

CARRIER RECOMBINATION AND LIFETIME IN HIGHLY DOPED SILICON†

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Abstract—The predominance of phonon-assisted band-band Auger recombination in highly doped silicon is demonstrated by showing that no recombination mechanism involving *common* (unavoidable) defects in silicon can yield carrier lifetimes that are consistent with the measured lifetimes, which exhibit an inverse-quadratic doping-density dependence, and/or with their temperature dependence. Both trap-assisted-Auger and Shockley-Read-Hall recombination mechanisms are considered, and dependences of the defect density on the doping density, which are implied by theory and experiment, are accounted for.

1. INTRODUCTION

The upper bound of minority-carrier lifetime in highly doped silicon is of practical importance in the determination of the ultimate performances of bipolar devices, e.g. transistors and solar cells. It has been generally presumed that this bound is defined by the fundamental phonon-assisted band-band Auger (BBA) recombination mechanism[1]. Indeed, BBA theory[2] predicts the inverse-quadratic dependence of lifetime on majority-carrier density, or doping density[3], that is shown by the preponderance of the lifetime measurements[4, 5]. This dependence is also characteristic of phonon-free band-band Auger recombination, but because of inconsistent theoretical-experimental temperature dependences, this mechanism does not appear to be important in indirect-gap materials like silicon[1, 6].

There are however three reasons to suspect that these measurements[4, 5] for n^+ silicon do not reflect the fundamental BBA mechanism. First, recent experiments[7-11] have implied minority-hole lifetimes that are much longer than those reported in [4] and [5] and that do not conform to the inverse-quadratic dependence on doping density. Second, the narrowing of the (optical) energy gap[12-14] due to many-body effects in highly doped silicon should cause the BBA lifetime to decrease with increasing doping density more rapidly than the inverse-quadratic variation. Third, theoretical analysis of the BBA mechanism in n -type silicon is not in accord with the experimental studies[2].

Based on the experimental results referred to above, it has been theorized[7-9] that the commonly observed hole lifetimes[4, 5], although showing the inverse-quadratic dependence on doping density, are defined by a defect-assisted mechanism, and hence

could be nonfundamental and process-dependent. This theory has been succinctly disputed by Haug and Schmid[6]. They concluded, by qualitatively but thoroughly reviewing theoretical and experimental studies of recombination mechanisms, including that in highly excited silicon[15], that BBA is the predominant process in highly doped silicon.

In this paper we provide quantitative support for this conclusion by demonstrating that no recombination mechanism involving bound states, or traps, of *common* (unavoidable) defects in silicon can yield hole lifetimes that are consistent with the measured lifetimes[4, 5] and/or with their temperature dependence[4]. We consider both trap-assisted-Auger (TAA)[16, 17] and Shockley-Read-Hall (SRH)[18] recombination mechanisms, and we account for the likely possibility that the defect density depends on the doping density[19].

To further support the conclusion that BBA predominates in highly doped silicon, we present new hole lifetime data that imply erroneous interpretations in previous measurements[9] and that are in good accord with [4] and [5]. We also discuss possible sources of error in the other measurements[7, 8, 10, 11] that have stimulated the controversy concerning recombination and lifetime in highly doped silicon. Finally we give brief theoretically based explanations for the inconsequence of the second and third reasons mentioned above to suspect the unimportance of BBA.

2. MEASURED LIFETIMES

Carrier lifetimes in highly doped and highly excited silicon have been measured and reported extensively in the literature. In Fig. 1 we have plotted the most familiar hole lifetime (τ_p) data vs the donor doping density N_D for n^+ silicon at room temperature. For $N_D > 5 \times 10^{18} \text{ cm}^{-3}$, there is an obvious inverse-quadratic dependence shown by a majority of the data[4, 5]:

$$\tau_p \approx \frac{1}{C_p N_D^2} \quad (1)$$

where $C_p \approx 1 - 3 \times 10^{-31} \text{ cm}^6/\text{sec}$ [4, 5]. For p^+ sil-

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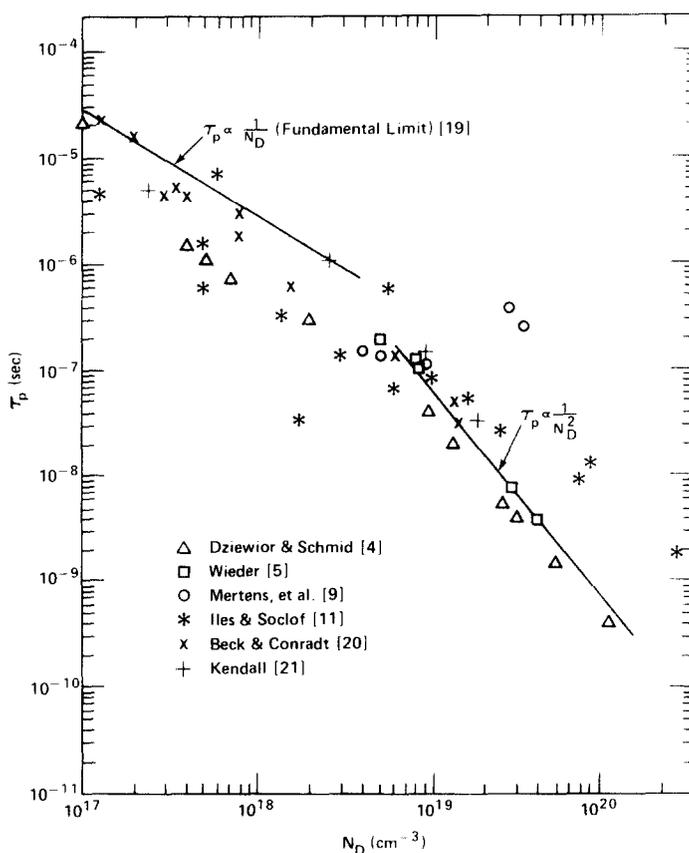


Fig. 1. Measured hole lifetimes vs donor doping density in n -type silicon at room temperature. The $\tau_p \propto 1/N_D$ line represents the fundamental upper limit in nondegenerate silicon suggested in [19]. The $\tau_p \propto 1/N_D^2$ line emphasizes the prevalent dependence observed in n^+ silicon.

icon virtually all of the electron lifetime (τ_n) data show

$$\tau_n \approx \frac{1}{C_N N_A^2} \quad (2)$$

where N_A is the acceptor doping density and $C_N \approx 1 \times 10^{-31} \text{ cm}^6/\text{sec}$ [4].

The hole lifetimes measured by Mertens, *et al.*[9] at $N_D \approx 3 \times 10^{19} \text{ cm}^{-3}$, which are much longer than the lifetimes characterized by (1), are representative of all the experimental results[7-11] that have led to the theory of prevalent defect-assisted recombination in n^+ silicon. This theory[7-9] contends that the lifetimes conforming to (1) are nonfundamental, i.e. they are limited by process-sensitive defects, and that the actual BBA rate coefficient is considerably smaller than C_p .

It is curious that no significant discrepancies have occurred among the independent determinations of electron lifetime in p^+ silicon. Indeed (BBA) rate coefficients consistent with (2) have been inferred experimentally[4, 10] and derived theoretically[2]. A possible explanation for the $\tau_n(N_A)$ consistency in contrast to the $\tau_p(N_D)$ inconsistency is that *common* defects are prevalent in n^+ silicon but not in p^+ silicon. This possibility is promoted by the recent recognition of a prevalent, unavoidable (fundamental) acceptor-

type defect in nondegenerate silicon[19]. This defect, possibly the silicon divacancy, is more soluble in n -type than in p -type silicon, which implies, for a particular doping density, longer ultimate minority-carrier lifetimes for electrons than for holes[19]. If indeed the measured $\tau_p(N_D)$ dependence in (1) is defined by a defect-assisted mechanism, then most probably the defect is this one, which is the only one thought to be both unavoidable and significant.

Thus the analytic approach we take here to identify the predominant recombination mechanism in n^+ silicon is first to extend the defect solubility model of [19] by accounting for electron degeneracy and energy-gap narrowing, and then to describe the TAA and SRH processes involving the trap levels associated with this common defect. These descriptions, and slight variations thereof that account for theoretical-experimental ambiguities concerning the defect, are finally compared with (1) to check the likelihood of significant TAA and/or SRH recombination in n^+ silicon.

3. COMMON DEFECT SOLUBILITY IN n^+ SILICON

To describe the defect solubility in highly doped silicon, we must account for the majority-carrier de-

generacy and the energy-band distortion, both of which were justifiably neglected in [19]. Here we employ a more general description of the defect density in terms of the band structure derived by Longini and Greene[22]:

$$N_i^- = N_{\bar{0}}^- \exp\left(\frac{E_F - E_i}{kT_f}\right) \quad (3)$$

where N_i^- and $N_{\bar{0}}^-$ are the ionized defect concentrations for the "extrinsic" ($N_D \gg n_i(T_f)$) and "intrinsic" ($N_D \ll n_i(T_f)$) cases respectively, and E_F and E_i are the "extrinsic" and "intrinsic" Fermi levels at the defect formation temperature T_f . The expression (3) is based on the fundamental law of thermodynamics that requires the total system free energy to be a minimum in equilibrium. Noteworthy assumptions[22] made in the derivation of (3) are that each defect is acceptor-type and creates a single level in the energy gap, which can be singly charged (negative) or neutral; and that $N_i \ll N_D$ where N_i is the total defect concentration.

To express (3) and ultimately N_i in terms of N_D , we assume complete ionization of the donors[3] and a rigid-energy-band model[14]. Then at T_f ,

$$N_D \simeq n \simeq N_c F_{1/2} \left(\frac{E_F - E_c^{el}}{kT_f}\right) \quad (4)$$

where E_c^{el} is the effective electrical conduction band edge, which is shifted below the intrinsic edge because of heavy-doping effects[14]. The position of E_F relative to E_c^{el} can be determined by using Nilsson's empirical approximation for the Fermi-Dirac integral $F_{1/2}$ [23]. The exponential argument in (3) can be written as

$$\begin{aligned} \frac{E_F - E_i}{kT_f} &\equiv \frac{(E_F - E_c^{el}) + (E_c^{el} - E_i)}{kT_f} \\ &\simeq \frac{E_F - E_c^{el}}{kT_f} + \frac{E_g^i/2 - \Delta E_c^{el}}{kT_f} \end{aligned} \quad (5)$$

where E_g^i is the intrinsic energy gap and ΔE_c^{el} is the electrical conduction-band-edge shift[14], which depends on N_D . Thus (3-5) describe N_i^- in terms of $N_{\bar{0}}^-$ and N_D . The total defect density is the sum of N_i^- and N_i^0 , the unionized defect concentration which we assume approximately equals $N_{\bar{0}}^-$ [19]. Hence, for n^+ silicon ($E_F - E_i \gg kT_f$),

$$N_i(N_D) \simeq N_i^0 \exp\left(\frac{E_F - E_i}{kT_f}\right) \quad (6)$$

where $N_i^0 \sim 10^{11} \text{ cm}^{-3}$ [19] and where the dependence of the exponential function on N_D is given by (4) and (5).

Note that (3-6) are evaluated at the defect formation temperature T_f , which was estimated to be about 600K [19]. Therefore E_g^i is calculated according to [24] at 600K and ΔE_c^{el} [14] is calculated using the electron density-of-states effective mass at 600K [25]. We find that (6) yields [26]

$$N_i \simeq N_D^{0.95}; \quad (7)$$

that is, the dependence of N_i on N_D is nearly linear for degenerate silicon, whereas it is exactly linear for nondegenerate silicon[19].

4. TAA AND SRH RECOMBINATION IN n^+ SILICON

The most probable defect-assisted recombination mechanisms in n -type silicon[1] are those involving multi-phonon and/or cascade-phonon emissions, i.e. the SRH process, and involving secondary electron excitation, i.e., the TAA process. We illustrate in Fig. 2 the four SRH[18] and the four predominant TAA[27, 28] hole and electron capture and emission processes involving a single bound state in n -type silicon. (We neglect processes that would involve the donor impurity level since in n^+ silicon this level is shifted, because of screening effects, into the conduction band[3, 14].) Process (a) is the TAA capture of an electron from the conduction band by the trap via the excitation of an Auger electron. Process (a') is the inverse process, i.e. the emission of an electron from the trap into the conduction band via an impact ionization. The rates (per unit time per unit volume) of processes (a) and (a') are given by [28]

$$r_a = C_n^{\text{TAA}} n^2 N_t (1 - f_t) \quad (8)$$

and

$$r_{a'} = e_n^{\text{TAA}} n N_t f_t, \quad (9)$$

where C_n^{TAA} is the electron capture coefficient for process (a), e_n^{TAA} is the electron emission coefficient for process (a'), and f_t is the trap occupation probability. Similarly process (b) is the TAA capture of a hole from the valence band by the trap, i.e. the transition of an electron from the trap level to the valence band via an Auger electron excitation. The inverse process (b') is the emission of a hole from the trap to the valence band, i.e. the capture of an electron from the valence band via an impact ionization. The rates of processes (b) and (b') are [28]

$$r_b = C_p^{\text{TAA}} n p N_t f_t, \quad (10)$$

and

$$r_{b'} = e_p^{\text{TAA}} n N_t (1 - f_t) \quad (11)$$

where C_p^{TAA} and e_p^{TAA} are the hole capture and emission coefficients.

The processes (c), (c'), (d) and (d') in Fig. 2 are the corresponding SRH processes. Their rates are [18]

$$r_c = C_n^{\text{SRH}} n N_t (1 - f_t), \quad (12)$$

$$r_{c'} = e_n^{\text{SRH}} N_t f_t, \quad (13)$$

$$r_d = C_p^{\text{SRH}} p N_t f_t, \quad (14)$$

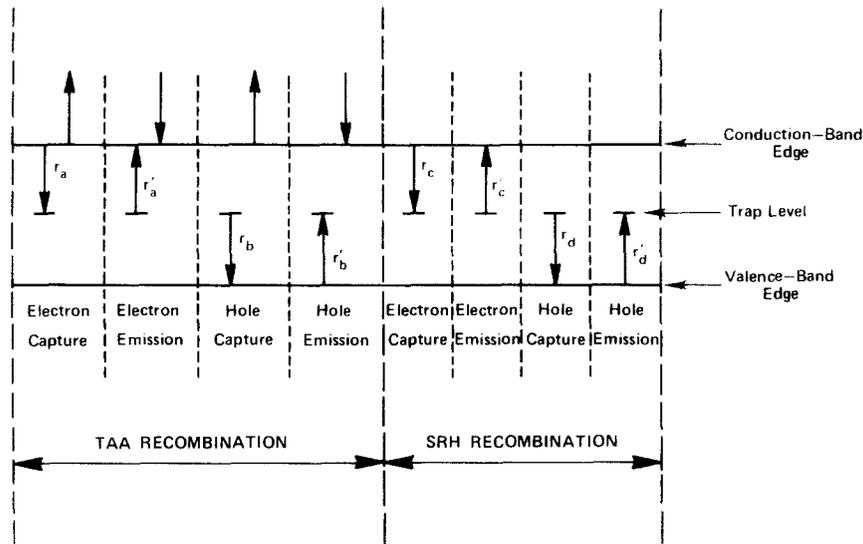


Fig. 2. Electron-transition diagram of TAA and SRH recombination mechanisms in n -type silicon.

and

$$r_{d'} = e_p^{\text{SRH}} N_t (1 - f_t) \quad (15)$$

where C_n^{SRH} , C_p^{SRH} , e_n^{SRH} and e_p^{SRH} are the SRH capture and emission coefficients for electrons and holes.

To characterize the composite defect-assisted recombination rate in n^+ silicon, we extend Landsberg's description[29] by accounting for the electron degeneracy and the energy-gap narrowing. Employing the principle of detailed balance at thermal equilibrium, we relate the electron and hole emission coefficients to the corresponding capture coefficients:

$$e_n^{\text{TAA}} = C_n^{\text{TAA}} N_c \exp\left(\frac{E_t - E_c^{el}}{kT}\right) \beta, \quad (16)$$

$$e_p^{\text{TAA}} = C_p^{\text{TAA}} N_v \exp\left(\frac{E_v^{el} - E_t}{kT}\right), \quad (17)$$

$$e_n^{\text{SRH}} = C_n^{\text{SRH}} N_c \exp\left(\frac{E_t - E_c^{el}}{kT}\right) \beta, \quad (18)$$

where E_t is the trap level. In (16) and (18) we have defined β as the degeneracy factor[26],

$$\beta = \frac{F_{1/2}\left(\frac{E_F - E_c^{el}}{kT}\right)}{\exp\left(\frac{E_F - E_c^{el}}{kT}\right)}. \quad (20)$$

In (16–20) the electron and hole densities have been defined based on a rigid-band model in which the conduction band edge E_c^{el} and the valence band edge E_v^{el} are the *effective* electrical edges, which are defined by a combination of the many-body effects and the effects of lattice disorder (random dopant distribution)[14].

The steady-state occupation probability f_t can be determined using the steady-state condition that the net electron capture rate is equal to the net hole capture rate, i.e.

$$(r_a - r_a') + (r_c - r_c') = (r_b - r_b') + (r_d - r_d'). \quad (21)$$

Then the net recombination rate due to SRH and TAA processes through the defect is

$$U = (r_a - r_a') + (r_c - r_c') \quad (22)$$

$$= \frac{pn - n_{ie}^2}{(C_p^{\text{TAA}} n + C_p^{\text{SRH}}) N_t \left[n + \beta N_c \exp\left(\frac{E_t - E_c^{el}}{kT}\right) \right] + (C_n^{\text{TAA}} n + C_n^{\text{SRH}}) N_t \left[p + N_v \exp\left(\frac{E_v^{el} - E_t}{kT}\right) \right]}$$

and

$$e_p^{\text{SRH}} = C_p^{\text{SRH}} N_v \exp\left(\frac{E_v^{el} - E_t}{kT}\right) \quad (19)$$

where n_{ie}^2 is the equilibrium pn -product in n^+ silicon[14]:

$$n_{ie}^2 = n_i^2 \beta \exp\left(\frac{\Delta E_g^{el}}{kT}\right); \quad (23)$$

ΔE_g^{el} is the electrical energy-gap narrowing[14]. The corresponding minority hole ($p \ll n$) lifetime is

$$\tau_p = \frac{p - p_0}{U} \approx \frac{1}{(C_p^{TAA}n + C_p^{SRH})N_t} \left[1 + \beta \frac{N_c}{n} \exp\left(\frac{E_t - E_c^{el}}{kT}\right) \right] + \frac{1}{(C_n^{TAA}n + C_n^{SRH})N_t} \left[\frac{N_v}{n} \exp\left(\frac{E_v^{el} - E_t}{kT}\right) \right]. \quad (24)$$

In the limiting case that $C_p^{TAA}n \ll C_p^{SRH}$ and $C_n^{TAA}n \ll C_n^{SRH}$, the TAA processes are insignificant and (24) reduces to the SRH lifetime, which is not explicitly dependent on n , or N_D . If the TAA processes predominate however, i.e. if $C_p^{TAA}n \gg C_p^{SRH}$ and $C_n^{TAA}n \gg C_n^{SRH}$, which is possible for sufficiently high electron density, then (24) defines a lifetime that is explicitly inversely proportional to n , or N_D .

As mentioned before, most of the measured hole lifetimes in n^+ silicon consistently show a $1/N_D^2$ dependence. This dependence reflects defect-assisted recombination (SRH and/or TAA) only if the trap density increases with the doping density. For SRH recombination to dominate, (24) shows that N_t must be proportional to N_D^2 ; for TAA recombination to dominate, (24) shows that N_t must be proportional to N_D . Thus the TAA process involving the common defect considered in Section 3 could yield the $\tau_p \propto 1/N_D^2$ dependence observed in n^+ silicon. To investigate this possibility, we must know C_n^{TAA} and C_p^{TAA} .

Landsberg and Robbins[28, 30] have estimated the TAA coefficients theoretically by assuming a hydrogen-like trap-level wave function and a rigid-parabolic-band model. Their analysis gives

$$C_{n,p}^{TAA} = 2.23 \times 10^{-26} \left(\frac{m_0}{\epsilon_s m_e^*} \right)^2 N_{n,p} |F|^2 \text{ cm}^6/\text{sec} \quad (25)$$

where m_e^* is the (sub-band) density-of-states electron effective mass, which is $0.36m_0$ at 300K[25], and where $N_{n,p}$, with units $(\text{eV})^{-3}$, is

$$N_n = \frac{1}{E_c^3} \quad (26)$$

for process (a) in Fig. 2 and is

$$N_p = \frac{E_{ic}^{5/2}}{(E_g^{op} - E_{ic})^{3/2} (E_g^{op})^4} \quad (27)$$

for process (b) in Fig. 2. In (26) and (27), E_{ic} is the trap level referenced to the optical conduction-band edge E_c^{op} ; $E_g^{op} = E_g^i - \Delta E_g^{op}$ is the optical energy gap, i.e. the gap in energy-momentum space, which is smaller than E_g^i because of many-body effects[12–14]. In (25), $|F|^2$ accounts for wave-function overlap and, although not precisely known, is estimated to be of

the order of 0.1[30]. Later we show that this uncertainty does not threaten the validity of our conclusions.

In evaluating (25) we assume that the trap level E_{ic} referenced to the intrinsic conduction-band edge is fixed since the screening effects on a deep level are negligible[17]. However E_{ic} varies with N_D :

$$E_{ic} = E_{ic}^i - \Delta E_c^{op} \quad (28)$$

where ΔE_c^{op} , which depends on N_D [14], is the rigid shift of E_c^{op} due to many-body effects. For the common defect under consideration, it was estimated that $E_{ic}^i \simeq 0.4 \text{ eV}$ [19]. Using (28), a model for $\Delta E_c^{op}(N_D)$ [14], and (25), we find that $C_n^{TAA} \sim 2 \times 10^{-27} \text{ cm}^6/\text{sec}$ and $C_p^{TAA} \sim 2 \times 10^{-29} \text{ cm}^6/\text{sec}$, with variations of less than a factor of two occurring as N_D increases from 10^{18} cm^{-3} to 10^{20} cm^{-3} [26].

With the $N_t(N_D)$ dependence defined by (6) and (7), (24) yields an approximate $\tau_p \propto 1/N_D^2$ dependence only if the TAA mechanism predominates over the SRH mechanism. This is the case, as indicated by (24), if

$$C_p^{TAA}N_D \gg C_p^{SRH}. \quad (29)$$

Assuming $C_p^{SRH} \sim 10^{-8} \text{ cm}^3/\text{sec}$ [19] and using our estimated value for C_p^{TAA} , we see that (29) is invalid for all practical values of N_D ($< 10^{21} \text{ cm}^{-3}$). Thus the predominant mechanism involving the common defect under consideration, for degenerate as well as nondegenerate silicon, is SRH, which implies a $\tau_p \propto 1/N_D$ dependence. The predicted $\tau_p(N_D)$ characteristic hence is totally inconsistent with the preponderance of the measured hole lifetimes in n^+ silicon[4, 5].

We now consider the possibility that uncertainties in the theoretical analysis might have caused this inconsistency. A major uncertainty is the value of $|F|^2$ in (25), to which C_p^{TAA} is directly proportional. However increasing $|F|^2$ to unity, which is its maximum possible value, although validating (29) for $N_D > 10^{20} \text{ cm}^{-3}$, still results in TAA lifetimes that are an order-of-magnitude longer than those measured[26]. Another uncertainty is E_t , or E_{ic} . However only when $E_{ic} \rightarrow E_g^{op}$ does C_p^{TAA} , given by (25) and (27), become large enough to yield TAA lifetimes that conform with the measured ones[26]. Such a shallow level is not characteristic of unavoidable defects in silicon. We therefore conclude that TAA recombination cannot be inferred to be a predominant mechanism in n^+ silicon.

As we mentioned previously with reference to (24), SRH recombination will yield a $\tau_p \propto 1/N_D^2$ dependence only if $N_t \propto N_D^2$. Furthermore, because of the consistency of most of the hole lifetimes measured in a variety of n^+ silicon samples[4, 5], the defect must be common, or unavoidable.

It is interesting to note that the defect we have considered has properties similar to those of the

silicon divacancy[19], which produces a second trap level in addition to the one that defines the defect solubility in nondegenerate silicon. This second level, located 0.23 eV below E_c [31], corresponds to a doubly-charged acceptor-type state. According to Shockley and Moll[32], such a level will cause an $N_i \propto N_D^2$ dependence for sufficiently high N_D . For the divacancy, the quadratic dependence results when $N_D > 10^{19} \text{ cm}^{-3}$, for which τ_p , given by (24) but controlled by the SRH mechanism, not only follows the $1/N_D^2$ dependence but also conforms to the measured lifetimes[26].

This consistency, although based on crude theoretical estimations necessitated by the uncertain properties of the defect, ostensibly supports the recent contentions[7–9] that defect-assisted recombination is predominant in n^+ silicon. To further investigate this, we examine the theoretical temperature dependence of the SRH lifetime, and compare it with the temperature dependence of the measured lifetime.

According to Mott[1] and to Henry and Lang[33], the SRH lifetime is quite sensitive to temperature. Its temperature dependence, as described by [33], is

$$\tau^{\text{SRH}} \propto T^{1/2} \exp\left(\frac{\Delta E}{kT}\right) \quad (30)$$

where ΔE is the activation energy for the minority-carrier capture process, which typically is ~ 0.1 eV.

Dziewior and Schmid[4] measured the minority-carrier lifetime in highly doped silicon at three different temperatures: 77, 300 and 400K. They found however that the measured hole lifetime is not strongly dependent on temperature; increasing T from 77 to 400K resulted in an increase in the measured τ_p of only 21%, which sharply contrasts (30). This large discrepancy between the measured $\tau_p(T)$ dependence in n^+ silicon and the theoretical $\tau^{\text{SRH}}(T)$ dependence thus eliminates the possibility that the SRH mechanism underlies the observed $\tau_p(N_D)$ dependence in n^+ silicon. Contrarily good agreement between the calculated temperature dependence of the BBA lifetime[2] and the measured temperature dependence[4] provides additional evidence for the predominance of BBA.

5. SUMMARY AND DISCUSSION

In an attempt to explain the observed inverse-quadratic dependence of hole lifetime on doping density in n^+ silicon[4, 5] in terms of a defect-assisted recombination mechanism, we have modeled the solubility of a prevalent, unavoidable defect[19] in n^+ silicon and have described the doping dependence of the TAA[16, 17, 27, 28, 30] and SRH[18] recombination mechanisms involving this defect. This characterization includes the effects of electron degeneracy and energy-gap narrowing[12–14]. The resulting $\tau_p(N_D)$ predictions are inconsistent with the measured lifetimes. Although the TAA mechanism implies an inverse-quadratic lifetime dependence on N_D , the val-

ues of τ_p predicted are much longer than those measured. The SRH mechanism could conform to the observed $\tau_p(N_D)$ characteristic, but its strong temperature dependence is inconsistent with the weak temperature dependence of the measured τ_p [4], which in fact is characteristic of BBA[2].

We thus conclude, in accord with [6], that the preponderance of the lifetime-vs-doping density data[4, 5], for holes as well as electrons, reflects the dominance of the phonon-assisted BBA recombination mechanism in highly doped silicon. This conclusion is supported further by the measured lifetime in highly excited, low-doped silicon, in which the BAA coefficient C^{BBA} is the sum of the BBA coefficients in n^+ and p^+ silicon, C_p^{BBA} and C_n^{BBA} respectively[1]. The value of C^{BBA} was inferred from luminescence-decay measurements to be about $3.9 \times 10^{-31} \text{ cm}^6/\text{sec}$ at 300K [15]. Based on the conclusion of this paper, C_p^{BBA} and C_n^{BBA} are, respectively, C_p and C_n in (1) and (2), the sum of which approximately equals C^{BBA} . This self-consistency supports the theory of predominant BBA in highly doped silicon and solidifies the results that $C_p^{\text{BBA}} \approx 1 - 3 \times 10^{-31} \text{ cm}^6/\text{sec}$ and that $C_n^{\text{BBA}} \approx 1 \times 10^{-31} \text{ cm}^6/\text{sec}$.

To illustrate our results, we plot in Fig. 3 [curve (a)] the calculated composite minority hole lifetime in highly doped n -type silicon that reflects recombination via the BBA mechanism and the SRH and TAA mechanisms involving the singly charged state of the common (unavoidable) defect, the density of which is described by (6) and (7) and the trap level of which is 0.4 eV below the conduction-band edge as estimated in [19]. The BBA rate coefficient is taken as $C_p^{\text{BBA}} \approx 2 \times 10^{-31} \text{ cm}^6/\text{sec}$ [4, 5]. We note that for high N_D , the calculated curve is in good agreement with most of the measured lifetime data, which are also plotted in Fig. 3. For lower N_D , the curve coincides with the ultimate lifetimes measured, which reflects the fundamental nature of the defect[19].

To illustrate the effects of nonfundamental defects in silicon, we have increased the defect density by an order-of-magnitude and have plotted the resulting $\tau_p(N_D)$ dependence also in Fig. 3 [curve (b)]. This lifetime is significantly lower than that of curve (a) for moderate N_D where SRH predominates, and reflects the commonly observed scatter in lifetimes measured at these doping densities. However for high N_D , curves (a) and (b) are nearly coincident reflecting the predominance of fundamental BBA. We observe that in the calculations for both curves (a) and (b), the TAA mechanism is never significant.

Note in Fig. 3 that for high N_D the lifetimes measured by [9] and [11] are considerably longer than those predicted by the curves. Indeed the data of [9] are representative of other measurements[7, 8, 10] that have led to the contention that defect-assisted recombination is prevalent in n^+ silicon. We now discuss possible errors and misinterpretations made in these measurements.

Possin *et al.*[7, 8] and Weaver and Nasby[10] inferred upper limits for C_p^{BBA} that are significantly

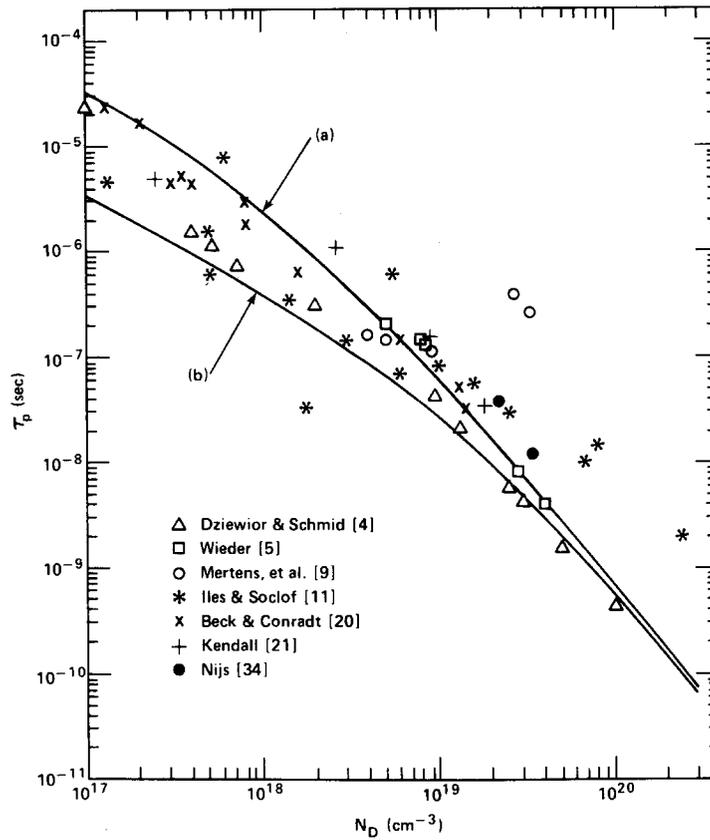


Fig. 3. Calculated composite [SRH + TAA + BBA ($C_p^{BBA} = 2 \times 10^{-31} \text{ cm}^6/\text{sec}$)] lifetime vs donor doping density in n -type silicon at room temperature. The properties of the defect corresponding to curve (a) are those described in Section 3 and in [19]. Curve (b) results when the defect density is increased by an order-of-magnitude. The measured lifetimes, including those of [34], are superimposed on the curves.

smaller than C_p in (1) by comparing measured electron-beam and light-induced currents in n^+p junctions with theoretical predictions. These limits however depend on complex carrier-transport models, the parameters of which cannot be accurately evaluated because of uncertainties in the physical characterization of heavy-doping effects in silicon[35]. For example, the induced currents depend strongly on the effective electric field for holes in the n^+ region, which is quite sensitive to the doping-density profile and the energy-gap narrowing. Moreover there is an additional uncertainty in the distribution of electron-hole pairs generated by the electron beam[36] and by the light[37].

Mertens *et al.*[9] and Iles and Soclof[11] determined the minority hole diffusion length L_p by comparing with theory measured photocurrents of p^+n^+ solar cells fabricated on uniformly doped n^+ silicon substrates. They then derived the hole lifetime using $\tau_p = L_p^2/D_p$ where D_p is the hole diffusion coefficient. This indirect determination of τ_p has inherent limitations in accuracy because of the uncertainty in D_p and because the actual experimental error is effectively doubled when τ_p is calculated. Moreover when N_D approaches 10^{20} cm^{-3} as in [11], L_p becomes very short and hence its determination by this technique becomes

inaccurate because the photocurrent comprises significant components from regions of the solar cell other than the n^+ base. Finally the measurement requires precise knowledge of the absorption coefficient, which is ambiguous in n^+ silicon[37].

We now discuss recent experimental results[34] that negate the lifetimes measured in [9]. The measurements of [9] have been repeated on new cells that were chemically etched prior to the junction formation to ensure a uniform doping density in the n^+ base. The new hole lifetimes[34], which are plotted in Fig. 3, are more than an order-of-magnitude shorter than those previously reported[9] and are in good accord with (1), which has been shown herein to represent the fundamental phonon-assisted BBA mechanism. It is interesting to note finally that these lifetimes were not affected by high-temperature phosphorus gettering nor by low-temperature hydrogen annealing[34], which is further evidence for the dominance of BBA.

We conclude by addressing the last two reasons mentioned in Section 1 to suspect the unimportance of BBA in n^+ silicon. The narrowing of E_g^{op} , which renders C_p^{BBA} dependent on N_D , can be seen to be insignificant by considering the theoretical dependence of C_p^{BBA} on E_g^{op} . According to Loch-

mann[38], $C_p^{BBA} \propto (E_g^{op})^{1/2}$. Since $\Delta E_g^{op} \sim 0.1 E_g^{op}$ in n^+ silicon [12–14], its influence on C_p^{BBA} and on $\tau_p(N_D)$ is negligibly small. The theoretical prediction of C_p^{BBA} at 300K [2] is two–four times smaller than the measured value C_p in (1) [4, 5]. This discrepancy is inexplicable and implies minor errors in the theory and/or the experiments. It does not weaken the arguments purporting predominant BBA in highly doped silicon. It does however stress the uncertainty in the actual value of C_p^{BBA} , which for now can only be given imprecisely as $1 - 3 \times 10^{-31}$ cm⁶/sec.

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