Naval Research Laboratory

Washington, DC 20375-5320

NRL/MR/6810--06-8980

A Theory of Radiation Effects in Cellular Devices

B.D. WEAVER

Solid State Devices Branch
Electronics Science and Technology Division

August 14, 2006

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1. REPORT DATE *(DD-MM-YYYY)*	**2. REPORT TYPE**	**3. DATES COVERED** *(From - To)*
14-08-2006	Memorandum Report	July 12, 2005 - July 12, 2006

4. TITLE AND SUBTITLE

A Theory of Radiation Effects in Cellular Devices

5a. CONTRACT NUMBER

5b. GRANT NUMBER

5c. PROGRAM ELEMENT NUMBER

6. AUTHOR(S)

B.D. Weaver

5d. PROJECT NUMBER

5e. TASK NUMBER

5f. WORK UNIT NUMBER
68-8746-06

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)

Naval Research Laboratory, Code 6818
4555 Overlook Avenue, SW
Washington, DC 20375-5320

8. PERFORMING ORGANIZATION REPORT NUMBER

NRL/MR/6810--06-8980

9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES)

Office of Naval Research
One Liberty Center
875 North Randolph St.
Arlington, VA 22203-1995

10. SPONSOR / MONITOR'S ACRONYM(S)

ONR

11. SPONSOR / MONITOR'S REPORT NUMBER(S)

12. DISTRIBUTION / AVAILABILITY STATEMENT

Approved for public release; distribution is unlimited.

13. SUPPLEMENTARY NOTES

14. ABSTRACT

A theory is presented on radiation effects in "cellular" devices comprised of small particles such as nanocrystals or quantum dots. The theory explains the surprising discovery that the photoluminscence of quantum dot devices can be significantly more radiation-tolerant than bulk or quantum well-based photodiodes.

15. SUBJECT TERMS
Radiation effects
Nanocrystals

16. SECURITY CLASSIFICATION OF:			**17. LIMITATION OF ABSTRACT**	**18. NUMBER OF PAGES**	**19a. NAME OF RESPONSIBLE PERSON**
a. REPORT	**b. ABSTRACT**	**c. THIS PAGE**			B.D. Weaver
Unclassified	Unclassified	Unclassified	UL	16	**19b. TELEPHONE NUMBER** *(include area code)* (202) 767-5456

A Theory of Radiation Effects in Cellular Devices

B.D. Weaver

Naval Research Laboratory, Code 6818

Washington, DC 20375

Abstract - A theory is presented on radiation effects in "cellular" devices comprised of small particles such as nanocrystals or quantum dots. The theory explains the surprising discovery that the photoluminscence of quantum dot devices can be significantly more radiation-tolerant than bulk or quantum well-based photodiodes.

Manuscript approved June 29, 2006.

I. INTRODUCTION

In the field of radiation effects, reduced dimension has been associated with increased sensitivity to radiation damage. High temperature superconductors (HTSs), for example, are several orders of magnitude more sensitive to displacement damage than their low temperature counterparts because Cooper pairs in HTSs are constrained to lie in two-dimensional (2-D) Cu-O planes, and when paired carriers scatter from radiation-induced defects they can be ejected from the planes and hence from the superconducting state with high efficiency.[1] In contrast, scattering from radiation-induced defects in isotropic low temperature superconductors does not lead to the decoupling of electron pairs because all directions are essentially equivalent.[2] Similarly, in high electron mobility transistors (HEMTs) carriers travel from source to drain via a 2-D electron gas (2DEG). Although radiation-induced defects outside the 2DEG do affect the operating parameters of HEMTs, the main displacement damage effect occurs in the 2DEG region, where defects can easily scatter carriers out of the transport state.[3] Given these examples, it is surprising to learn that in irradiated bulk (3-D), quantum well (2-D) and quasi-zero dimensional quantum dot photodiodes radiation tolerance increases as dimensionality is reduced. If this effect could be exploited, the radiation tolerance of numerous device types susceptible to radiation damage could be improved. Unfortunately, although a qualitative explanation for this phenomenon exists, there is as yet no quantitative theory to describe how some low-dimensional devices have high radiation tolerance. Following a brief review of the available experimental results, such a theory is presented here.

II. EXPERIMENTAL RESULTS

Results of displacement damage experiments have been reported for a number of bulk, quantum well (QW) and quantum dot (QD) devices, including $In_xGa_{1-x}As$/ GaAs and GaAs/InAs QWs,[4-12] and GaAs/InAs, $In_xGa_{1-x}As$/GaAs and InAlAs/AlGaAs QDs.[4-11] Generally, the

optoelectronic performance of devices decreases with increasing particle fluence Φ. For example, GaAs *pn* junction bulk diodes are about 30 times more sensitive to displacement damage than GaAs/AlGaAs QW diodes;[12] and QW devices are 10-100 times more sensitive than QD devices.[5,7,8-12]

The effect of radiation damage on the photoluminescence P of bulk, QW and QD devices is illustrated in Table 1 for various materials systems, incident particles and particle energies. Normalized values of the change in P, $\Delta P/P_o$ are shown in column 7. To obtain a basis for comparing different values of $\Delta P/P_o$ the actual particle fluences (column 5) were converted to equivalent fluences Φ_{eq} of 2 MeV protons (column 6) by using a standard conversion relation involving the nonionizing energy loss (NIEL, the density-normalized rate at which the incident particle energy is converted to atomic displacements[13]). Although the equivalent fluences listed in Table 1 are not identical, it is clear that in terms of photoluminescence QD devices are significantly more disorder-tolerant than QW or bulk devices. Similar trends have been observed for absorption coefficients and electroluminescence in QDs and QWs.

The currently-accepted explanation for increasing radiation tolerance with decreasing dimensionality is that carriers in bulk devices can move in three dimensions, and so are susceptible to scattering, trapping and recombination at all radiation-induced defects within a given volume. Carriers in QWs are confined to two dimensions and so are susceptible only to those defects lying on a disk, while carriers in QDs are dimensionally constrained and so are not susceptible to radiation-induced defects outside their own locales. Within this scenario, carriers in QDs remain unaffected by radiation damage in other "cells".

The theory presented below on radiation damage effects in charge-localized systems bears a resemblance to Target theory, which was developed in the 1940s to describe survival curves obtained by irradiating living tissue with ionizing radiation.[14] However, the present Cell

theory allows for a distribution of cell sizes and responses, while in standard Target theory all unirradiated cells are assumed to be the same. Also, Cell theory addresses displacement damage while Target theory was developed to describe ionization damage.

III. THEORY

We define a *cell* as one of many initially similar (but not necessarily identical) components which contribute piecewise to the function of a device, so that under the application of an external stimulus cell i exhibits a response r_i in such a way that the device a response R is

$$R = \sum_{i=1}^{D} r_i \quad , \tag{1}$$

where the number of cells, D, is expected to be large. For instance, a QD cellular device having an area A = 50x50 μm^2 and an areal dot density $\rho_c = 10^{10}$/cm^2 (a commonly encountered value) would contain 2.5x10^5 dots. For devices having more than one layer of cells it is convenient to rewrite Eq. (1) as

$$R = \sum_{l=1}^{L} \sum_{i=1}^{D_l} r_{il} \quad , \tag{2}$$

where L is the number of layers, D_l is the number of cells in layer l and r_{il} is the response of cell i in layer l.

The problem at hand is to determine how Eq. (2) evolves as particle irradiation creates displacement damage in a device. Imposing a few initial conditions is helpful in this regard (but not necessary; the conditions can be relaxed in a more general formalism). First, incident particles are required to have sufficient energy that they pass through all cells without significant energy loss, thus creating a uniform damage profile. Second, incident angles are not allowed to be so steep that an incident particle is likely to strike two cells in the same layer. Third, layers in

multilayer devices must be sufficiently similar in composition that an imaginary average layer can be defined in such a way that the individual real layers are distributed smoothly if not symmetrically about a mean. Then, the term r_{il} in Eq. (2) can be rewritten as

$$r_{il} = r_{ia} + \Delta r_{il} \quad , \tag{3}$$

where r_{ia} is the the value of r_i in the average layer and Δr_{il} is the deviation of the real r_{il} from that average. Substituting Eq. (3) into Eq. (2) gives

$$R = \sum_{l=1}^{L} \sum_{i=1}^{D_l} (r_{ia} + \Delta r_{il}) = L \sum_{i=1}^{D} r_{ia} + \sum_{l=1}^{L} \sum_{i=1}^{D} \Delta r_{il} \quad . \tag{4}$$

Since the individual layer structures are required to be smoothly distributed about a mean, the double summation over the deviations Δr_{il} tends to zero because cumulative deviations tend to cancel for large D. Hence,

$$R = L \sum_{i=1}^{D} r_{ia} \quad . \tag{5}$$

The collapse of the summation over l indicates that the effect of irradiation need only be studied for a single layer, albeit an imaginary one, then applied to all layers. For convenience, then, r_{ia} is subsequently shortened to r_i, and it is assumed without loss of generality that $L = 1$.

Because by definition all cells must be initially similar, their initial responses r_i should map a smooth or at least piecewise continuous distribution about a mean value $r_c = R/D$. Similarly, the cross sectional areas A_i of cells as seen by an incident particle should be distributed about a mean value A_c. Then, if the *device* area is A, the areal density of cells ρ_c is

$$\rho_c = D/A \quad , \tag{6}$$

and the coverage ζ of cells is

$$\zeta \quad = \frac{1}{A}\sum_{i=1}^{D} A_i = \frac{1}{A}\sum_{i=1}^{D}(A_c + \Delta A_i) \quad , \tag{7}$$

where ΔA_i is the deviation of A_i from A_c. But due to the distribution about A_c the summation over ΔA_i tends to zero as well, so Eq. (7) reduces to

$$\zeta = D\,A_c\,/A = A_c\,\rho_c \ . \tag{8}$$

If the $2.5\text{x}10^5$ QDs of the above example have diameters near 25 nm (again, a common value), they cover about 123 μm^2 of the 2500 μm^2 device and result in a coverage $\zeta \approx 0.05$.

Now, if an energetic particle impinges on a device, the probability that it strikes any cell is ζ and the probability that it strikes a specific cell is ζ/D. Thus for a particle fluence Φ the total number of incident particles is $N = \Phi A$ and the most likely number of cells hit is $n = \Phi A\zeta$. As an initial exercise it is assumed that once a cell is hit it ceases to function (i.e., $r_i \rightarrow 0$). It is also initially assumed that $\Phi A\zeta << D$, which means that the probability of a cell being hit more than once is negligible. Then, if the initial device response is R_o, the response R_n after $n = \Phi A\zeta$ cells have been hit is

$$R_n = R_o - \sum_{j=1}^{n}(r_c - \Delta r_j) = R_o - n\,r_c - \sum_{j=1}^{n}\Delta r_j \ . \tag{9}$$

The term Δr_j represents the initial deviation of the response of cell j from the average. In experiments, values of N are determined by the ion beam current, the beam area and the exposure time, and are generally large. For the QDs and QWs of Table 1 the maximum equivalent fluences range from about 10^{13}- 10^{14} particles/cm^2. Assuming a minimum fluence of 10^{-3} times the maximum, the previously-mentioned 2500 μm^2 device would receive a minimum of between 10^5 and 10^6 hits, resulting in a minimum of about n = 5000-50000 cells hit. Because n is large the summation over Δr_j in Eq. (9) also tends to zero. Thus, substituting R_o/D for r_c and $\Phi A\zeta$ for n yields

$$R(\Phi) = R_o\left(1 - \frac{\Phi A\zeta}{D}\right) \quad . \tag{10}$$

If the second assumption above becomes invalid and $\Phi A\zeta$ is not much less than D, allowance must be made for the possibility that a cell is hit more than once. Then, it is useful to note that the probability $p(0)$ that a given cell remains *un*damaged after n cells have been hit is

$$p(0) = (1 - 1/D)^n \quad . \tag{11}$$

In this equation, $1/D$ is the probability that a specific cell is hit by one of the n incident particles to strike a cell.

After N particles have hit the device and n of those have hit cells, the remaining number of undamaged cells is $Dp(0)$ and the overall device response is $r_cDp(0)$ (assuming again that the deviations Δr_i cancel upon summation). Substituting for r_c and $p(0)$ then yields

$$R_n = R(\Phi) = R_o\,(1 - 1/D)^{\Phi A\zeta} \quad . \tag{12}$$

Expanding Eq. (12) to lowest order in $1/D$ (which for the above example is $4\text{x}10^{-6}$) yields Eq. (10), as expected when $\Phi A\zeta$ is small. Also, because D is large, Eq. (12) can be approximated by

$$R(\Phi) = R_o\,\mathrm{e}^{-\Phi A\zeta/D} \tag{13}$$

for all but the highest fluences. Eqs. (12) and (13) complete the initial exercise.

An equation analogous to Eq. (13) is obtained when the so-called "single-hit" model is used in Target theory to describe the survival curves of living tissue exposed to ionizing radiation. In Target theory the next step in refining the equivalent of Eq. (13) is to switch models from the single-hit to the more elaborate "multi-target-single-hit" or "single-target-multi-hit" models. The multi-target model is used to distinguish between various components of a cell (e.g., cell wall, nucleus) while the multi-hit model is used to describe cells which die after undergoing a fixed number of ionization events. These models have been used to describe ionization effects in both tissue and electronic memory cells,[15] but are inappropriate for describing displacement damage effects in QDs and nanocrystals because a method is required for describing the gradual degradation of cells. Thus, rather than using the Target theory models, we introduce an effectiveness parameter E to describe the particle-induced damage done to a cell. Without loss of generality, $E = 0$ can be chosen to represent an impact that does not alter a cell's response and $E = 1$ can be chosen to represent cell destruction.

The value of E for displacement damage is expected to vary among cells for several reasons. First, an incident particle can impact a cell at a variety of locations and thereby cause different amounts of damage. Second, the stochastic nature of ion impacts results in various

amounts of damage even for impacts at otherwise identical sites. As a result, values of E will range from near zero for low energy, low mass particles striking a cell's perimeter to a certain particle- and material-dependent maximum, E_{max}. (Values of E_{max} will probably depend on the incident particle's mass and energy via the nonionizing energy loss.) As mentioned, even the smallest typical fluences result in large numbers of cells being hit, which means that information about the distribution of the values of E is likely to be difficult to extract from experimental data. Thus, only an overall average effectiveness parameter is expected to be evident. For convenience, this average value is called E.

Within the above construct the function of a cell after being hit once is $r_i(1 - E)$. Therefore, after being hit m times, a cell's function is on average

$$r_{m,i} = r_i (1 - E)^m \quad . \tag{14}$$

If the particle fluence is sufficiently small that cells are unlikely to be hit more than once, then the most likely number of cells *not* hit is $D - \Phi A\zeta$. It is straightforward to use Eq. (14) and a summation process similar to that of Eq. (9) to show that

$$R(\Phi) = R_o\left(1 - \frac{\Phi A E \zeta}{D}\right) \quad . \tag{15}$$

It can also be shown that if there is a likelihood of multiple hits on a given cell, Eq. (15) becomes

$$R(\Phi) = R_o (1 - 1/D)^{\Phi A E \zeta} \tag{16}$$

through a sequence similar to the replacement of Eq. (10) by Eqs. (12) and (13).

Having now defined the necessary parameters and derived equations applicable to several special cases, a more comprehensive development is undertaken. After $n = \Phi A\zeta$ cells have been hit, the device funtion can always be written as

$$R_n = \sum_{i=0}^{n} N_i < r_i > \quad , \tag{17}$$

where N_i is the number of cells hit i times and $<r_i>$ is the average function of cells hit i times. The probability that a given cell has not been hit is given by Eq. (11). The probability $p(j)$ that a cell has been hit j and only j times can be shown to be

$$p(j) = \left(1 - \frac{1}{D}\right)^{n-j} \left(\frac{1}{D}\right)^{j} \frac{n!}{(n-j)!\, j!} \quad . \tag{18}$$

The first and second terms on the right hand side of Eq. (18) represent the probability that a cell is not hit (n-j) of the n times but *is* hit j times. The third term represents the number of ways the hit/no-hit sequence can occur. Summing $p(j)$ over all j from 0 to n shows that it is normalized to unity. The number of cells hit j and only j times is then $Dp(j)$.

Rather than now applying the mean value theorem as was done following Eqs. (7) and (9), a related statistical principle is invoked: If an arbitrary subset is selected at random from a global distribution, the best initial estimate of the subset's average values, standard deviations, and so on is that these values are the same as those of the global distribution. For example, the average initial function of any randomly chosen subset of cells should be $r_c = R_o/D$. The *actual* function of a specific subset depends on how many times the cells in it have been hit. Using Eq. (14) to describe a group of cells hit j times and Eq. (18) for $p(j)$ allows Eq. (17) to be written as

$$R_n = R_o \sum_{j=0}^{n} (1-E)^j \left(1-\frac{1}{D}\right)^{n-j} \left(\frac{1}{D}\right)^j \frac{n!}{(n-j)!\, j!} \quad . \tag{19}$$

Extracting a factor of $(1 - 1/D)^n$ from the summation and introducing the variable $y = (1\text{-}E)/(D\text{-}1)$ allows Eq. (19) to be rewritten as

$$R_n = R_o \left(1-\frac{1}{D}\right)^n \sum_{j=0}^{n} \frac{y^j n!}{(n-j)!\, j!} \quad . \tag{20}$$

Since E must be between zero and one, and since D is large, the value of y will be much less than one. Hence, y^j vanishes as $j \rightarrow n$ and Eq. (20) is little changed if the summation is performed from 0 to ∞ rather than from 0 to n. In that case, the value of the summation is simply $(1 + y)^n$, so

$$R_n = R_o \left[\left(1-\frac{1}{D}\right)(1+y)\right]^n = R_o \left[1-\frac{E}{D}\right]^{\Phi A \varsigma} . \tag{21}$$

Eq. (21) reduces to the trivial case for $E = 0$ and to Eq. (12) for $E = 1$. In most practical cases Eq. (21) can be approximated by Eqs. (15) and (16). Using Eq. (6) for ρ_c and Eq. (8) for ζ yields the final equation for the displacement damage response of cellular devices:

$$R_n = R_o \left[1-\frac{E}{D}\right]^{\Phi D A_c} \approx R_o e^{-\Phi A_c E} \quad . \tag{22}$$

IV. CONCLUSION

The response of bulk optoelectronic devices to displacement damage commonly depends on parameters such as carrier diffusion lengths, recombination constants, donor and acceptor

concentrations, defect trap levels *etc*. For cellular devices, however, the predicted response depends almost exclusively on the particle fluence, the cell area and the damage effectiveness parameter. Thus cellular devices do not respond to radiation damage in the same way that bulk and (probably to a lesser extent) QW devices do.

The Cell theory presented here for describing displacement damage effects in quasi-zero dimensional devices such as QD photodiodes represents the first time that charge localization has been built into a model of radiation effects. This theory can be used to predict that the radiation tolerance of cellular devices can be improved simply by employing smaller cells. Finally, if Eq. (22) is validated by experiments, Cell theory could have a significant impact on the way future radiation-hard devices are designed

The author wishes to thank E.A. Burke and G.P. Summers for helpful comments. This work was sponsored in part by the Office of Naval Research.

Material	**Device**	**Particle**	***NIEL***	**Φ**	**Φ_{eq}**	**$\Delta P/P_o$**	**Ref.**
GaAs *pn*	Bulk	50 MeV H^+	3.45E-3	5E10	5.56E9	0.87	12
GaAs/InGaAs	QW	1.5 MeV H^+	5E-2	3.4E9	5.66E9	~ 0 *	10
GaAs/InGaAs	QD	1.5 MeV H^+	5E-2	3.4E9	5.66E9	~ 0 *	10
GaAs/InGaAs	QW	2.4 MeV H^+	2.5E-2	1E13	8.1E12	0.9925	8,11
GaAs/InGaAs	QD	2.4 MeV H^+	2.5E-2	2E13	1.6E13	0.71	8,11
GaAs/InGaAs	QW	1.5 MeV H^+	5E-2	6E13	9.7E13	0.997	9,10
GaAs/InGaAs	QD	1.5 MeV H^+	5E-2	6E13	9.7E13	0.4-0.8	9,10
GaAs/InGaAs	QW	1.5 MeV H^+	5E-2	4E13	6.4E13	0.995	5
GaAs/InGaAs	QD	1.5 MeV H^+	5E-2	4E13	6.4E13	0.75	5

Table 1. Radiation-induced changes in the normalized photoluminescence, $\Delta P/P_o$, for selected bulk, QW and QD devices. Interpolated data are marked by an asterisk (*). Units of *NIEL* are MeV·cm^2/g. Units of fluence are particles/cm^2. Φ_{eq} is the equivalent fluence of 2 MeV protons as described in the text. Despite variations in Φ_{eq}, values of $\Delta P/P_o$ are consistently smallest for QDs, indicating they are the most radiation-tolerant.

REFERENCES

[1] B.D. Weaver, E.M. Jackson, G.P. Summers and E.A. Burke, Phys. Rev. B **46**, 1134 (1992).

[2] P.W. Anderson, J. Phys. Chem. Sol. **11**, 26 (1959).

[3] B.D. Weaver and E.M. Jackson, Appl. Phys. Lett. **80**, 2791 (2002).

[4] W.V. Schoenfeld, C.-H. Chen, P.M. Petroff and E.L. Hu, Appl. Phys. Lett. **73**, 2935 (1998).

[5] R. Leon, G.M. Swift, B. Magness, W.A. Taylor, Y.S. Tang, K.L. Wang, P. Dowd and Y.H. Zhang, Appl. Phys. Lett. **76**, 2074 (2000).

[6] P.G. Piva, R.D. Goldenberg, I.V. Mitchell, D. Labrie, R. Leon, S. Charbonneau, Z.R. Wasilewski and S. Fafard, Appl. Phys. Lett. **77**, 624 (2000).

[7] N.A. Sobolev, A. Cavaco, M.C. Carmo, M. Grundmann, F. Heinrichsdorff and D. Bimberg, Phys. Stat. Sol. B, **224**, 93 (2001).

[8] C. Ribbat, R. Sellin, M. Grundmann, D. Bimberg, N.A. Sobolev and M.C. Carmo, Electron. Lett. **37**, 174 (2001).

[9] R. Leon, S. Marcinkevičius, J. Siegert, B. Čechavičius, B. Magness, W. Taylor and C. Lobo, IEEE Trans. Nucl. Sci. **49**, 2844 (2002).

[10] S. Marcinkevičius, J. Siegert, R. Leon, B. Čechavičius, B. Magness, W. Taylor and C. Lobo, Phys. Rev. B **66**, 235314 (2002).

[11] F. Guffarth, R. Heitz, M. Geller, C. Kapteyn, H. Born, R. Sellin, A. Hoffmann and D. Bimberg, Appl. Phys. Lett. **82**, 1941 (2003).

[12] B.D. Evans, H.E. Hager and B.W. Hughlock, IEEE Trans. Nucl. Sci. **40**, 1645 (1993).

[13] B.D. Weaver and G.P. Summers, in Studies of High Temperature Superconductors, Vol. 47, A.V. Narlikar, Ed. (Nova Science Publishers, New York, 2003) p. 1.

[14] E.L. Alpen, Radiation Biophysics (Prentice-Hall, NJ, 1990).

[15] P.J. McNulty, L.Z. Scheick, D.R. Roth, M.G. Davis and M.R.S. Tortora, IEEE Trans. Nucl. Sci. **47**, 2237 (2000).