Direct monitoring of minority carrier density during light induced degradation in Czochralski silicon by photoluminescence imaging

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In this paper we present a new method for studying the light induced degradation (LID) process, in which the minority carrier density is monitored directly during light soaking by photoluminescence (PL) imaging. We show experimentally that above a certain minority carrier concentration limit, $\Delta n_{\text{lim}}$, the boron oxygen (B-O) defect generation rate is fully independent of the injected carrier concentration. By simulation we determine $\Delta n_{\text{lim}}$ for a range of p-type Czochralski silicon samples with different boron concentrations. The normalized defect concentrations, $N_t^*$, is determined for the same samples by time-resolved Quasi Steady State Photoconductance (QSSPC) measurements. After 10 minutes of light degradation, no correlation between $\Delta n_{\text{lim}}$ and $N_t^*$ is observed. These results indicate that the role of the excess carriers during the rapid decay is to first change the charge state of the defects by shifting the electron quasi-Fermi level across the energy level of the defect centre in its passive state ($E_{\text{lat}} = E_V + (635\pm18) \text{ meV}$) and that, subsequently, another rate-determining step proceeds before the defect centre becomes recombination active.

I. INTRODUCTION

Severe lifetime reduction with carrier injection is an unfortunate characteristic of p-type Czochralski silicon materials. During a time period of a day or less the electron lifetime is typically reduced to about 10% of the initial lifetime. The lifetime reduction is attributed to a recombination centre that becomes activated by charge carriers. Two exponential decay processes, one rapid and one slow, describe the degradation kinetics. Both decays are found to be related to the simultaneous presence of oxygen and boron in the Si material, through a possible boron-oxygen defect (B-O defect), but neither the chemical composition nor the energy level of the defect has so far been determined experi-
mentally. It has been shown that the rates of the rapid and slow degradation are proportional to the square of the boron concentration \([B]^2\), while they are independent of the oxygen concentration.\(^2\) The role of the excess carriers has, however, not been studied in detail.

In the model of Schmidt et al. the role of the excess minority carriers in the defect generation were believed not to be directly involved in the defect reaction, but their presence was thought to trigger the reaction by (i) charging the state of the \(O_{2i}\), which might lead to an increase in the \(O_{2i}\) diffusivity; or (ii) enhancing the \(O_{2i}\) diffusivity via a recombination-enhanced diffusion process.\(^3\) Recent work published in 2011\(^4\) stated, however, that this type of motion of an oxygen dimer is a highly unlikely mechanism in solar grade silicon. Accordingly this hypothesis disputed the role of the excess carriers in the model.

In 2010/2011, Voronkov and Falster\(^5,6\) proposed a new degradation model wherein a latent recombination centre is already incorporated in the crystal before carrier injection. According to this model, the centres responsible for the slow and rapid lifetime decays are \(B_iO_{2i}\) and \(B_sO_{2i}\) respectively. In p-type silicon the activation of \(B_iO_{2i}\) (defect centre responsible for the rapid lifetime decay) is explained through a series of steps where the quadratic proportionality between hole concentration and lifetime is accounted for by the capture of holes by a negative latent centre. The positive latent centre is then reconstructed into a transient centre. One of the final transitions leading to the formation of a recombination-active centre is a de-charging of the transient centre from positive to neutral by electron injection.

Commonly, the light induced degradation (LID) is monitored either by Microwave Photo Conductance Decay (\(\mu w\)-PCD) or Quasi Steady State Photo Conductance (QSSPC) lifetime measurements of a sample during light exposure, or by measuring the change in open circuit voltage \((V_{OC})\) of a solar cell being subjected to an applied forward bias. However, none of these methods retain the information of the minority carrier density in the sample during the extended light exposure or applied forward bias treatments. It has been shown that very low carrier density levels are sufficient to activate the recombination centres\(^7,8\) but the degradation process is, nevertheless, completely reliant on increased carrier densities.\(^8\) If the injection or heat source is removed, the degradation will cease im-
mediately. Several groups have studied the influence of varying illumination intensity on the B-O defect generation rate. These studies\textsuperscript{9,10,11} have shown that the generation rate of the defect saturates at a certain illumination intensity. For highly degradable material, i.e. material with high concentrations of boron and oxygen, the defect generation rate saturates at about 1mW/cm\textsuperscript{2}. The intensity of the illumination source and the excess carrier density is linked through the optical properties of the sample and the carrier lifetime. A certain illumination intensity threshold value therefore corresponds to a certain excess carrier threshold value, $\Delta n_{\text{lim}}$, but the same illumination intensity gives widely different excess carrier densities for different materials.

In this work we experimentally demonstrate that such an excess carrier threshold value, $\Delta n_{\text{lim}}$, exists and that above $\Delta n_{\text{lim}}$ the generation rate is fully independent of carrier concentration. These findings are uncovered by a new way of studying the LID process, in which the excess carrier density is monitored directly during the light soaking. Local light induced defect generation, wherein the wafer is exposed to light in a confined area, causes the excess electrons to diffuse out into the wafer, allowing us to study the degradation effect of a continuously varying electron concentration. This concentration can be monitored by means of photoluminescence (PL) imaging. By applying simulations for the very low carrier concentrations, we are able to quantify $\Delta n_{\text{lim}}$. The procedure is performed on a series of samples and the results are used to investigate whether there is a correlation between $\Delta n_{\text{lim}}$ and the B-O defect density. Finally, we discuss whether the role of the excess carriers is to simply shift the quasi-Fermi energy causing a charge state change.

II. EXPERIMENTAL METHODS

The samples used in this study are boron doped Cz-Si wafers with a boron concentration ranging from $3.5 \times 10^{15}$ to $8.5 \times 10^{15}$ cm\textsuperscript{3}. Prior to the measurements, the wafers were cleaned with RCA and thereafter received a double side passivation by plasma-enhanced chemical-vapor deposited (PECVD) hydrogenated amorphous silicon (a-Si:H). a-Si:H-passage has the advantage compared to silicon nitride (a-SiN\textsubscript{x}:H) passivation that no inversion channel is formed on p-type material. An inversion channel could possibly introduce a smearing effect on the PL signal detected by the camera.\textsuperscript{12} The
a-Si:H.-passivation still provides an excellent and stable level of surface passivation with a surface recombination velocity below 40 cm/s. In order to ensure that no defect centres are generated before the start of the measurements, the samples were deactivated by an annealing at 210 °C for 15 min on a hotplate.\(^2\)

The B-O defect densities are determined by time resolved lifetime measurements. The lifetime degradation was monitored by a custom built, automated QSSPC setup using an externally controlled bias lamp for in-situ illumination between measurements. The bias light source was a halogen lamp with an intensity of approximately 50 mW/cm. The temperature of the sample was monitored by a thermistor coupled to the sample stage, but the temperature change of the stage and sample due to illumination was not significant for the measured lifetime. According to Shockley Read Hall theory\(^{13}\), the defect concentration can be directly related to the lifetime, provided that the generated defects are the dominating recombination mechanism. The normalized defect concentration, \(N_t^*\), is related to the measured lifetime by the following equation:

\[
N_t^* (t) = \frac{1}{\tau(t)} - \frac{1}{\tau(0)}
\]

where \(\tau(0)\) is the initial carrier lifetime before LID and \(\tau(t)\) is the carrier lifetime after a certain exposure time.\(^{14}\) The lifetimes were measured at a fixed injection level corresponding to one tenth of the doping level for each sample respectively.\(^2\)

FIG. 1 Sketch showing the setup for direct monitoring of excess carrier density during light induced degradation. The wafer is exposed to a focused and shaped laser beam while the PL signal is collected by a CCD camera.
The PL measurements were performed with a LIS-R1 system from BTimaging. In the chamber, a 4.5 mW diode laser with a wavelength of 780 nm was installed in order to illuminate a specific area of the wafer. A principle sketch of the setup is given in Fig 1. The laser beam was shaped and focused into a narrow line with a Gaussian half width of $50 \pm 5 \mu m$. The incidence angle was set to 10 degrees to avoid detection of the specular reflection from the laser beam by the charge-coupled device (CCD) camera. When the laser is turned on, the generated carriers will diffuse from the illuminated area into the adjacent area following the continuity equation. In silicon containing boron and oxygen the diffusing carriers generate B-O defects that reduce the lifetime far beyond the borders of the illuminated area. As the concentration of B-O defects increases with time, the diffusion length of the carriers decreases. This can easily be observed by imaging the PL generated from a localized area in a p-type Cz-Si sample at $t = 0$ and $t \neq 0$. See Fig. 2.

![Image](image.png)

**FIG. 2** PL intensity data (normalized to exposure time) taken a) immediately when the light soaking is started, $t = 0$ and b) after light soaking for $t = 3$ min. The position and width of the exposed area is superimposed on the image, as indicated by the vertical grey line. Data acquisition time is 0.5 s. By comparing the images it is obvious that the carrier density decreases with increased time of light exposure. This is due to the shorter minority carrier lifetime caused by the generation of B-O defects.

The laser shown in Fig. 1 has two functions. One is to provide enough excess carriers over time to create an area of reduced lifetime, visible as a groove-shaped feature in the lifetime maps. The other is to act as an excitation source during the PL measurements of direct monitoring of minority carrier density with local excitation, which in principle can be performed on any type of silicon material. As discussed in the introduction, the B-O defect generation rate is independent of carrier concentration above a certain value, $\Delta n_{\text{lim}}$. Therefore, a lifetime plateau in the bottom of the generated lifetime groove-shaped profile is formed. In Fig. 3, a principle sketch of approximately one half of the lifetime profile is shown. The four regions correspond to the degraded region, the exposed region, the transition region and the unaffected region. Looking at the charge carrier concentration profile for an arbi-
trary time \( t \neq 0 \), the carriers that are generated in the exposed region will be subject to a uniform diffusion length in the already degraded region. However, upon reaching the transition region the lifetime will rapidly change to the better. This will appear as a change in the slope of the spatial carrier concentration profile in the transition region. The carrier concentration that corresponds to this change in the slope is thus the carrier concentration limit value, \( \Delta n_{\text{lim}} \), where the defect generation rate becomes saturated.

**FIG. 3** Principle sketch showing the spatial carrier concentration in a sample exposed to a thin laser line. For simplicity only half of the lifetime groove is illustrated. The continuous line shows the spatial carrier concentration and the dashed line shows the spatial lifetime profile. The figure shows four regions; the shaded region corresponds to the degraded region, the grey to the exposed, the white to the transition region and the dotted to the unaffected region. The duration of the exposure determines the depth of the lifetime groove.

In the present work we illuminated the material with a focused, low-flux laser for 10 min (peak photon flux, \( \Phi_{\text{low}} = 2.9 \times 10^{16} \text{ s}^{-1}\text{cm}^{-2} \)). After 10 min a groove-shaped lifetime profile is formed and an image of the spatial carrier profile was taken. Unfortunately, the CCD camera cannot detect a PL signal low enough to directly observe the carrier concentrations in the transition region. With a larger carrier concentration (increased excitation) the effect can, however, be seen on the spatial carrier profile. A high photon flux will thus demonstrate the change in the slope on the carrier profile, but will not represent the carrier concentration that was responsible for creating the defects. In order to determine the actual \( \Delta n_{\text{lim}} \) where the defect generation rate becomes saturated we have simulated the spatial carrier profile for these low carrier concentrations. The procedure will be described in section III. The basis for the simulations are; (i) a calibrated PL image of the lifetime groove measured by conventional PL imaging (5 sec) where another (integrated) laser, with a wavelength of 808 nm, is used
to expose the entire wafer and the resulting band-to-band PL signal is measured by a one-megapixel silicon CCD camera, and (ii) two QssPC measurements of the sample performed at \( t = 0 \) and \( t = 10 \) min illumination.

III. DATA ANALYSIS AND SIMULATIONS

The goal of the simulations and analysis presented here is to determine the value of \( \Delta n_{\text{lim}} \). As explained in the previous section, the minority carrier density in the transition region during the spot illumination is too low to be measured directly with the PL imaging setup. Therefore we rely on simulations of the minority carrier profile, which is determined by measurements of the lifetime spatial profile and injection-level dependence before and after illumination.

The simulations of the spatial carrier concentration profile are based on the 2D continuity equation, describing a plane \((x,z)\) normal to the focused laser line:

\[
G(x,z) + D_n \nabla^2 n(x,z) \frac{\Delta n(x,z)}{\tau(\Delta n,x)} = 0
\]  

(1)

Where \( D_n \) is the electron diffusion length. The generation rate \( G(x,z) \) for the focused laser line is given by

\[
G(x,z) = (1 - R_\lambda) \phi \exp \left( - \frac{x}{\sigma} \right) \alpha_\lambda \exp(-\alpha_\lambda z)
\]  

(2)

Here \( \alpha_\lambda \) is the attenuation coefficient for silicon, and \( R_\lambda \) is the reflectance at the laser wavelength (\( \lambda = 780 \) nm). The peak photon flux \( \phi \) and Gaussian beam half width \( \sigma \) were measured by a beam profilometer and a calibrated photo spectrometer to be \( \phi = 2.9 \times 10^{16} \text{ s}^{-1} \text{ cm}^2 \) and \( \sigma = 50 \pm 5 \mu m \) for the spot used during the 10 minutes of light soaking. This illumination generates the defects and hence creates a spatial lifetime profile that reflects the carrier concentration profile during illumination. The carrier density in steady state is approximately homogeneous in the \( z \)-direction (depth of the sample),
so the resulting carrier density values from the simulations are integrated throughout the thickness of
the sample and reported only as function of x.

After the light soaking, the spatial dependence of the lifetime is determined by acquiring a stand-
ard PL lifetime image. The procedure to extract the true spatial lifetime profile from the diffusion-
affected PL image is described in 7. This yields a sigmoidal curve that rises from a low lifetime plat-
eau $\tau_{\text{low}}$ near the spot location to a high lifetime plateau $\tau_{\text{high}}$ far away from the spot, as sketched in Fig.
3.

The injection level dependence is determined by QSSPC measurements on the samples in their de-
activated state, and after being light soaked for 10 minutes with a halogen lamp illuminating the
whole sample. The light source is intense enough to reach the regime of constant defect generation,
and the sample temperature change due to the illumination is negligible. The QSSPC data obtained
before light soaking (deactivated sample) is then representative for the injection level dependence of
the lifetime in the unaffected region ($\tau_{\text{high}}(\Delta n)$), far away from the laser spot ($x \gg \sigma$). The QSSPC da-
ta obtained after light soaking is representative for the low-lifetime ($\tau_{\text{low}}(\Delta n)$) plateau near the spot.
An example of such measurements is shown in Fig. 4. For the transition region, the lifetime is as-
sumed to follow the sigmoidal shape determined from the standard PL lifetime image, but with
$\tau_{\text{low}}(\Delta n)$ and $\tau_{\text{high}}(\Delta n)$ lifetimes adjusted by the injection dependence. The QSSPC data is fitted to a
lifetime model to obtain values below the detection limit in the low-injection regime. The effective
lifetime of the samples is typically not perfectly uniform, due to surface scratches and damage to the
passivation layers caused by handling. Thus, the area-averaged QSSPC lifetime is lower than the lifet-
time in the spot-illuminated areas. The latter are measured in as homogeneous areas as possible to
avoid discrepancies with the simulations. A factor is introduced to scale the averaged QSSPC lifetime
to the PL-determined lifetime in the uniform region. The assumption is then that the injection level
dependence measured by QSSPC is representative also for the small uniform area probed by the fo-
cused line laser.
FIG. 4 Example of QSSPC measurements before and after light soaking for 10 minutes with a normal halogen lamp illuminating the whole sample.

With $\tau(\Delta n, x)$ and $G(x, z)$ determined from measurements, Eq. 1 is solved numerically using the partial differential equation solver FlexPDE.\textsuperscript{15} The carrier concentration limit, $\Delta n_{\text{lim}}$, is extracted from the simulated carrier profile by fitting two exponentially decaying curves to the regions corresponding to the two lifetime plateaus. In this low-injection regime (of constant lifetime with injection level) the decay constant of each fitted curve corresponds to the diffusion length in the corresponding region. The limit, $\Delta n_{\text{lim}}$, is extracted from the carrier profile curve where the normal of the simulated curve crosses the intersection of the two fitted curves, see Fig. 5.

FIG. 5 Simulated carrier profile for the low-flux beam exposure at which the 10 min degradation was performed. The intersection of the two exponential curve fits (dashed lines) gives the carrier concentration limit, $\Delta n_{\text{lim}}$. 

\textit{Minority carrier density [cm}$^{-2}$\textit{]}
The simulations are verified by comparison to a measurement carried out with a laser line spot of higher intensity than the one used during the light soaking. A laser line spot with peak flux of $\phi_{\text{high}} = 1 \times 10^{18} \text{s}^{-1}\text{cm}^{-2}$ and $\sigma = 50 \pm 5 \mu\text{m}$ Gaussian half width was used during acquisition of the spatial carrier concentration profiles. Fig. 6 shows an example where the simulation fits well to the measured carrier concentration profile. The noise floor around $\Delta n = 5 \times 10^{12} \text{cm}^{-3}$ is also visible.

![Graph showing verification of the employed model.](image)

**FIG. 6** Verification of the employed model. The black curve corresponds to a measurement of the spatial carrier profile carried out with the high-flux laser beam. The dashed curve corresponds to Eq. 2 with $\tau(\Delta n, x)$ and $G(x, z)$ determined from measurements.

The simulation and analysis procedure are summarized below:

1. The **spatial profile of the lifetime** is obtained from a calibrated PL image taken after 10 min spot illumination at low photon flux, $\phi_{\text{low}}$.

2. The **injection level dependence** of the lifetime is determined by QSSPC measurements of the samples in their deactivated state and after 10 min light soaking.

3. The **continuity equation is solved numerically**, with the spatial and injection level dependent lifetime data from point 1 and 2.

4. The **carrier concentration limit, $\Delta n_{\text{lim}}$** is extracted from the simulated carrier profile.
IV. RESULTS AND DISCUSSION

A. Direct monitoring of locally injected carriers

In this work, a region of light induced degraded lifetime is generated by locally exposing a sample to a low-flux laser beam. The regions of interest are the degraded, the unaffected and the transition region, where the lifetime changes. (See Fig. 3) By a new method, described in the previous sections, we monitor the minority carrier density directly by photoluminescence imaging. By exciting a large enough concentration of carriers to detect the subsequent PL signal, we can observe what happens to the carrier concentration in the regions of interest. As already discussed, the actual values for $\Delta n_{\text{lim}}$ are too low to be measured directly but carrier profiles generated by a high-flux laser provide valuable information as well. The diffusion length of the material is given directly by the slope of the measured spatial carrier profile independently of the excited carrier concentration. By analyzing the slope of the spatial carrier concentrations generated by the high-flux laser, changes in the diffusion lengths can be identified. In Fig. 7, two spatial carrier profiles, generated by a high-flux laser beam, at two different positions $x$, are plotted. The grey carrier concentration profile is resulting from a high-flux laser line positioned in $x = 0$ but the carrier concentration is not high enough to reach the transition region before the PL detection limit is reached. The slope of the profile is, however, completely uniform demonstrating that the diffusion length in this region is uniform. This proves that above a certain $\Delta n_{\text{lim}}$, the B-O defect generation rate is fully independent of the carrier concentration and that the assumption of a lifetime plateau, inside the degraded region, used in section III is correct.

The black carrier concentration profile in Fig. 7, is generated by moving the high-flux laser beam closer to the transition region. This time the carrier concentration is high enough to reach the transition region before the PL detection limit is reached. The detection of the direct PL signal in the transition region at high injection must be performed very quickly in order to not degrade the transition region, which is highly sensitive to degradation by excess carriers. Even though we use an exposure time of 15 seconds we are still able to observe a change in the slope of the profile. The $x$-position of the change in the slope is determined by fitting two exponentially decaying curves to the right side of the decaying carrier profile. The intersection between these two curves corresponds well to the transi-
tion region of the measured lifetime profile, where the lifetime changes from being degraded to unaf-
fected by the carrier concentration, given by the dashed curve. This finding shows that there exists a
carrier threshold value at which the generation rate of B-O defects is no longer limited by carrier ac-
cess.

FIG. 7 Figure showing measured minority carrier density and lifetime after 10 min of low-flux laser beam ex-
pposure in $x = 0$ for a given sample. The grey curve corresponds to the carrier density profile when the high-
flux laser beam is positioned in $x = 0$ and the black curve when the beam is positioned at $x = 0.2$ cm. Two in-
dividual exponential fits are shown on the $x = 0.2$ mm curve. The intersection of these fits gives the x position
of where the diffusion length of the excess carriers changes. The dashed line is the measured lifetime profile.

B. Comparison of $\Delta n_{lim}$ for different $N_{t, rapid}$

The carrier concentration limit, $\Delta n_{lim}$ is extracted from the simulated carrier profiles, according to
the method described in section III, for a set of six samples with different doping and normalized de-
fect concentration, $N_{t}^*$. In this study the $\Delta n_{lim}$ values have been determined after exposing the samples
for 10 min with a localized laser beam. This time is chosen for different reasons; (i) After 10 min a
lifetime groove with a well-defined shape is formed, suitable to test whether the slope of the spatial
carrier profile will change in the transition from the degraded to the unaffected region. (ii) At 10 min
small concentrations, $\Delta n < \Delta n_{lim}$, have not yet had time to cause significant degradation in the transit-
ton region, which would complicate the understanding of the spatial lifetime profile. (iii) The life-
time profile after 10 min exposure is a result of the injection history during the whole 10 min ex-
posure sequence. This means that at $t = 0$, when the lifetime of the sample is not yet degraded, the carrier
concentration will be larger for all $x$-positions. A large difference in the diffusion length of the carriers
is, however, only significant during the first few seconds of the exposure and according to previous
work\textsuperscript{7} it was found that the extension of the degraded lifetime plateau does not change considerably over time. It is therefore realistic to employ the determined $\Delta n_{\text{lim}}$ at 10 min as the actual threshold value for the $\Delta n$ dependence of the B-O defect generation rate. (iv) To ease the interpretation of the results it is convenient to look at one degradation mechanism at a time; At 10 min the rapid degradation is complete while the slow degradation is not yet evident. This is demonstrated in Fig. 8. where the normalized defect concentration $N_t^*$ is plotted as a function of time for the samples of interest in this study. Accordingly, we can relate our measurement results to the rapid decay alone.

![Image](image.png)

**FIG. 8** Normalized defect concentration plotted as function of time for the six uncompensated boron-doped Cz-Si samples investigated in this study. The doping concentrations are given in the legend.

Individual $\Delta n_{\text{lim}}$ values determined for each material together with their respective error bars are plotted in Fig. 9. as a function of the normalized defect concentration for the rapid decay, $N_t^{\text{rapid}}$. The black error bars give the uncertainty of $\Delta n_{\text{lim}}$ when the uncertainties of the calibration factor and the QSSPC measurements are taken into account. The red error bars indicate when the measurement with high-flux laser deviates from the simulation results. As can be seen from Fig. 9 the right-most point deviates quite substantially. This sample is the sample corresponding to $[B] = 8.4 \times 10^{11} \text{ cm}^{-3}$ in Fig. 8. In Fig. 8 we see that at 10 min the slow degradation decay has already started for this specific sample. This might explain the deviation in simulation result to high-flux laser measurement. The deviations in the low-flux simulations are, however, expected to deviate much less from the experimental values, as the carrier lifetime is independent of the carrier injection level in this injection region.
Figure showing the minority carrier concentration limit, $\Delta n_{\text{lim}}$, as a function of $N_t^{*\text{rapid}}$. No correlation between $\Delta n_{\text{lim}}$ and $N_t^{*\text{rapid}}$ can be observed. The black error bars give the uncertainty of $\Delta n_{\text{lim}}$ when the uncertainties of the calibration factor and the QSSPC measurements are taken into account. The red error bars indicate when the measurement with high-flux laser deviates from the simulation results.

C. Physical interpretation of $\Delta n_{\text{lim}}$

There is no indication of any obvious correlation between $\Delta n_{\text{lim}}$ and $N_t^{*\text{rapid}}$ in the plot in Fig. 8. This signifies that the amount of carriers required to enable the degradation rate at full speed is independent of how many latent rapid B-O defect centres that occur in the sample. The finding is in agreement with a model where the activation is attributed to the change of the charge-state occurring when the electron quasi-Fermi level shifts across the energy level of the latent centre in its passive state. The determined $\Delta n_{\text{lim}}$ concentration enables us to determine this energy level directly, since the $\Delta n_{\text{lim}}$ concentration corresponds to the quasi-Fermi level at which a further rise in the band gap will not cause any further increase in the defect generation rate. This is, however, only true if the equilibrium constant, $k$, of the charge state reaction $LC + e^{-} \rightleftharpoons LC^{-}$ is sufficiently high ($LC =$ Latent Centre).

If $k$ is $\ll 1$ it is possible that the defect energy level is positioned lower in the band gap and that an increase in the quasi-Fermi energy above this level simply causes an increase in $k$. This scenario cannot, however, be correct according to a measurement by Bothe,\textsuperscript{16} where sub band gap illumination was used. In the first case, photons with energy above 0.50 eV were blocked. In this case, no defects were created. Subsequently, photons with energy above 0.73 eV were blocked and the experiment was repeated. In this case, defect generation was observed. Thus, the energy level must lie in between these limits. The determined $\Delta n_{\text{lim}}$ concentration therefore enables us to determine the quasi-Fermi level responsible for the charge-state change of the latent defect centre directly.
The arithmetic average $\Delta n_{\text{lim}}$ concentration from the measurement series shown in Fig. 9 is given by the dashed line and yields $\Delta n_{\text{lim}} = 1.84 \pm 0.9 \times 10^{11} \text{ cm}^{-3}$. The quasi-Fermi energy from this concentration gives $E_{\text{lat}} = E_V + (635\pm18) \text{ meV}$ determined by Eq. 3.\(^{17}\)

\[
E_f^n - E_i = kT \times \ln \frac{n}{n_i}
\]  

(3)

Where $E_f^n$ is the quasi-Fermi energy level for the electrons in this case equivalent to $E_{\text{lat}}$, $E_i$ is the intrinsic Fermi energy (eV), $k$ is the Boltzmann’s constant, $T$ is the temperature, $n$ is the electron concentration in this case equivalent to $\Delta n_{\text{lim}}$, and $n_i$ is the intrinsic concentration of electrons. Previously Bothe et al.\(^2\) have determined this level by a fundamentally different route, including temperature dependence on forward bias induced open circuit voltage degradation and PC1D simulations. They calculated the level to be $E_{\text{int}} = E_V + (635\pm20) \text{ meV}$ which is in good agreement with our result. The activation of the fast recombination centre is thus after all likelihood attributed to the change of the charge-state occurring when the electron quasi-Fermi level shifts across an energy level corresponding to the reported value. A simple recharge cannot, however, explain the complete transition from a latent to recombinative defect centre. The total supply of electrons over time under illumination is much larger than the concentration of B-O defects which is assumed to be in the order of $1 \times 10^{11} \text{ cm}^{-3}$.\(^3\)\(^,\)\(^5\) Accordingly, if a recharging effect was the final step in the generation of recombinative centres, the hypothetical reaction $\text{LC} + e^- \leftrightarrow \text{RC}$ ($\text{RC}$ = Recombinative Centre) should be complete almost immediately. From Fig. 8. we see that this is not the case. The normalized rapid defect concentration does not saturate until approximately 200-300 s. Thus, the charging of the latent defects cannot be the last step before a final recombinative complex is in place. Another rate limiting event, when $\Delta n > \Delta n_{\text{lim}}$, must be occurring subsequently after the recharging. This is in correspondence to the model proposed by Voronkov et al.\(^8\)
V. CONCLUSION

We have developed a new method based on photoluminescence imaging to directly monitor the excess carrier density during light induced degradation. The laser used for light soaking is continuously exposing the sample to light, generating a spatially varying profile of activated B-O defects. By this method we show experimentally that above a certain minority carrier concentration limit, $\Delta n_{\text{lim}}$, the B-O defect generation rate is fully independent of the minority carrier concentration. By simulation we are able to determine the carrier concentration where the generation rate of the B-O defect saturates, $\Delta n_{\text{lim}} = 1.84 \pm 0.9 \times 10^{11} \text{ cm}^{-3}$. Our results are consistent with a model where the role of the excess carriers during the rapid decay is to change the charge state of the defects by shifting the electron quasi-Fermi level across the energy level of the centre in its passive state. The energy level of the passive state of the latent defect centre is determined and yields $E_{\text{lat}} = E_V + (635\pm18) \text{ meV}$.

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