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Nucleation of hydrogen-induced platelets in silicon

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Hydrogen-induced platelet generation in single-crystal silicon was investigated as a function of the Fermi energy. Platelet formation is observed only for Fermi-level positions of $E_C - E_F \leqslant 0.3\,\mathrm{eV}$. With decreasing $E_C - E_F$ the platelet density increases monotonically to $2.45 \times 10^{17}\,\mathrm{cm}^{-3}$. Experiments performed on electrically compensated silicon demonstrate that the formation of hydrogen-induced platelets is solely controlled by the Fermi energy.

For some years the properties of hydrogen in semiconductors have attracted a great deal of interest that is primarily stimulated by a variety of beneficial and nonbeneficial effects such as the ability to passivate both shallow-level acceptors and donors and deep-level defects in silicon.[1] Hydrogen diffused into single-crystal or polycrystalline silicon at moderate temperatures generates extended and electrically active defects commonly known as platelets. These two-dimensional defects are unrelated to plasma and radiation damage.[1,2] Platelet formation is not limited to silicon. These two-dimensional defects have also been observed in GaAs.[3] In this paper, we present a detailed picture of the influence of the Fermi energy on the nucleation process of platelets.

Previously, Johnson *et al.*[4] showed that the exposure of single-crystal silicon (c-Si) to a hydrogen plasma at moderate temperatures causes the formation of extended structural defects in the crystalline lattice. Platelets appear within ≈0.1 μm of the exposed surface and are predominantly oriented along {111} crystallographic planes. Platelet nucleation is suppressed at temperatures above 250 °C. However, platelets nucleated at lower temperatures grow markedly at higher temperatures.[5] Raman measurements showed that the added H atoms are bound to Si atoms suggesting that platelets are a consequence of the coordination formation of Si-H bonds driven by strain. In addition to structural changes of the lattice, platelet formation is accompanied by the generation of electrically active gap states.[4]

In this paper, we demonstrate the influence of the Fermi energy on the platelet nucleation process. Platelets are only observed in n-type silicon with a Fermi energy of $E_C - E_F \leqslant 0.3\,\mathrm{eV}$. The platelet concentration increases monotonically as E_F moves closer to the conduction band. Our experimental results clearly establish that the nucleation of H-stabilized platelets is unrelated to the presence of dopants and hence, depends solely on the Fermi-level position.

The experiments described in this paper were performed on n-type poly-Si and on a series of undoped, P-doped, B-doped, and compensated c-Si samples. Compensation of P-doped c-Si to a depth of about 1.0 μm was achieved by multiple boron implantations. The dopants were activated in a furnace anneal at 1200 °C. Transmission-electron-microscopy (TEM) micrographs showed that the implantation damage was completely removed by the furnace anneal. Hydrogenations were performed by exposing a set of samples simultaneously to monatomic deuterium or hydrogen generated in an optically isolated remote-plasma system that eliminates surface damage resulting from direct immersion in the plasma. This hydrogenation system has the advantage that platelet generation is driven only by chemical reactions of H with the silicon lattice. In order to maximize the platelet concentration a two-step hydrogenation was performed. A first hydrogenation step at 150 °C for 20 min was performed to nucleate platelets followed by a second exposure to monatomic H at 275 °C for 1 h. The second hydrogenation causes the existing platelets to grow to a length of up to 150 nm.[5] Platelet concentrations were obtained from TEM micrographs.

Cross-sectional TEM micrographs of P-doped c-Si and poly-Si are shown in Figs. 1(a) and 1(b), respectively. In this particular case, the second hydrogenation step of the c-Si sample was carried out for 30 min, while the poly-Si sample was exposed to monatomic H for 1 h. The TEM micrographs, viewed in the ⟨110⟩ projection, clearly reveal the presence of H-stabilized platelets in the near-surface region. In both samples, poly-Si and c-Si, platelets are oriented along {111} crystallographic planes. TEM micrographs show that platelets are confined to a subsurface region with a depth of approximately 0.1–0.15 μm, which is consistent with previous observations.[4] It is important to note that platelets do *not* penetrate the surface of the samples [Figs. 1(a) and 1(b)].

In contrast to c-Si, poly-Si contains a high degree of lattice strain and disorder in the grain-boundary regions. In these regions the formation of platelets should be favorable and one would expect to see enhanced platelet nucleation and platelets crossing the grain boundaries. This, however, is not the case. High-resolution TEM micrographs taken along grain boundaries showed neither evidence for preferred nucleation of platelets nor the penetration of grain boundaries [Fig. 1(c)]. In fact, no platelets were found within a distance of ≈40 nm from grain boundaries.

Platelets have only been observed in n-type c-Si (Ref. 4) and poly-Si samples.[2] In order to illuminate this phenomenon we investigated the influence of the Fermi energy on the platelet concentration in more detail. For this purpose a se-

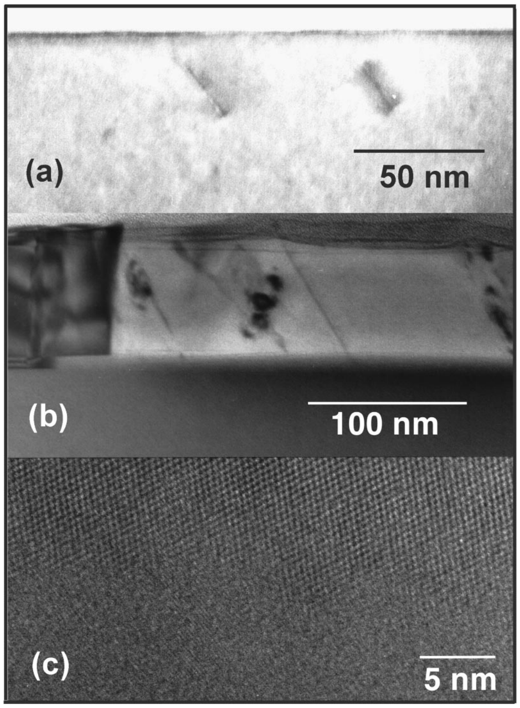

FIG. 1. Cross-sectional TEM micrographs, viewed in a ⟨110⟩ projection; (a) single crystal silicon and (b) poly-Si doped with a phosphorous concentration of $1\times10^{20}\,\mathrm{cm}^{-3}$. (c) High-resolution image of a grain boundary rotated by 90°. The periodicity of the silicon lattice does not appear in the lower half of the micrograph due to a different orientation of the grain.

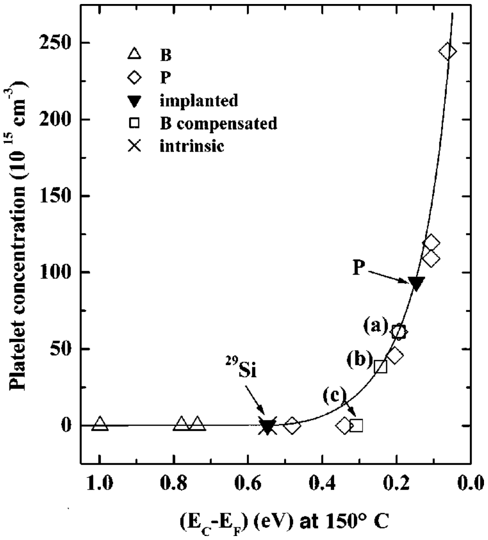

FIG. 2. Platelet concentration in c-Si as a function of the Fermi energy E_C-E_F at 150 °C. The open triangles and diamonds correspond to B- and P-doped c-Si, respectively. Data represented by the solid triangles were obtained on intrinsic c-Si implanted with ^{29}Si and P, respectively. The open squares represent data taken on P-doped c-Si where (a) is the control sample, and (b) and (c) are compensated with a B concentration of 2×10^{17} and $2.6\times10^{17}\,\mathrm{cm}^{-3}$, respectively.

ries of B- and P-doped c-Si samples was exposed to monatomic H according to the two-step hydrogenation schedule and subsequently the platelet concentrations were determined from TEM micrographs. The data are summarized in Fig. 2. Since platelet nucleation occurred at 150 °C the platelet concentration is plotted vs the Fermi energy at 150 °C. The two-step hydrogenation schedule did not produce platelets in B-doped c-Si (open triangles). On the other hand, in P-doped c-Si platelets were detected only in specimens with a Fermi energy of $E_C-E_F\leqslant0.3\,\mathrm{eV}$. With increasing P content the platelet concentration increases monotonically. The highest platelet concentration of $2.45\times10^{17}\,\mathrm{cm}^{-3}$ was obtained at a Fermi energy of $E_C-E_F=0.06\,\mathrm{eV}$.

It is important to note that the H concentration in the silicon bulk (depth greater than 0.3 μm) depends on the doping concentration. As E_C-E_F increases from 0.06 to 0.3 eV the H concentration decreases from 1.3×10^{19} to $\approx10^{17}\,\mathrm{cm}^{-3}$. Simultaneously, the platelet density decreases to zero. For deeper Fermi energies (up to 0.74 eV) the H concentration remains constant at $\approx10^{17}\,\mathrm{cm}^{-3}$. This behavior suggests that platelet generation requires a minimum H concentration of $\approx10^{17}\,\mathrm{cm}^{-3}$.

These observations provide an important hint regarding the microscopic mechanism for platelet generation. On the one hand, platelet nucleation could occur at P sites while, on the other hand, the driving force could be the Fermi energy. In the first case a lattice defect would be required for the nucleation process while in the latter case platelet creation would *not* be limited to special sites but could occur homogeneously throughout the specimen.

In order to elucidate the nucleation mechanism intrinsic c-Si with a Fermi energy of $E_C-E_F=0.55\,\mathrm{eV}$ was implanted with a P concentration of $1\times10^{18}\,\mathrm{cm}^{-3}$. According to TEM micrographs the subsequent activation anneal at 1200 °C completely removed the implantation damage. The P implantation caused a shift of the Fermi energy to $E_C-E_F=0.15\,\mathrm{eV}$ and post-hydrogenation produced a platelet density of approximately $9.4\times10^{16}\,\mathrm{cm}^{-3}$. To rule out the possibility that some residual implantation damage could act as a source for platelet nucleation a second intrinsic c-Si sample was implanted with a ^{29}Si concentration of $1\times10^{18}\,\mathrm{cm}^{-3}$. Again, the implantation damage was removed in a 1200 °C furnace anneal. The subsequent hydrogenation experiment did *not* result in the formation of H stabilized platelets demonstrating that residual implantation damage does *not* give rise to the nucleation of platelets.

Based upon these results a key experiment was performed that unambiguously demonstrates that platelet nucleation and growth is controlled by the Fermi energy. P-doped c-Si samples were electrically compensated by implanting boron to a depth of about 1.0 μm. Then, the samples were exposed to monatomic H. In Fig. 2 the uncompensated sample is labeled (a). The specimens labeled (b) and (c) were compensated with a B concentration of 2×10^{17} and $2.6\times10^{17}\,\mathrm{cm}^{-3}$, respectively (open squares in Fig. 2). While the partially compensated specimen (b) contained a platelet concentration of approximately $3.8\times10^{16}\,\mathrm{cm}^{-3}$ we did not observe platelets in the completely compensated sample [(c) in Fig. 2]. This observation clearly demonstrates that the H-induced generation of platelets is controlled by the Fermi energy.

Based upon the observation that the generation of platelets is accompanied by an increase of the concentration of Si-H bonds[4] clustering of interstitial molecular H_2 can be

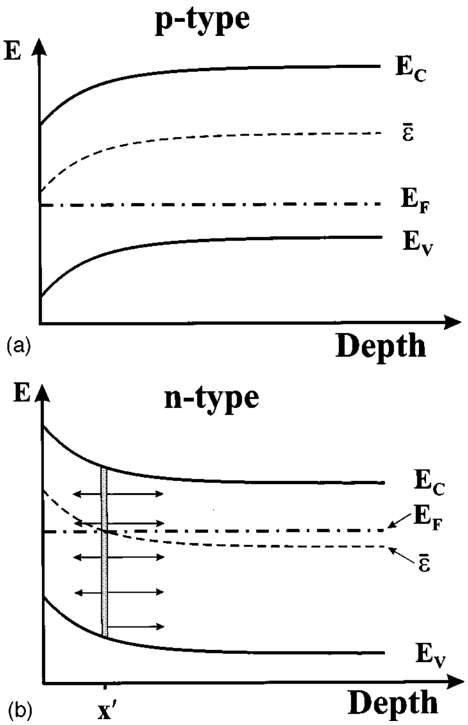

FIG. 3. Schematic illustration of the band diagram in the surface region of (a) *p*-type and (b) *n*-type *c*-Si. E_C, E_F, and E_V denote the conduction band, the Fermi energy, and the valence band, respectively. $\bar{\varepsilon}$ is the mean value of the hydrogen donor and acceptor levels.

ruled out as a microscopic model for platelet formation. Also the high mobility of H_2 at 100 °C (Ref. 6) is inconsistent with a platelet dissociation temperature of 400 °C.[7] On the other hand, it has been suggested that the microscopic structure of platelets consists of aggregated $H_2{}^*$ complexes.[8,9] The $H_2{}^*$ complex consists of one H in a Si-Si bond center site forming a Si-H bond and the other H residing in the antibonding interstitial site forming a Si-H bond with the remaining dangling bond.[10] The $H_2{}^*$ complex distorts the Si sp^3 bonds of one of the silicon atoms costing an energy on the order of 1 eV. By stacking up the $H_2{}^*$ complexes along a {111} lattice plane the strain can be reduced by as much as 0.4 eV per hydrogen pair.[8]

The exact microscopic structure of platelets is still unknown. However, the data presented above show that platelet nucleation and growth are controlled by the Fermi energy. Hence, platelet formation occurs only in the presence of positively and negatively charged H atoms. The concentrations of H^+ and H^- depend on the Fermi energy. The dominant H species changes quickly from H^+ to H^- as the Fermi level passes above the mean value of the H donor and acceptor levels $\bar{\varepsilon} = E_C - E = 0.38\,\text{eV}$. Since H in silicon is a negative-*U* impurity H^0 is unstable.[11–13]

At the sample surface the Fermi energy is pinned approximately at midgap due to a high concentration of surface states. Although we cannot determine the position of the Fermi level during the plasma hydrogenation it is conceivable that the influence of the remote plasma will not cause a significant deviation of E_F from its midgap position. Therefore, in *p*-type silicon the Fermi level always resides below $\bar{\varepsilon}$ [Fig. 3(a)] and monatomic H introduced from the remote plasma becomes positively charged. Thus, in *p*-type material where the Fermi level is close to the valence band platelets cannot nucleate.

On the other hand, in the bulk of *n*-type silicon the Fermi energy resides between the valence band and $\bar{\varepsilon}$. However, in the near-surface region at a depth x' the Fermi energy crosses $\bar{\varepsilon}$ due to surface band bending [Fig. 3(b)]. At this position the concentrations of H^+ and H^- are approximately the same and hence, platelet nucleation can occur. Although the generation of platelets is accompanied by the formation of acceptorlike defects their concentration is too small to compensate the *n*-type dopants.[4] On the other hand, the presence of monatomic hydrogen in *c*-Si influences the Fermi energy. Hydrogen entering the specimen becomes positively charged donating electrons. This results in a decrease of $E_C - E_F$ and the depletion layer width causing x' to move closer to the surface. Thus, platelet nuclei will extend in size and grow towards the surface. This is consistent with the observation that the distance between the surface and the beginning of platelets is independent of the Fermi energy. Hydrogen atoms diffusing deeper into the sample (depth greater than x') change their charge state and become negatively charged ($H^+ + 2e^- \rightarrow H^-$) compensating dopants. According to secondary ion mass spectroscopy measurements the H concentration in the silicon bulk exceeds the doping concentration by a factor of 3 and 1.6 at a doping concentration of 2.7×10^{17} and $2\times10^{18}\,\text{cm}^{-3}$, respectively. At higher doping concentrations the H concentration is somewhat lower than the doping concentration. Therefore, the concentration of H^- is sufficient to compensate the *n*-type dopants causing the Fermi level to move closer to $\bar{\varepsilon}$. As a consequence, the depth of equal concentrations of H^+ and H^- (x' in Fig. 3) moves deeper into the bulk providing the essential condition for the growth of already nucleated platelets. Two conditions can terminate the nucleation and growth process: (i) The H concentration decreases below a threshold concentration of $\approx 10^{17}\,\text{cm}^{-3}$, or (ii) the total H^- concentration is small compared to the doping concentration. Thus, the P dopants are only partially compensated and the Fermi energy remains between the conduction band and $\bar{\varepsilon}$. In this case the distance that x' shifts into the bulk is determined by the difference between the Fermi energy and $\bar{\varepsilon}$. However, x' cannot move beyond the depletion-layer width.

This model explains several generally observed phenomena related to platelets. Figure 1 shows that platelets neither penetrate the sample surface nor the grain boundaries. This is due to band bending caused by high concentrations of surface or grain-boundary defects. Hydrogen in the immediate vicinity of grain boundaries (≈ 40 nm) and the sample surface is therefore positively charged. Hence, platelet nucleation is suppressed due to a lack of H^-.

The dependence of the platelet density on the hydrogenation temperature was investigated by Johnson *et al.*[4] Platelet formation occurs preferentially at low temperatures (e.g., <250 °C). With increasing hydrogenation temperature the platelet concentration decreases as a consequence of the suppression of platelet nucleation. This is due to the temperature dependence of the Fermi energy. With increasing temperature the Fermi energy shifts towards midgap and the semiconductor becomes more intrinsic. Hence, H diffuses in the positive charge state and platelet nucleation is suppressed due to a lack of H^-. An influence of the hydrogenation temperature on the Fermi energy is therefore similar to the compensation experiments described above.

With increasing P concentration the platelet density and the thickness of the subsurface region containing platelets are increasing (Figs. 2 and 4). Under these conditions the total H concentration in the bulk (depth greater than 0.3 μm) is smaller than the doping concentration. Since H advances from the sample surface by diffusion and the diffusion coefficient decreases with increasing P concentration the time to achieve compensation by forming H^- increases resulting in a slower shift of x'. As a consequence the platelet nucleation rate increases.

In summary, we have demonstrated that nucleation and growth of H-stabilized platelets is controlled by the Fermi energy. Platelets are only observed in *n*-type silicon with a Fermi energy of $E_C - E_F \leqslant 0.3$ eV. In addition platelet generation requires a minimum H concentration of $\approx 10^{17}\,\mathrm{cm}^{-3}$. The platelet density and the thickness of the subsurface layer containing platelets are increasing with decreasing $E_C - E_F$. Platelet nucleation experiments performed on compensated c-Si clearly establish that the nucleation mechanism is controlled by the Fermi energy. Although the microscopic structure of platelets is still unknown their nucleation requires positively (H^+) and negatively charged hydrogen (H^-) atoms. Since the dominant H species depends on the Fermi energy platelets can nucleate and grow only when the Fermi energy is equal to the mean value of the H donor and acceptor levels, $\overline{\varepsilon}$.

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