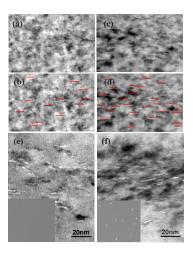


FIG. 1. (Color online) TEM observation of H-platelets in the implanted region: [(a) and (b)] as implanted, [(c) and (d)] after subsequent irradiation, (e) implanted sample after annealing, and (f) subsequently irradiated sample after annealing. Annotations are added on images (b) and (d) to show the location of platelets. The insets in (e) and (f) are the corresponding SEM micrographs after annealing.

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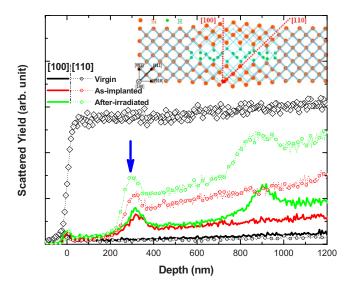


FIG. 2. (Color online) Comparison of $\langle 100 \rangle$ and $\langle 110 \rangle$ aligned spectra from 30 keV hydrogen implanted Si before and after 110 keV H ion irradiation. Two dimensional lattice structure around (100) platelet is illustrated in the inset. (The depths for $\langle 100 \rangle$ and $\langle 110 \rangle$ aligned spectra were calibrated to account for the different tilt angles along different crystal axes).

lets is significantly enhanced after irradiation [Figs. 1(c) and 1(d)], while the mean size remains nearly the same compared to the as-implanted sample. A statistical analysis of these TEM data⁹ shows that the overall volume occupied by the platelets is increased by a factor of 2 after irradiation. Correspondingly, our analysis of the as-implanted and irradiated samples following thermal annealing at 500 °C [Figs. 1(e) and 1(f)] shows that the total platelet volume in the latter is also larger than that in the former. Overall, the further radiation stimulates processes that lead to the nucleation and growth of new platelets, resulting in an increased density of platelets. Thus, the volume occupied by the platelets in the irradiated sample after annealing is consequently enhanced relative to the sample without irradiation. Blisters, which apparently require a minimum platelet volume to occur, are only observed on the surface of irradiated samples but not found for samples without irradiation.

Platelet nucleation and growth are also evident from channeling experiments. A damage peak corresponding to 30 keV H implantation increases after irradiation for ion channeling along the $\langle 100 \rangle$ and $\langle 110 \rangle$ axes, Fig. 2. (Note that Si substrate was used instead of SOI for better channel characterization.) The damage peaks for (110) channeling are significantly larger than that obtained for $\langle 100 \rangle$ channeling. For pure displacement damage created by the implantation process, the more open (110) channel should produce less scattering than the less open (100) channel. However, the reverse trend is observed in Fig. 2. The enhanced backscattering for the (110) channeling condition can be understood by considering the effect of platelets on the displacement field of the surrounding lattice. The expansion of the lattice in the direction normal to the plane of the platelet, i.e., along $\langle 100 \rangle$ for a (100) platelet, creates a shift in the registry of off-normal lattice planes on either side of the platelet. Therefore, ions traveling in $\langle 110 \rangle$ channels above the platelet will encounter a plane of atoms on the outer side of the platelet, like a stacking fault 11 (see inset in Fig. 2). Since ion channeling is sensitive to atomic displacements perpendicu-

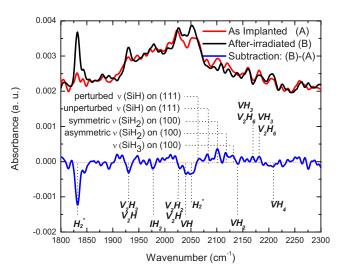


FIG. 3. (Color online) Si–H chemical-bond evolution under ion irradiation as observed by MIR-IR spectra. The changes in absorbance of Si–H bonds are extracted by the subtraction. Several of the modes presented in these data correspond to more than one defect, i.e., $(V_2H \text{ and } V_2H_2)$ and $(VH_3 \text{ and } V_3H_4)$

lar to the channel, the lattice expansion along the $\langle 100 \rangle$ direction is essentially invisible to the (100) channeled ion. The stacking fault nature of the (100) platelets should be primarily visible to ion channeling along $\langle 110 \rangle$ orientations. The direct backscattering observed for (100) axial channeling results from the local atomic displacements that take place near the surface of the platelet, which result from a combination of atomic reconstructions at the surfaces of the platelet 12,13 and local strains from pressure buildup as H₂ content increases in the platelet. Thus, as the platelet density increases after irradiation, an increase in the direct scattering peak along either the $\langle 100 \rangle$ or $\langle 110 \rangle$ axis is expected, with an enhanced signal for the latter, consistent with TEM data. In fact, the increase in the direct scattering peak in thermally annealed H implanted Si known as reverse annealing has been long observed. ¹⁴ Many researchers ^{15,16} speculated the platelets might be a candidate for the source of displacements. Here, we show the first conclusive evidence that the reverse annealing effect is truly due to the platelets.

The evolution of Si–H complexes during ion irradiation was monitored by FTIR spectrometry using multiple internal reflection (MIR) geometry (Fig. 3). Both as-implanted sample and irradiated samples exhibit strong broadband absorption from 1800 to 2300 cm⁻¹, which consists of various discrete Si-H stretching modes associated with siliconhydrogen (SiH_x) bonds, vacancy-hydrogen (V_xH_y) bonds, and interstitial silicon-hydrogen (I_xH_y) bonds, as assigned in Fig. 3. 8,17 The modes at 2070–2130 cm⁻¹ are generally attributed to Si-H bonds at extended internal surfaces, i.e., platelets. 18 As observed from the difference between two FTIR spectra in the bottom of Fig. 3, subsequent ion irradiation produces a general attenuation of VH₁₋₄ defects, unsaturated hydrogen-divacancy defects, hydrogen-saturated interstitial IH₂, as well as significant loss of the H₂* modes. In contrast, the Si-H stretching modes due to internal hydrogen-terminated surfaces at 2070-2130 cm⁻¹ and the modes at 2182 cm⁻¹ corresponding to VH₃ or V₂H₆ are all enhanced relative to the as-implanted spectra. (It should be metry in vibration, are hardly distinguishable experimentally and theoretically, and have been suggested to be V_2H_6 in H-implanted Si). 17

An energetic ion incident on a solid loses its energy by elastic collisions between the ion and target nuclei (nuclear stopping) and by excitation and ionization of target electrons (electronic stopping). The electronic and nuclear stopping powers for 110 keV proton ions amount to 123 and 0.26 eV/nm at the depth of the implanted H, respectively. Therefore, almost 99.8% of all energy deposited in this region is locally stored as potential energy in the form of trapped excitons (localized electron-hole pairs). Such excitons are formed by an electron, trapped in the conduction-band tail state and a hole, trapped in the valence-band tail state of the Si–H bond in the Si–H complexes sites. ¹⁹ The energy released during nonradiative recombination of such band tail excitons can result in the breaking of Si–H bonds and reconstruction of new bonds. ²⁰

As calculated by Van de Walle and Neugebauer,²¹ the binding energy of H to the self-interstitial (i.e., IH₂) is about 2.4 eV (relative to free atomic H). The bonding energy of H to Si in the form of H₂* is about 1.65 eV.²² Robertson and Estreicher²³ calculated the binding energies for the different VH_n complexes with the energy decreasing from 3.6 to 3.0 eV with *n* increasing from 1 to 4. Estimates of the electronic energy deposited during proton irradiation show that there is sufficient energy for Si-H bond breaking;^{6,24} the actual bonding state after ion irradiation depends on the competition between bond dissociation and recombination. If there are no other potential hydrogen traps within a critical recombination distance, the freed H atom may fall back into its original bonding configuration. For H bound in H₂, the Si–Si bond will reconstruct after breaking the Si-H bond, thus removing the recombination site for H. The freed H will diffuse until it locates another trap or another free H to form a H₂ molecule. The decrease in the IH₂ FTIR signal results from the migration of interstitials, which are highly mobile at 300 K.²⁵ An interstitial released from IH2 will rapidly diffuse away from the original defect, to the surface, or annihilate with free vacancies, and the released H will react with other trapping sites. While VH is likely to be mobile in Si, its diffusivity is much lower than a free single vacancy and VH_n with n=2, 3, or 4 are immobile at room temperature even during irradiation.²³ Because of the high diffusivity of vacancies in Si, the VH absorption peak decreases after ion irradiation similar to IH₂. On the contrary, because of the low mobility of VH and even lower mobility of VH₂ and VH₃, the absorption peaks corresponding to VH₂ and VH₄ decrease only very slightly after irradiation. Changes in VH3 cannot be assessed because of signal overlap with the V₂H₆ absorption peak. Since divacancies and V₂H/V₂H₂ are immobile, they are likely traps for freed H which explains why their signal decreases as the V₂H₆ signal at 2182 cm⁻¹ increases.

It is known that the prevailing defects in H implanted Si are of the vacancy type. ¹⁵ In the presence of H, vacancies can be stabilized by forming the extended vacancy-H complexes, as observed by FTIR in Fig. 3. When in-plane compressive stresses are present, which is typical of ion implanted materials, molecular dynamics studies have shown that vacancies tend to form small planar hexagonal ring clusters (HRCs) that orient in the plane of stress, ²⁶ making them ideal sinks for liberated H from H_2^* , IH_x , or VH_x defect com-

plexes. Hydrogen decorated HRCs are one possible source for the H-platelets, ²⁷ as observed after ion irradiation in Figs. 1(c) and 1(d), and the enhancement of the Si–H stretching modes at 2070–2130 cm⁻¹. Other defects may also contribute to platelet nucleation; the decrease in V₂H/V₂H₂ signal along with the corresponding increase in V₂H₆ suggests the transformation from the former to the latter, when freed hydrogen bonds pre-existing H-divacancy defects.

In conclusion, we have found that ionizing radiation deposited during ion irradiation can induce the breaking and reconstruction of H-related bonds in Si. We propose that H atoms liberated by irradiation can diffuse and attach to vacancy-type defects and form new platelets. Furthermore, our channeling data and proposed model conclusively show that the reverse annealing effect observed in thermally annealed H implanted Si is due to platelets.

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