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Nuclear Instruments and Methods in Physics Research B 240 (2005) 142–145
NIM B
Beam Interactions with Materials & Atoms
www.elsevier.com/locate/nimb

The influence of the ion implantation temperature and the dose rate on smart-cut© in GaAs

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Available online 28 July 2005

Abstract

The temperature and dose rate dependence of the smart-cut© process in GaAs have been investigated in this paper. The distribution of hydrogen and the implantation damage in the samples were studied by ion beam analysis and X-ray diffraction. It was found that at higher temperatures, hydrogen is mobile in the lattice and can rearrange into the platelets, microcracks and bubbles which are present in blistered material, thus relieving the strain in the lattice. The dose rate was also found to be significant for the smart-cut process, as blistering and exfoliation are inhibited at low dose rates.

PACS: 61.72.Vv; 61.72.Cc; 61.18.Bn

Keywords: Smart-cut; Exfoliation; Blistering; Hydrogen implantation

1. Introduction

Smart-cut© is a layer transfer technique, which offers a route to the monolithic integration of dissimilar materials. The technique, invented by Bruel in 1995 [1], exploits hydrogen implantation-induced exfoliation and wafer bonding to transfer thin layers of a semiconductor onto another material, which may have a different lattice constant. One advantage of the smart-cut method is that it facilitates the combination of GaAs and Si, which has been a goal for many years. Indeed, the layer transfer of GaAs onto Si was demonstrated by the smart-cut method in 1998 [2]. However, it is still unclear exactly how Smart-Cut is affected by different ion implantation parameters, especially

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doi:10.1016/j.nimb.2005.06.104

for III–V materials such as GaAs. In particular, there is no consensus in the literature as to the implantation temperatures required to blister GaAs – an initial study by Tong et al., concluded that the implant temperature should lie within 150–250 °C for smart-cut to be possible [3]. An explanation for this phenomenon was offered, but this was not verified by experimental results. However, subsequent research suggests that smart-cut can occur at temperatures outside of these windows [4,5]. Clearly, it is most probable that the discrepancy lies either in the measurement of the implant temperature or due to the significance of another implantation parameter, which had not previously been considered. This paper aims to investigate the role of the implant temperature and the dose rate on blistering in GaAs, in order to reconcile these findings, and to further the understanding of the smart-cut process.

2. Experimental

Semi-insulating GaAs wafers were implanted with 190 keV H_2^+ ions, to a fluence of 5×10^{16} H^+/cm^2, at sample temperatures of −90 °C, 30 °C, 100 °C, 200 °C and 300 °C respectively. Each implant was studied by elastic recoil detection (ERDA) and Rutherford backscattering in channelling mode (RBS) to obtain profiles of the implanted hydrogen and the damage. Changes in the strain profile of the implant were monitored using double axis X-ray diffraction.

A second batch of implants was performed at dose rates of 3.3×10^{12}, 9.8×10^{12}, 1.4×10^{13} and 2.5×10^{13} $H^+/cm^2/s$ 180 °C. These implants were performed at the same energy but at a higher fluence of 1×10^{17} H^+/cm^2. The wafer temperature was measured by a thermocouple attached to a silicon guard ring which held the sample in place. The temperature of the silicon guard ring was calibrated against the temperature of the sample. Samples from each of the wafers were inspected by atomic force microscopy (AFM) and Nomarski optical microscopy before and after annealing to investigate blister formation. Some samples were also studied by transmission electron microscopy.

3. Results and discussion

Fig. 1 shows the channelling RBS spectra obtained for the first batch of samples. The lattice disorder of the samples, proportional to the normalised counts on the y axis of these spectra, grows with increasing temperature between 30 °C and 200 °C. Transmission electron microscopy images of the 200 °C sample show that the hydrogen agglomerates into the platelets, microcracks and bubbles which are responsible for blistering and exfoliation, in agreement with previous studies [6]. Therefore we assign the increase in the damage to the rearrangement of point defects into these defect complexes. The X-ray diffraction data obtained from these samples is shown in Fig. 2. As the implant temperature is raised, the strain is relieved, indicated by the attenuation of fringes to the left of the high intensity substrate peak. This is in agreement with the idea that at higher implant temperatures, hydrogen becomes more mobile in the lattice, and forms into complexes, such as platelets, which relieve the strain.

However, at 300 °C, the RBS spectra is lower than for the 200 °C sample, indicating that the lattice disorder is lower, and therefore that platelet formation is not as prolific, despite the relief of strain (see Fig. 1). The ERDA spectra for the first batch of wafers are shown in Fig. 3. Analysis of the spectra showed that the amount of hydrogen retained in the sample directly after implantation gradually decreased with implant temperature – for the 300 °C implant, the total hydrogen content was 32% lower than for the −90 °C implant. The peak of the profile for the 300 °C implant is also shifted 9 channels to the right (i.e. towards the surface), indicating that the hydrogen is migrating towards the surface, and that the loss of hydrogen is due to out-diffusion. This suggests that platelet formation, and therefore the lattice disorder of the 300 °C implant is inhibited by the amount of hydrogen present. We suggest that in this situation there is simply not enough hydrogen present to evolve the defect structure into the platelets, microcavities and bubbles observed in blistered and exfoliated samples.

Inspection of the second batch of samples by Nomarski microscopy showed that blistering was