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Defect Density Reduction in 4H-SiC (0001) Epilayer via Growth-Interruption during Buffer Layer Growth

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Abstract. In this paper, we have investigated the influence of growth-interruption during buffer layer growth on killer defect density in SiC epilayer grown over 4H-SiC (0001) substrates. We have observed that the growth-interruption method reduces total killer defect density by ~45(±5)%. Implementing growth-interruption in the buffer layer is a novel approach to mitigate epitaxial defects such as in-grown stacking faults (SFs), triangular defects, and basal plane dislocations (BPDs) in the drift layer and provide an extra margin to bipolar degradation by terminating BPDs early in the heavily doped buffer layer. The defect reduction mechanism in the presence of hydrogen has been simulated using Kinetic Monte Carlo (KMC) simulations.

Introduction

SiC demonstrates excellent physical properties for power devices; however, defects in the epitaxial layer have a detrimental impact on their performance. Although a 4° off-axis 4H-SiC (0001) epitaxial layers have shown improved quality, but they still fail to meet the requirements for high-voltage devices due to low yield [1-5]. In the SiC epitaxial layer, various morphological defects exist, including triangles, carrots, pits, step bunching, stacking faults (SFs), BPDs, and downfalls. These defects can be classified into two types: process-induced defects formed during the epitaxial process and extended defects propagating from the substrate. Previous studies have used growth interruption in the drift layer [6-8]. To reduce total killer defect density in the epitaxial layer, it’s essential to optimize the epitaxy growth process to prevent the propagation of BPDs and SFs into the drift layer. This study focuses on utilizing growth-interruption during the buffer layer to minimize the defects propagating from the substrate, as well as during the epitaxy growth process. The mechanism of defect reduction by hydrogen inclusion at the surface of the buffer layer during growth interruption and its subsequent interaction in growth has been investigated by Kinetic Monte Carlo (KMC) simulations.

Experiment

We used 150 mm Si-face 4H-SiC (0001) n-type substrates (Cree, Inc.) with 4° off-oriented toward $(11\bar{2}0)$ for epitaxy growth. Processing was started with RCA cleaning to remove surface particles. The epitaxial growth was carried out using a single-wafer CVD reactor, PE1O8 (ASM International N.V.), with growth rate >50µm/hrs and doping concentration ~$1 \times 10^{16}\ cm^{-3}$. The source gases were trichlorosilane (HCl_3Si) and ethylene (C_2H_4) with hydrogen (H_2) as carrier gas. We kept the wafer in the reactor for the *in-situ* growth-interruption. We used KLA Candela 8520 for substrate and epitaxy defect inspection, FT-IR for the epitaxy thickness, Hg-CV for carrier concentration, and AFM for surface roughness measurement after epitaxy.

Results and Discussion

In this study, we have used four substrates for epitaxy growth *viz.* G1:W1, G2:W2 (without interruption), and IG1:W3, IG2:W4 (with interruption growth). Epitaxy was done in consecutive runs to minimize process variation related to the reactor. The temperature, pressure, and C/Si ratio were kept constant throughout these runs. Fig. 1(a) shows the temperature profile for the growth-interruption process. The interruption temperature is slightly below the epitaxy growth temperature to minimize hydrogen etching. The epilayer thickness was measured at 32 points, with a 5 mm edge exclusion [Fig. 1(b)]. The results demonstrate that the uniformity across the wafer is better than 1.8%.

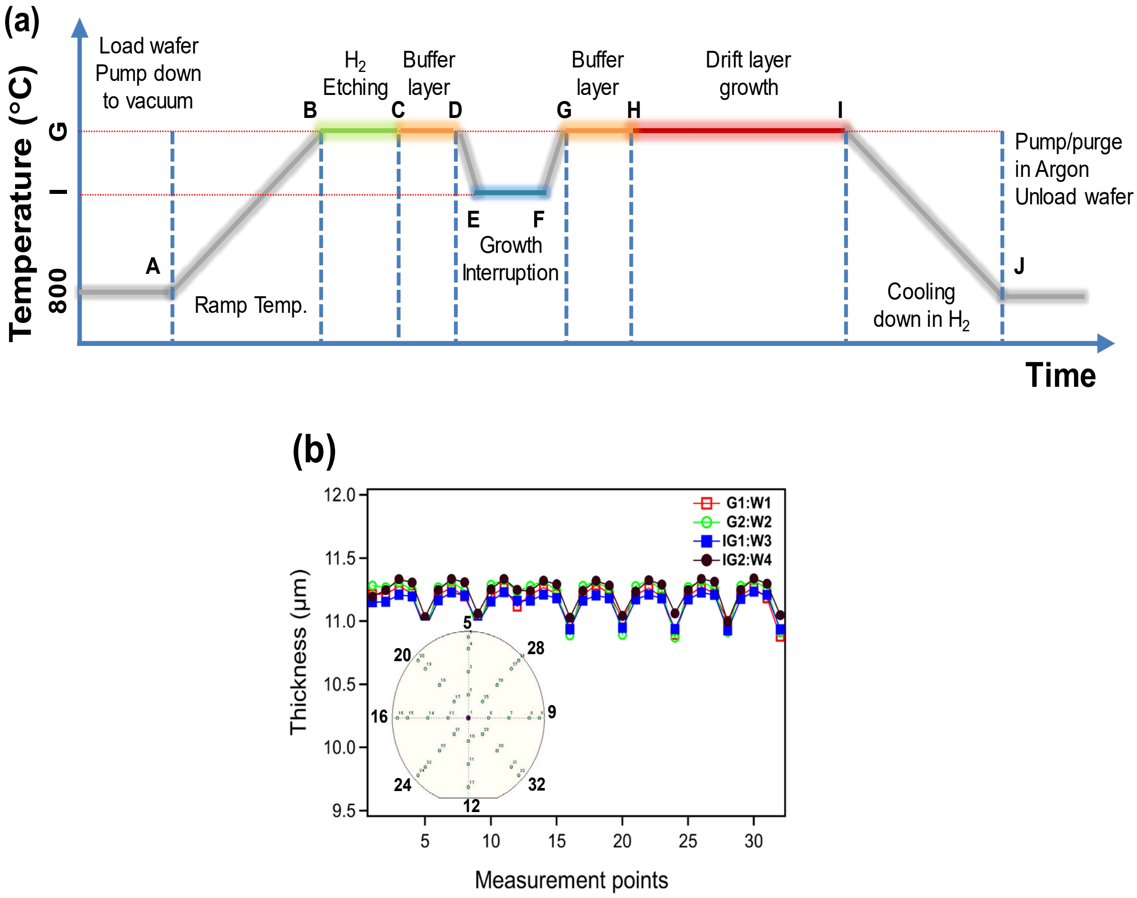

Fig. 1. (a) Process sequence of the 4H-SiC epitaxy for growth interruption experiments. (b) Thickness measurement of the epilayer across (inset shows the measured points map) the wafer with 5 mm edge exclusion. The thickness uniformity is better than 1.8% across the wafer.

Fig. 2(a) and (b) show the defect scan maps without and with the interruption-growth, respectively. We observed a ~45 (±5)% reduction in total killer defect density compared to the growth without interruption [Fig. 2(c)]. This is attributed to the conversion of BPDs to TED due to growth-interruption. To better understand how interrupting the growth of the buffer layer can reduce defect density, we used an additional SiC substrate from the same supplier. We assessed the roughness immediately after H_2-etching at the growth temperature (G °C) and then at the interruption temperature (I °C) [Fig. 2(e)]. AFM scans reveal that the substrate exhibits a smoother surface ($R_q \leq 0.2\ nm$) measured at the interruption temperature "I" °C compared to H_2-etching at drift layer growth temperature "G" °C ($R_q \geq 0.8\ nm$) on a $10 \times 10\ \mu m^2$ area.

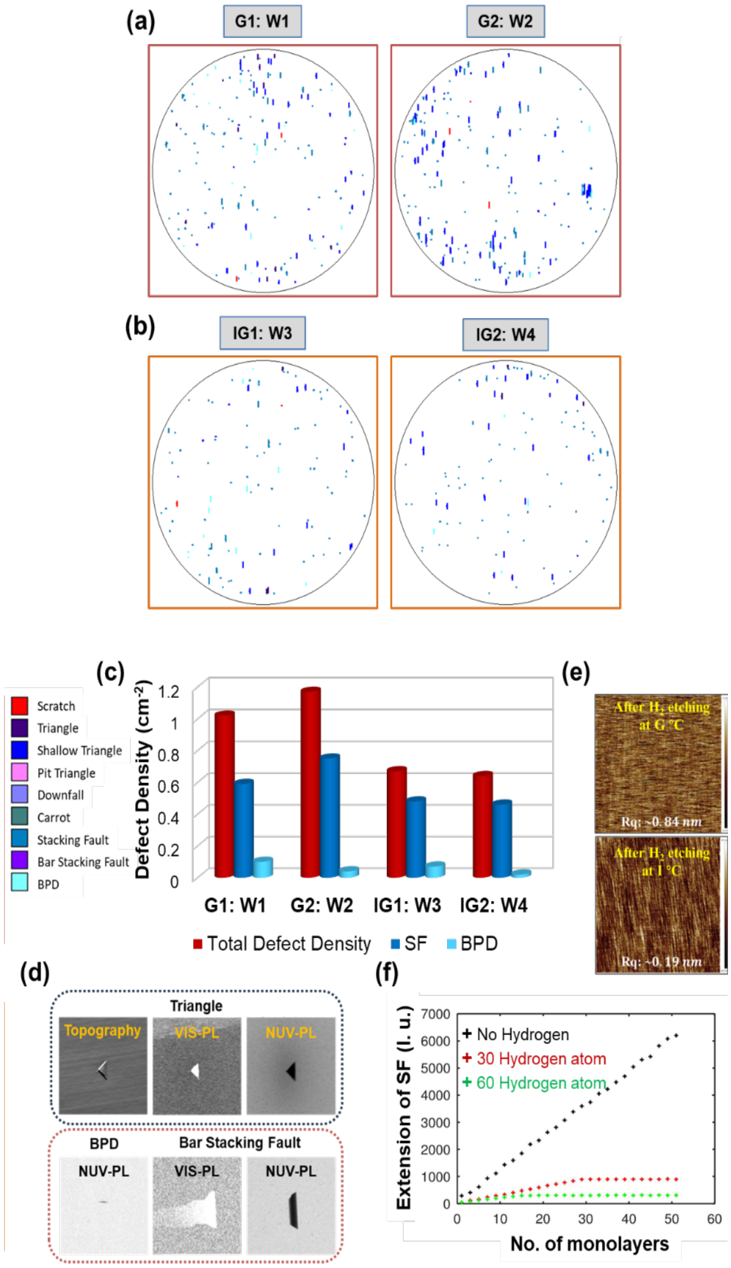

Fig. 2. Candela 8520 defect scan maps (a) without and (b) with interruption growth. (c) Defect density without and with the interruption growth. (d) High-resolution images ($1.5 \times 1.5\ mm^2$) of typical SiC defects in different channels in Candela. (e) AFM images at the growth (G) and interruption (I) temperatures at the center of the substrate. (f) KMC simulations on the extension of SFs in the epilayer as a function of the thickness of the epitaxial layer.

This reduction in surface roughness leads to a decrease in surface free energy, which minimizes the disruption of intermolecular bonds and results in a significant reduction in defects during the subsequent growth of the second buffer layer. The mechanism of defect reduction by hydrogen (H) inclusion at the surface of the buffer layer during growth interruption and its subsequent interaction in growth has been investigated theoretically. We adopted the Kinetic Monte Carlo (KMC) simulations with a superlattice model [9] in which the neighboring atoms of the regular SiC lattice are considered part of the defective lattice, and vice versa. During the growth interruption process, it is assumed that H-atoms from the subsurface region diffuse into the epitaxial layer and can occupy in the defect sites due to the lower surface energy. When an H-atom occupies a defective lattice site, it prevents the formation of stacking faults (SFs) in that site. As the nearest neighbor to the defective site is the regular lattice, these atoms become part of the regular SiC lattice, limiting the extension of SFs. Figure 2(f) depicts the reduction in SF extension as the epitaxial layer thickness increases, with and without H-atoms. The results clearly show that the presence of H-atoms occupying potential SF extension sites can prevent the formation of SFs in the epitaxial layer. Both experimental and simulation results demonstrate that interrupting the growth process at an early stage during buffer growth significantly reduces defect density in the epilayer. We achieved a calculated epi-yield of >96.5% based on $2 \times 2\ mm^2$ die size.

Conclusion

In summary, growth interruption during buffer layer epitaxial growth for defect reduction in 4H-SiC was successfully performed. This led to the significant reduction of total killer defect density by ~45(±5)%. Moreover, we achieved defect-free usable area on a 150 mm substrate with a yield of >96.5% based on $2 \times 2\ mm^2$ die size.

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