# Chapter 1

# Introduction to Ion Implantation and Dopant Diffusion

## 1.1 Introduction

In this chapter, the motivation for studying the effects of ion implantation damage on dopant diffusion is given. Next, the two basic mechanisms for dopant diffusion are discussed. Finally, the scope and organization of this thesis are presented.

## 1.2 Motivation for Studying the Effects of Implantation Damage

Today, ion implantation is the most common technique used to introduce dopant atoms into silicon during integrated circuit fabrication. The main advantages of ion implantation are its mass selectivity of the desired species, the control of the total number of ions implanted into the sample, and the ability to distribute the ions at different depths by controlling the initial energy of the implanted ions.

Immediately after implantation, most of the implanted atoms will not lie on substitutional sites and thus will not be electrically active. In order to activate the dopants as well as repair crystal damage caused by the implant, a high temperature anneal is

typically done. One side effect of the high temperature anneal is that the dopant atoms also diffuse. Previously, when device dimensions were much larger than 1 micron, diffusion models that accounted for concentration-dependent dopant diffusion were sufficient to describe diffusion after implantation. Today, in order to fabricate faster devices and denser memory arrays, shorter anneal times and lower anneal temperatures are used to try to minimize dopant diffusion after implantation while still activating the implanted dopant. Unfortunately, for boron [1], phosphorus [2],[3], and arsenic [3], anomalous diffusion after implantation has been observed. For both boron and phosphorus, much larger diffusion than that predicted by the simple concentration-dependent diffusion is observed. Arsenic also exhibits enhanced diffusion but to a lesser degree compared to boron and phosphorus. Also, the enhanced diffusion can be larger at lower temperatures resulting in more profile broadening at lower anneal temperatures.

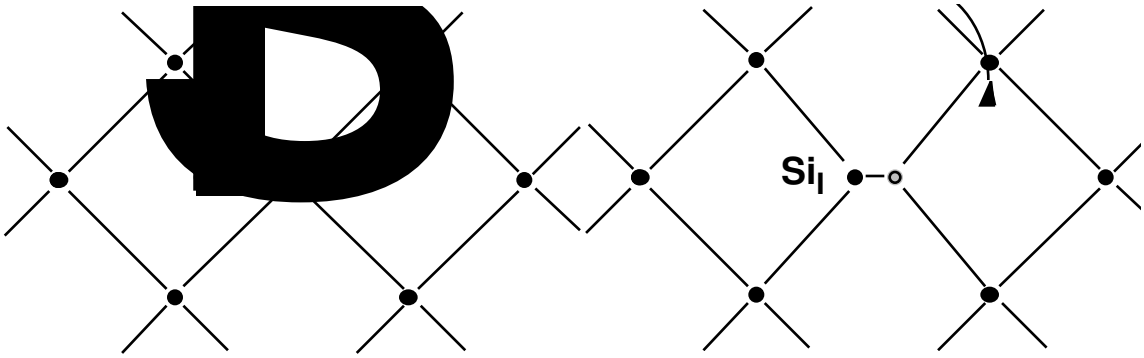
This anomalous enhanced diffusion has been attributed to the annealing of the damage caused by the implant process itself. In particular, the generation and recombination of point defects, interstitials and vacancies, directly influences dopant diffusion. Furthermore, since these point defects are very mobile in silicon, an implantation at the silicon surface can affect dopant diffusion several microns away. Thus, implantation damage results in broader junctions both vertically and horizontally. In the case of metal-oxide-semiconductor (MOS) devices, the source/drain implant or the lightly-doped drain (LDD) implant may cause dopant redistribution that degrades device performance. Deeper source/drain junctions result in worse short channel effects [4] in which the threshold voltage of the MOS device decreases at short channel lengths. Broader channel doping profiles may result in poor threshold voltage control and even reverse short channel effects [5],[6] in which the threshold voltage actually increases at short channel lengths before exhibiting the standard short channel effects. Thus, it is important to quantify the effects of implantation damage on dopant diffusion in order to be able to control device characteristics.

Furthermore, process models for dopant diffusion must be investigated to better understand the interaction between implantation damage and dopant diffusion. This consists of evaluating existing models and, if necessary, the implementation of new

models. Overall, though, for the models to be useful from an industrial perspective, they must be physically-based such that they can be used in a predictive manner. Although process simulators cannot completely eliminate the need to run wafer lots in the lab, they should at least be able to narrow the process window, thereby reducing the number of lots that must be run.

## 1.3 Basic Mechanisms for Dopant Diffusion

Dopant diffusion occurs via the interaction with point defects, interstitials and vacancies. The two mechanisms are depicted two-dimensionally in Figure 1.1 and Figure




(a) (b)

Figure 1.1 Two-dimensional schematic representations of (a) the kick-out interstitial diffusion mechanism and (b) the interstitialcy diffusion mechanism.

1.2, respectively. The interstitial mechanism can occur via either the kick-out mechanism whereby the dopant becomes purely interstitial (Figure 1.1(a)) or the interstitialcy mechanism whereby the dopant shares a lattice site with a silicon atom (Figure 1.1(b)). Since mathematically, there is no difference between the two mechanisms, throughout the rest of this thesis, interstitial will refer to either the kick-out or interstitialcy mechanism.

In this dual point defect diffusion framework, dopants diffuse only when paired with a point defect. The pairing of a dopant with a point defect can be represented by:

$$A + X \Leftrightarrow AX \tag{1.1}$$

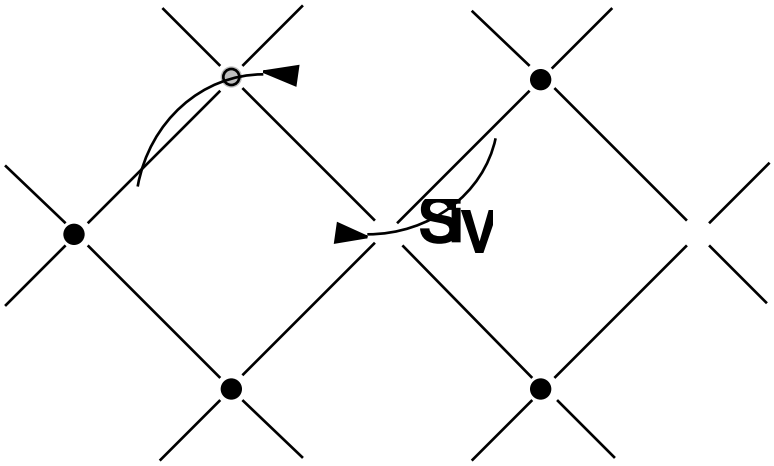



Figure 1.2 Two-dimensional schematic of the vacancy diffusion mechanism.

where A represents a dopant atom, X represents a point defect, either an interstitial or a vacancy, and AX represents either a dopant/interstitial pair or a dopant/vacancy pair.

The total dopant flux can be written as:

$$-J_A = D_{AI}\frac{\partial C_{AI}}{\partial x} + D_{AV}\frac{\partial C_{AV}}{\partial x} \tag{1.2}$$

where $D_{AI}$ and $D_{AV}$ are the diffusivities of AI and AV pairs, respectively, and $C_{AI}$ and $C_{AV}$ are the AI and AV pair concentrations, respectively.

If Eq. 1.1 is in equilibrium and the dilute concentration approximation is valid (i.e. $C_{AX} << C_A, C_X$), $C_{AX}$ can be calculated from:

$$C_{AX} = K_{AX}C_A C_X \tag{1.3}$$

where $K_{AX}$ is the equilibrium pair constant between the dopant, A, and the defect, X. Equation 1.2 can now be rewritten as:

$$-J_A = D_{AI}K_{AI}\frac{\partial}{\partial x}(C_A C_I) + D_{AV}K_{AV}\frac{\partial}{\partial x}(C_A C_V) \tag{1.4}$$

In intrinsically doped material under quasiequilibrium conditions, $C_I$ and $C_V$ will be approximately constant, and Eq. 1.4 further simplifies to:

$$-J_A = (D_{AI}K_{AI}C_I + D_{AV}K_{AV}C_V)\frac{\partial C_A}{\partial x} = D_A\frac{\partial C_A}{\partial x} \tag{1.5}$$

If one defines the equilibrium dopant diffusivity $D_A^*$ to be:

$$D_A^* = D_{AI}K_{AI}C_I^* + D_{AV}K_{AV}C_V^* \tag{1.6}$$

and $f_{AI}$, the fractional interstitial component of diffusivity to be:

$$f_{AI} = \frac{D_{AI}K_{AI}C_I^*}{D_A^*} \tag{1.7}$$

and $f_{AV}$, the fractional vacancy component of diffusivity to be:

$$f_{AV} = 1 - f_{AI} = \frac{D_{AV}K_{AV}C_V^*}{D_A^*} \tag{1.8}$$

then the following expression can be written for $D_A/D_A^*$:

$$\frac{D_A}{D_A^*} = f_{AI}\frac{C_I}{C_I^*} + (1 - f_{AI})\frac{C_V}{C_V^*} \tag{1.9}$$

This last equation expresses the instantaneous dopant diffusivity normalized to the equilibrium dopant diffusivity as a function of interstitial and vacancy concentrations normalized to their equilibrium concentrations. Obviously, if the interstitial and vacancy concentrations remain at equilibrium then normal inert diffusion will occur. However, if either $C_I$ or $C_V$ deviates from equilibrium, enhanced or retarded dopant diffusion with respect to inert diffusion can occur. For example, oxidation is known to inject silicon interstitials causing an interstitial supersaturation. This results in enhanced diffusion of dopants that diffuse predominantly via an interstitial mechanism. Conversely, direct nitridation is known to inject excess silicon vacancies causing enhanced diffusion of dopants that diffuse primarily with silicon vacancies. Ion implantation is yet another process whereby point defect concentrations are perturbed from their equilibrium values causing anomalous dopant diffusion.

## 1.4 Scope and Organization

The main objectives of this thesis are to experimentally characterize the effects of ion implantation damage on boron diffusion, to evaluate the ability of existing diffusion models to account for such effects, and to implement new physically-based models when current models are not sufficient. The emphasis of the experimental work in this thesis is on high-dose implants which are implants above the amorphization threshold although some experimental data spans the low-dose regime below the amorphization threshold. The diffusion models in SUPREM-IV [7], Stanford s two-dimensional process simulator, are assumed to be representative of existing equilibrium point defect based diffusion models. Equilibrium in this context means that the diffusing species can be calculated from Eq. 1.3. Besides using SUPREM-IV to test current diffusion models, it is also used as a vehicle with which to test new models.

The remainder of this thesis is organized in the following manner. In Chapter 2, experiments characterizing the effect of extended defects on boron diffusion are investigated. A one-dimensional model for extended defects is implemented into SUPREM-IV, and simulations using the new model are compared to the experimental data. In Chapter 3, a special test structure has been developed in order to isolate the effect of implantation damage on boron diffusion. Silicon is used to create the implant damage, and the effect of the damage is characterized as a function of silicon damage dose and energy, final anneal time, and final anneal temperature. In Chapter 4, the existing diffusion models in SUPREM-IV are evaluated and compared to the data presented in Chapter 3. The data presented in Chapter 3 indicates there are two sources for point defects, and the current models in SUPREM-IV cannot predict the correct time-dependence in enhanced diffusion. However, the extended defect model introduced in Chapter 2, with one modification, can be used to mimic the dissolution of interstitial-type clusters providing a second source for interstitials. Improved fits to the data are obtained with the addition of the cluster dissolution model. Finally, Chapter 5 summarizes the major contributions of this work and presents suggestions for possible directions of future work.