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Investigation of stress-induced ⟨100⟩ platelet formation and surface exfoliation in plasma hydrogenated Si

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We have studied the mechanisms underlying stress-induced platelet formation during plasma hydrogenation. The stress is purposely introduced by a buried SiGe stained layer in a Si substrate. During plasma hydrogenation, diffusing H is trapped in the region of the SiGe layer and H platelets are formed. The platelet orientation is controlled by the in-plane compressive stress, which favors nucleation and growth of platelets in the plane of stress and parallel to the substrate surface, and ultimately leads to controlled fracture along the SiGe layer. Also, the Si/SiGe/Si structure is found to be more efficient in utilizing H for platelet formation and growth compared to H ion implanted Si because there are fewer defects to trap H (e.g., V_nH_m and I_nH_m); therefore, the total H dose needed for layer exfoliation is greatly reduced. © *2007 American Institute of Physics.*
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It is well established that hydrogen implantation in Si is capable of inducing surface exfoliation. Several mechanisms have been proposed to explain this effect.[1–3] Weldon *et al.*[1] has suggested that the implanted H produces extended planar defects (platelets), which can trap H_2 molecules which build up the internal pressure, cause coalescence, and finally lead to cracking. While the general features of this exfoliation description are commonly accepted, many of the details associated with the various processes leading to platelet nucleation and H_2 molecule trapping are still under discussion. For example, it is well-known that platelets only form on {111} planes during plasma hydrogenation[4] and exfoliation is not observed. However, when hydrogen is introduced into Si by ion implantation, platelets are primarily observed to form parallel to the substrate surface[5] and exfoliation is observed. Recent work by Nastasi *et al.*[6] showed that the platelet orientation in both H ion implanted and plasma hydrogenated Si could be explained by a Volmer-type nucleation model that accounted for the in-plane compressive stress that is present in the implanted material.[7,8] Since the stress in ion implanted materials results from radiation defects, it is unclear if the model proposed by Nastasi *et al.* is also appropriate for situations where an external stress is introduced by other means. In this work, we explore platelet formation and surface exfoliation in plasma hydrogenated single crystalline Si where a region of plane-stress is introduced by a buried SiGe strain layer.

A heterostructure composed of a 5-nm-thick epitaxial $Si_{0.80}Ge_{0.20}$ layer followed by a 150-nm-thick crystalline Si capping layer was grown on a ⟨100⟩ *p*-Si substrate at 650 °C using molecular beam epitaxy. The samples were subjected to low pressure (1 mTorr) plasma hydrogenation in pure hydrogen at 300 °C, in a distributed electron cyclotron resonance plasma reactor[9] with a low frequency (2 kHz) bias of approximately −100 V. Durations of plasma treatments varied from 30 min to 2 h. Scanning electron microscope (SEM), elastic recoil detection (ERD), and multiple internal reflection infrared spectroscopy (MIR-IR) were used to characterize the samples after hydrogenation.

Figure 1 presents SEM micrographs of the Si/SiGe/Si samples after hydrogenation. For the shortest treatment (0.5 h), only tiny blisters (∼30 nm) are detected [Fig. 1(a)]. As the hydrogenation proceeds, these small blisters grow and coalesce. For 1 h hydrogenation [Fig. 1(b)], large blisters, with a diameter generally of less than 1 μm have developed. The 1.5 h hydrogenated sample [Fig. 1(c)] shows a very high density of blisters with blister diameters between 1 and 1.5 μm. However, the morphology has visibly changed when the hydrogenation is extended to 2 h [Fig. 1(d)], where many large exfoliated blisters (∼3.5 μm) surrounded by a scattering of small blisters can be seen. This local exfoliation is due to the built-up molecular hydrogen which promotes crack propagation along the SiGe layer.[10]

Figure 2(a) shows ERD hydrogen depth profiles from hydrogenated Si/SiGe/Si samples. The amount of integrated hydrogen in each peak is plotted as a function of hydrogenation time in Fig. 2(b). Figure 2(a) shows that even at the lowest hydrogenation exposure time, the H concentration is

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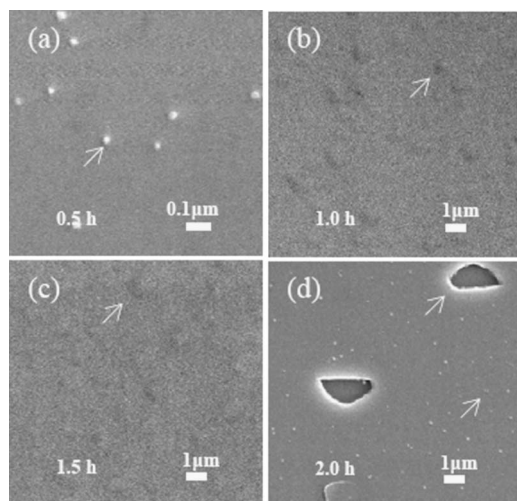
FIG. 1. SEM micrographs from Si/SiGe/Si after plasma hydrogenation for various durations: (a) 0.5, (b) 1.0, (c) 1.5, (d) 2.0 h.

FIG. 3. MIR-IR spectra from hydrogenated Si/SiGe/Si with different hydrogenation durations: (a) 1800–2300 and (b) 2000–2200 cm^{-1}.

well above the solubility limit in Si.[11,12] The peak H concentration increases with hydrogenation time in the range of 0.5–1.5 h. However, the H concentration drops significantly in the 2 h hydrogenated sample because of out diffusion of gaseous H_2 from exfoliated blisters along with the loss of bonded hydrogen from inner walls of exfoliated blisters.

The hydrogen concentration required for blister formation is about 1×10^{16} cm^{-2}, which is much lower than the fluence of 6×10^{16} cm^{-2} necessary for commercial ion-cut technology, and comparable with the total fluence of 1.75×10^{16} cm^{-2} used in H/He coimplantation for layer transfer.[13] This phenomenon can be explained by the intrinsic difference between ion implantation and plasma hydrogenation. In contrast to ion implantation, the energy of plasma H ions is not high enough to create significant concentrations of vacancies and interstitials, which in ion implanted samples trap H in the form of V_nH_m or I_nH_m defects.[14] Therefore, for plasma hydrogenation, the lack of irradiation damage means that there are fewer traps competing for H other than the buried SiGe strained layer. In this way, H introduced by plasma hydrogenation can be more efficiently utilized for platelet formation.

The bonding between H and Si that results during plasma hydrogenation is investigated using MIR-IR (Fig. 3).
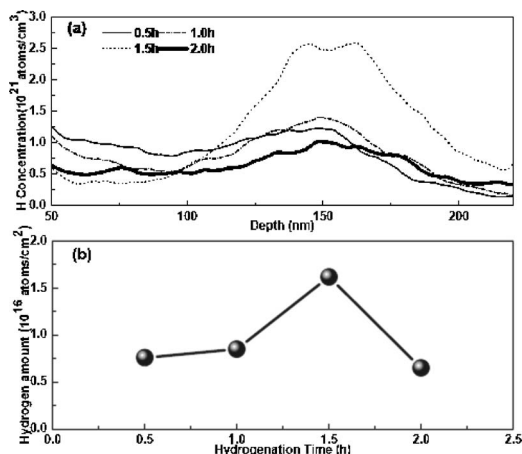

FIG. 2. (a) ERD spectra of hydrogenated Si/SiGe/Si with different durations. (b) The total amount of trapping hydrogen as a function of hydrogenation time.
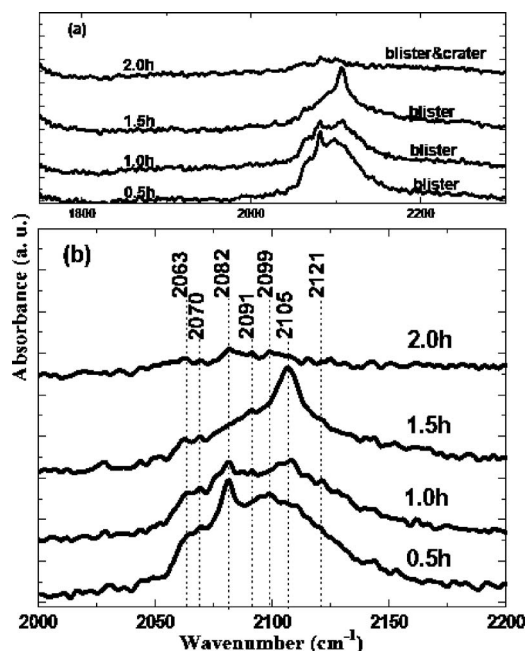
Different from the broadband absorption (1800–2300 cm^{-1}) observed in H-implanted samples,[1] plasma hydrogenated Si/SiGe/Si only shows a narrow absorption region from 2050 to 2150 cm^{-1} in Fig. 3(a), which is indicative of a lack of V_nH_m and I_nH_m defects. The intensity of the absorption peaks from the 2 h hydrogenation sample is significantly reduced due to the loss of bonded hydrogen following blister rupture. All Si–H bond stretching modes are marked in Fig. 3(b). Since (111) platelets form in the near-surface region of hydrogenated Si,[4] the modes at 2063, 2070, and 2082 cm^{-1} are assigned to the unperturbed and perturbed Si–H bonds at Si (111) surface.[10,15–17] The modes at 2091 and 2099 cm^{-1} are assigned to the asymmetric and symmetric stretching modes of a coupled monohydride species $(SiH)_2$ on atomically smooth Si(100) surfaces,[18] and the mode at 2121 cm^{-1} is close to the assignment for the asymmetric dihydride stretching motion on atomically rough Si(100) surfaces.[1,19] It should be noted that the stretching mode for $\nu_s(SiH)_2$ at 2099 cm^{-1} shifts to 2105 cm^{-1} for hydrogenation durations of 1 and 1.5 h; upon further hydrogenation for 2 h, it returns back to 2099 cm^{-1}.

It is well established that the interaction of bonded H on opposite inner Si (111) surfaces would lower the Si–H frequency, i.e., produce a redshift.[20] Therefore, the blueshift from 2099 to 2105 cm^{-1} observed during hydrogenation should not arise from such a dynamic coupling. Weldon *et al.*[20] attribute the 2099→2105 cm^{-1} blueshift to the steric interaction between Si–H bonds and overlying H_2 gas. This hypothesis is confirmed by our results which show that the shift from 2099 to 2105 cm^{-1} is strongly correlated with the H content in our samples. For the 0.5 h hydrogenated sample, only a small amount of hydrogen has accumulated at the buried SiGe layer. As hydrogenation increases to 1 and 1.5 h, the hydrogen content, and the probability of H_2 molecule formation within platelets, also increases. Gaseous H_2 inside large platelets or microbubbles will have steric interactions with Si–H bonds on internal (100) surface[20] and lead to a blueshift from its relaxed state at 2099 to 2105 cm^{-1}, as

shown in Fig 3. At extended plasma-hydrogenation times, microbubbles explode, gaseous H_2 diffuses out though the "popped" blisters, and the mode associated with Si–H bonds on internal (100) surfaces returns back to its unperturbed frequency, i.e., 2099 cm^{-1}.

In conventional ion cut, the defects and the corresponding in-plane compressive stress introduced by ion implantation are responsible for hydrogen trapping and platelet nucleation during postimplantation annealing.[6] For the plasma-hydrogenation based layer exfoliation process, exfoliation proceeds from the strained SiGe layer, which supplies the needed in-plane compressive stress for (100) platelet nucleation and H trapping. The nucleation of platelets during Si hydrogenation was discussed by Johnson *et al.*,[21] where the nucleation of platelets in Si supersaturated with hydrogen was qualitatively described using a Volmer-type theory. The model proposed by Johnson *et al.* considered a circular nucleus of radius R forming on a {111} plane. The total free energy change upon nucleation of a H platelet of radius R and thickness t was expressed as

$$\Delta G(R) = -\pi R^2 t \Delta G_v + 2\pi R \gamma t + 2\pi R c \ln(R). \quad (1)$$

In Eq. (1), ΔG_v is the energy difference per unit volume between the supersaturated state and the platelet state. The term $2\pi R \gamma t$ is the perimeter energy of the platelet nuclei, with γ as surface energy. Since Si–H bond formation is exothermic relative to the Si–Si bond,[11] γ will be negative for platelet formation. The third term, $2\pi R c \ln(R)$, is the strain energy resulting from formation of nuclei in the Si lattice (with c as a constant), which is modeled as the self-energy of a dislocation loop of radius R. For H platelet nucleation in Si, the first two terms in Eq. (1) supply the driving force for nucleation, while the strain energy is a barrier term.

Equation (1) is sufficient to describe the nucleation process in the absence of applied stresses, such as at the surface of our hydrogenated sample. However, in the region of the SiGe strain layer, a state of biaxial compressive stress exists that adds an additional term to Eq. (1) of the form[22]

$$-\sigma_{nn} b_n \pi R^2, \quad (2)$$

where b_n is the Burgers vector of the platelet loop, σ_{nn} is the externally applied stress (i.e., in-plane residual stress in our case) acting on the platelet, and $\sigma_{nn} b_n$ is a tensor product. Equation (2) will be zero when the stress and Burgers vector are orthogonal, indicating that there will be no effect from Eq. (2) on platelets nucleating with their surface parallel to the Si/SiGe interface [i.e., (100) platelets]. However, for any other platelet orientations, Eq. (2) will be positive. Therefore, there is an energetic barrier to any platelet not nucleating in the plane of stress. This explains why (111) platelets are observed in the stress-free surface region of our hydrogenated sample but not near the SiGe strain layer.[17] This same reasoning also explains why absorption modes from (100) platelets are observed in our samples with a SiGe strain layer but not in low temperature hydrogenated bulk Si.[23]

As supersaturated H is consumed in the nucleation and growth of platelets, the local concentration of free H diminishes and creates a local H sink. The further trapping of H allows the platelet to grow as well as accumulate H_2. As shown by Freund,[24] the final size of a platelet is proportional to the square root of the amount of gaseous H_2 molecules in the platelet. The accumulation of H_2 pressurizes the platelets, which drives them to grow into microbubbles and ultimately to exploded. Consequently, the highly pressurized H_2 in the microbubbles perturbs the Si–H bonds attached to the platelet surfaces, consistent with the observed blueshift from 2099 to 2105 cm^{-1} in the MIR-IR spectra.

In summary, the buried SiGe layer acts as a H trapping center during hydrogenation and H platelets are formed at the depth of SiGe layer. The platelet orientation is strongly dependent on the in-plane compressive stress introduced by the SiGe layer, which determines the cleavage location (at the depth of the stain layer) and cleavage orientation (in the plane of stress and parallel to the substrate surface).

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