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Retarding Mechanism of Si Selective Epitaxial Growth on CMOS Structure due to Doped Arsenic in the Si Substrate

Kiyotaka MIYANO, Ichiro MIZUSHIMA, and Yoshitaka TSUNASHIMA

Process and Manufacturing Engineering Center, Semiconductor Company, Toshiba Corporation

8 Shinsugita-cho, Isogo-ku, Yokohama-shi, Kanagawa 235-8522, Japan

Phone: +81-45-770-3661, Fax : +81-45-770-3577, E-mail: kiyotaka.miyano@toshiba.co.jp

1. Introduction

An elevated source/drain (S/D) engineering greatly contributes to the shallow junction formation [1]. Si selective epitaxial growth (SEG) [2] is a potential solution to form elevated S/D structure.

Precise thickness control of the epitaxial layer is one of the most important factors for the purpose of obtaining uniform shallow junction. However, the thickness of the epitaxial layer fluctuates by the dopants and doping level in the substrate. Moreover, the dependence of the growth rate on the doping level has not been well understood despite many experiments have been performed to date [3].

The purpose of this study is to clarify its dependence. A new model is proposed for the retardation of SEG on the doped area in Si substrate, based on the experimental results that reveal what is going on during the incubation time at the initial stage of the deposition.

2. Effect of device structure on the epitaxial growth

The experimental procedure is shown in Fig. 1. Samples with CMOS structure were used, because the actual device that requires elevated S/D will be this structure. The thickness of the deposited film showed a clear dependence on the doping of the impurities as shown in Fig. 2. In case of the epitaxy on the doped layers, a long incubation time is needed before the growth starts.

It is also interesting that the incubation time for n- and p-type regions has the same value. This suggests that the doping into one region might have some influence on the other region. So, thickness of the epitaxial layer was measured on the wafer, into which a single impurity was partly implanted, as shown in Fig.3.

Thicknesses of the grown films on doped and undoped regions are shown in Table I. Effect of the doping is quite different from those shown in Fig.2. The results obtained from Table I are summarized as follows. 1) Doping of arsenic retards the growth not only on the doped region but also on the neighboring undoped region. 2) Doping of boron has no effect on the retardation of the growth.

These results suggest that the retardation on the both n- and ptype regions in CMOS is due to the arsenic, and boron has no influence.

SIMS profiles of n- and p-type regions of CMOS, as shown in Fig. 4 (a) and (b), demonstrate the above mentioned. Only arsenic was detected on the arsenic implanted region, while arsenic and boron were detected on the boron-implanted region. These results indicate that arsenic out-diffuses from the n-type region and re-deposits on the p-type region before the epitaxial growth starts.

3. Arsenic mediated suppression of epitaxial growth

The following ingenious experiment revealed that arsenic mediated the suppression of epitaxial growth during the incubation time. In order to investigate the retardation mechanism of SEG due to the arsenic doping, the condition of the Si surface during the incubation time was examined. The experimental sequence is shown in Fig. 5. The sample, on which only arsenic was doped as shown in Fig.3, was exposed to the SiH₂Cl₂/HCl/H₂ ambient within the incubation time, and then the source gas and processing temperature were changed to deposit polycrystalline Si at low temperature. By means of the polycrystalline Si deposition, the Si surface condition could be examined by SIMS as an interface between Si substrate and deposited Si without changing the condition.

With this measurement technique, it was revealed that chlorine adsorbed during the incubation time. Typical example is shown in Fig.6. High concentration of chlorine was detected at the interface in addition to adsorbed arsenic.

The dependence of adsorbed chlorine concentration on the arsenic concentration suggests that the chlorine adsorption is accompanied by arsenic. Figure 7(a) and (b) show the arsenic and chlorine concentrations at the interface with high and low dose arsenic implantation, respectively. The effect of the exposure to the SiH₂Cl₂/HCl/H₂ ambient is clearly observed. In the case of highly doped sample, the chlorine concentration showed a distinct increase caused by the exposure, and this increase coincides with the increase of the incubation time.

4. Retardation model of epitaxial growth by chlorine

The mechanism for the increase of the incubation time was modeled as shown in Fig. 8. Arsenic atoms out-diffuse and redeposit during the H_2 annealing step. Then, at the initial stage during the exposure to the SiH₂Cl₂/HCl/H₂ ambient, arsenic attracts the chlorine atoms to adsorb on the undoped region as well as the n-type region. The adsorbed chlorine atoms suppress the adsorption of the SiH₂Cl₂ during the incubation time.

A quantitative estimation was performed for the SiH_2Cl_2 adsorption in terms of chlorine coverage. The SiH_2Cl_2 sticking coefficient is supposed to depend on the chlorine atom coverage on the surface [4], as is written by,

$$S(\theta_{Cl}) = S_0 \left(1 - \frac{\theta_{Cl}}{\theta_{S,Cl}} \right) \tag{1}$$

where $S(\theta_{Cl})$ is sticking coefficient of SiH₂Cl₂ as a function of surface chlorine coverage, S_0 is initial sticking coefficient, θ_{Cl} is chlorine coverage on Si, and $\theta_{S,Cl}$ is the saturation chlorine coverage on Si. The chlorine coverage θ_{Cl} was obtained from SIMS profiles to be 2.5×10^{13} cm⁻² for high-dose sample and 5.5×10^{11} cm⁻² for low-dose sample, respectively. And the saturation chlorine coverage for Si(100) was 5.7×10^{13} cm⁻². Substituting these values into eq.1, the value of the SiH₂Cl₂ sticking coefficient of high-dose sample is found to be about a half of that of low-dose sample (Table II). Thus, the retardation of SEG was reasonably explained to be brought about by adsorption of chlorine atoms.

5. Conclusions

The effect of the doping on the retardation of SEG on the CMOS structure was investigated, and the mechanism was revealed for the first time. Arsenic evaporates from the n-type region and adsorbs on the p-type region. Chlorine adsorption is induced by the arsenic atoms on the surface and suppresses the adsorption of SiH₂Cl₂.

References

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the thickness of the epitaxial layers on CMOS structure.



Fig. 3 Schematic cross-sectional illustration of the sample for the experiment using single impurity-implanted wafer.

	H ₂ cleaning	SEG	T _{As(B)} [nm]	T _{undope} [nm]
Ref. (undoped)	800°C 1min. 10Torr	800°C 350s 10Torr. H ₂ /DCS/HCL =15/0.4/0.11 slm	31.5	
As 50keV 5e15cm ⁻²			14.0	14.0
B 50keV 5e15cm ⁻²			33.0	33.0









Experimental sequence for the investigation of the surface state just before SEG.









Fig.7 (b)

Areal densities of arsenic and chlorine with and without Fig. 7 exposure to the SiH2Cl2/HCl/H2 ambient

- high-dose(5x1015 cm-2) arsenic-implanted sample a)
- low-dose(5x10¹⁴cm⁻²) arsenic-implanted sample b)



Fig. 8

Model of the SEG retardation. Chlorine adsorption is induced by arsenic.

Sticking of SiH,Cl, to the surface is suppressed by chlorine.

As I/I (cm ²)	$\theta_{S,Cl}(cm^2)$	$\theta_{Cl}(cm^2)$	$S(\theta_{Cl})(cm^2)$
5 x 10 ¹⁴	5.7x 10 ¹³	5.5 x 10 ¹¹	0.99 S ₀
5 x 10 ¹⁵	1	2.5 x 10 ¹³	0.56 So

SiH₂Cl₂ sticking coefficient $S(\theta_{cl})$ Table. II