Low energy electron stimulated desorption from DNA films dosed with oxygen

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Abstract

Desorption of anions stimulated by 1–18 eV electron impact on self-assembled monolayer (SAM) films of single DNA strands is measured as a function of film temperature (50–250 K). The SAMs, composed of 10 nucleotides, are dosed with O₂. The OH[−] desorption yields increase markedly with exposure to O₂ at 50 K and are further enhanced upon heating. In contrast, the desorption yields of O[−], attributable to dissociative electron attachment to trapped O₂ molecules decrease with heating. Irradiation of the DNA films prior to the deposition of O₂ shows that this surprising increase in OH[−] desorption, at elevated temperatures, arises from the reaction of O₂ with damaged DNA sites. These results thus appear to be a manifestation of the so-called “oxygen fixation” effect, well known in radiobiology.

INTRODUCTION

Since the discovery that low-energy electrons (LEEs) can cause considerable damage to DNA,¹ numerous attempts have been made to determine the precise mechanisms leading to fragmentation of this molecule.^{2–4} It is now established that, for energies below 15 eV, LEEs localize on the DNA subunits to form transient negative ions (TNIs).^{2–4} These latter can damage DNA by dissociating into a stable anion and one or more radical fragments (i.e., by dissociative electron attachment (DEA)) or by decaying into dissociative electronically excited states.^{2,3} LEEs can also transfer from one DNA subunit to another, particularly from a base to the phosphate group, where they can induce cleavage of the C–O bond (i.e., break a DNA strand).² Since LEEs contain a large fraction of the energy deposited by high-energy particles,^{5,6} these results are relevant to a precise understanding of the mechanisms of the direct effect of DNA damage induced by high-energy radiation and its application to radiotherapy. In fact, it has recently been shown that the modification of these fundamental mechanisms by chemotherapeutic agents considerably increases DNA damage.⁷ Since cancer patients are usually treated with both chemotherapy and radiotherapy,⁸ this finding could have far reaching applications in the clinic. However, before reaching a definitive conclusion, the mechanism of action of LEEs must be investigated under conditions closer to those of the cell.

Our present knowledge of LEE-DNA interactions derives essentially from theoretical calculations⁴ and LEE impact experiments on gaseous DNA sub-units³ and thin films of pure DNA held under ultra-high vacuum.² Although dry film experiments in vacuum were essential to unveil basic mechanisms of damage, they clearly do not correspond to cellular

conditions. Thus, a series of experiments were recently undertaken to add to vacuum DNA other components found in the cell nucleus, so as to systematically investigate modifications of the mechanisms of action of LEEs by molecular constituents of the nucleus. Recently, it has been shown that organic ions⁹ and proteins¹⁰ protect DNA against LEE-induced damage and that the presence of water around DNA modifies the TNI manifold and corresponding decay channels.^{11,12}

O₂ is another molecule present with DNA in the cell nucleus and is known as an efficient radiosensitizer. Studies on the radiosensitization of biological damage by molecular oxygen in living cells began about a century ago and have remained one of the most significant subjects in radiobiology.¹³ Exposure to high concentrations of O₂ causes larger radiation damage in experimental animals and to cellular DNA; e.g., in cultured mammalian cells an O₂ enhancement ratio of approximately 3 is commonly observed upon X-ray irradiation. These toxic effects of O₂ exposure^{14–16} were interpreted as due to the reaction of O₂ with damage sites within DNA, a phenomenon called the “Oxygen-Effect”.^{13,17} More specifically, when plasmid DNA is surrounded by oxygen at atmospheric pressure, the damage to DNA induced by LEEs is found to double.¹⁸ Thus, O₂ considerably sensitises DNA to LEE derived damage. Here, we present results on the mechanism of this sensitization process.

With a recently improved system,¹⁹ we measure the electron stimulated desorption (ESD) of anions from self-assembled monolayers (SAMs) of DNA molecules^{20,21} exposed to monolayer quantities of O₂ at various film temperatures in the range 50–250 K, under ultra-high vacuum (UHV). We inspect any modifications in the DEA and dipolar dissociation (DD) processes as a result of oxygen deposition onto and within SAM-DNA samples. Molecular oxygen is found to react with DNA during LEE irradiation. The decay of TNI associated with these reaction products, via the DEA channel increases ESD of OH[−] from SAM-DNA by an order of magnitude. Measurements as a function of electron fluence, indicate that the reaction involves damage sites of DNA, consistent with the long-held *oxygen fixation hypothesis*, which accounts for radio-sensitization by O₂.¹³ Thus, these results provide insight into the basic mechanisms responsible for DNA radiolysis in the presence of O₂. More generally, studying the temperature dependence of condensed-phase DEA reactions appears to be an efficient new method to detect the formation of reaction products between a small molecule and a larger organic molecule.

EXPERIMENTAL

SAMs of the single-stranded DNA oligonucleotide 5′AAAGGACAAA-3′ phosphothiolated at the 3′ end were prepared by chemisorption onto gold substrates via the terminal thiol. DNA samples with this sequence have previously been used to investigate the effects of film preparation procedure, (alternatively lyophilization and molecular self-assembly onto gold) on the ESD of anions²² and are presently employed to study the radio-sensitization effects of Pt-based chemotherapeutic drugs.²³ Previous measurements of the ESD yields of anions from lyophilized, short oligonucleotide samples,²⁴ show that the desorption signals H[−], O[−] and OH[−] at low electron energies are largely insensitive to base identity and DNA length, at least with respect to the shape and energies of resonant features. We thus expect results

obtained with the present samples to be representative of short, single-stranded DNA in general.

The gold substrates were cleaned by multiple exposures to UV/ozone and rinsing with ethanol and doubly-distilled deionized water. Each SAM-DNA film ($\sim 0.8 \text{ cm}^2$) was prepared from a pure water solution of the purified oligonucleotide as previously described.²⁵ With this preparation,²⁵ the counter ion on the negative oxygen atom of the phosphate group is a proton²⁶ and the molecules have a tendency to stand perpendicular to the surface and parallel to each other.^{20,21} X-ray photoelectron spectra were recorded to verify the formation of the SAM-DNA monolayers on gold substrates.²⁵ The molecular surface number density of the film was deduced to be $1.9 \times 10^{13} \text{ cm}^{-2}$ by measuring the radiation emitted from SAM-DNA labelled with radioactive ^{32}P at the end of the chain facing vacuum (i.e., the 5' end). Considering this density and the geometry of the films and taking $20 \text{ \AA}^2$ as the surface of the end group facing vacuum (this assumes a single DNA strand has a diameter of $5 \text{ \AA}$), we estimate that the ratio of the area of empty space to that occupied by the oligonucleotide at the film-vacuum interface is a factor 25. *Although crude, this estimate clearly shows that O_2 molecules, leaked in front of the sample, can easily migrate into the SAM and the grooves of DNA.*

The ESD measurements were performed using a high sensitivity time-of-flight (TOF) mass analyzer. This system, which has been previously described in detail,¹⁹ is housed in an UHV system reaching a base pressure of 5×10^{-10} Torr. Recently, a custom load-lock has been coupled to the TOF mass analyzer to allow samples, prepared under ambient conditions, to be introduced into UHV.¹⁹ Once transferred into the analysis chamber, the temperature of the samples can be controlled between 10 and 300 K with a closed-cycle He cryostat and resistive heating elements. Samples were degassed within the load-lock chamber for at least 4 hours prior to their transfer into the main chamber, where they were bombarded with a pulsed electron beam. Anions were analyzed by the TOF mass analyzer. The electron energy scale was calibrated within $\pm 0.3 \text{ eV}$ from the onset of electron transmission through the film. By recording the desorbed anion signals as a function of incident electron energy, a *yield function* for each desorbed ion was obtained. Unless otherwise stated, yield functions were recorded between incident electron energies of 0 and 18 eV, using an incident current of $\sim 4 \text{ nA}$, an energy step-size of 0.5 eV (occasionally 0.25 eV), 50,000 electron pulses per point. Each of the presented yield functions were recorded during a single energy scan of duration of 360 s.

The amount of O_2 required to form a monolayer (ML) on the SAM-DNA at 20 K was determined by volumetric dosing within an accuracy of about $\pm 30\%$.²⁷ The surface number density of O_2 molecules in such a film is of the order of 10^{15} cm^{-2} as calculated from the molar volume of liquid O_2 .²⁸ Dosing the SAM-DNA at 50 K to 1 ML, thus corresponds to an exposure of $\sim 10^{15}$ molecules per cm^2 , even if only a fraction of O_2 molecules eventually remains on and within the DNA film. We note that if each of the 1.9×10^{13} 10-mer oligonucleotides (present within an area of 1 cm^2) is considered as containing 30 sub-units (i.e., one base, sugar, and phosphate per nucleotide) the an exposure of 1 ML of O_2 (10^{15} molecules/ cm^2) corresponds to an initial deposition of approximately two oxygen molecules per subunit. In this work, O_2 exposure is expressed in equivalent MLs of O_2 at 20 K.

RESULTS AND DISCUSSION

The ESD of H^- (1 amu), CH_2^- (14 amu), $\text{CH}_3^-/\text{NH}_-$ (15 amu), O^- (16 amu), OH^- (17 amu), and CN^- (26 amu) were measured from the pure SAM-DNA samples. The results are essentially the same as those previously recorded.^{19,22,24,26} With the exception of O^- and OH^- , the yield functions of these anions did not change significantly with film exposure to 0.75 to 3.5 ML of O_2 at 50 K. Since H^- , CH_2^- , and CH_3^- can only arise from the bases and sugar ring and NH^- and CN^- only from the bases, either adsorption of O_2 on these subunits did not change anion yields or O_2 adsorbs only on the phosphate group. Below 12 eV, the phosphate group is thought to be responsible for O^- and OH^- ESD via DEA to pure DNA;² i.e.,

or

where the DNA phosphate charge is neutralized by protonation as in the present experiment.

The $^{16}\text{O}^-$ and $^{16}\text{OH}^-$ ESD yield functions for pure SAM-DNA samples held at 165 K are shown in Fig. 1 as the solid black lines in panels (a) and (b), respectively. The data are consistent with those published earlier,^{19,22,26} where the structures peaking at 8.5 and 7.5 eV in the respective O^- and OH^- yield functions are due to DEA to DNA and the linearly increasing signal above ~12 eV to DD.¹⁹ The threshold energies for desorption of O^- and OH^- are estimated as being 3.9 and 2.9 eV, respectively. The vertical arrow in the figure is positioned at the energy of the dominant O^- DEA peak of gaseous O_2 ,²⁹ which coincidentally, is also that for O_2 condensed on benzene at low coverage.³⁰ When, at temperatures from 165 K down to 55 K, the SAM-DNA films are exposed to the equivalent of 0.75 ML of O_2 , the ESD yield of both O^- and OH^- anions increases considerably across all incident energies. At 55 K, the O^- signal in panel (a) is seen to increase by about an order of magnitude and the OH^- signal in panel (b) by a factor of six relative to their intensity from the neat DNA. The latter factor easily reaches an order of magnitude by further exposition to O_2 . The O^- signal, however, does not arise completely from DEA to DNA. In fact, the 165 K + O_2 curve in Fig. 1(a) results from the contribution of one peak near 8.5 eV due to DEA to DNA and another one around 6.5 eV, that is strongly reminiscent of DEA to O_2 (i.e., $\text{e}^- + \text{O}_2 \rightarrow \text{O}_2^- (^2\Pi_u) \rightarrow \text{O}^- + \text{O}$). This is more clearly seen in the inset (c) of Fig. 1, which shows in detail the resonance features between 3–12 eV. When the temperature is lowered to 55 K, only the 6.5 eV peak persists, indicating that since much more O_2 adsorbs

on the SAM surface and within the DNA groves, the signal arises essentially from DEA to the added O₂ and blurs any further increase in DEA to DNA (i.e., reaction 1). With O₂ exposure at 165 K, the ESD O⁻ signal from the film is easily visible and its line shape (squares) in Fig. 1(c) can be reproduced by adding a scaled version of the O⁻ data at 55 K from Fig. 1(a) to the signal recorded for the pure SAM-DNA (solid black line). The relative magnitude of the two curves in the inset indicates that additional adsorption of O₂ does not change considerably O⁻ ESD from DNA.

Unlike the O⁻ signal, that of OH⁻ cannot arise from O₂ alone and must necessarily involve the DNA molecule. One mechanism previously identified as linking O⁻ production to OH⁻ desorption is that of reactive ion scattering, first observed in films containing molecular oxygen and various simple alkanes.^{31,32} In this process, O⁻ produced via DEA to O₂, initiates hydrogen abstraction by reactions of the type:

Similar scattering processes have been observed in film mixtures containing O₂ with unsaturated hydrocarbons,³³ aniline³⁴ and tetrahydrofuran,³⁵ among other molecules. As discussed in these earlier works, the production of OH⁻ was attributed to scattering by O⁻ rather than H⁻, due to the great similarity in terms of energetic thresholds and structure, between O⁻ and OH⁻ yield functions and to the absence of OH⁻ production in gas-phase collisions of H⁻ with O₂ (at relevant ion collision energies).

Unless reactive scattering of O⁻ from DEA to O₂ occurs to an appreciable extent, adding O₂ to the DNA sample either increases the OH⁻ arising from reaction 2 or engenders a new source of OH⁻ from DEA to a DNA•O₂ complex¹¹ or a reaction product. In previous investigations of reactive scattering by O⁻ (e.g.,^{31,33,35}), at comparable dosing of O₂ (0.75 ML), the OH⁻ signal arising from reactive scattering was typically 1/50 that of O⁻ from O₂; i.e., much smaller than the ratio of $\sim \frac{1}{2}$ seen in Fig. 1. Moreover, in reactive scattering, the threshold energy of the signal of the final anion product is higher than that of the primary ion.^{31,33,35} Here, the inverse is seen: the energy threshold for OH⁻ desorption occur at 2.9 eV, which 1 eV lower than that for O⁻ desorption. Therefore, the OH⁻ curves in Fig. 1(b) cannot arise primarily from reactive scattering.

When ¹⁸O₂ is used instead of ¹⁶O₂ in these experiments, the ¹⁸O⁻ signal exhibits the same behaviour as that shown in Fig. 1(a), but at 165 K, no peak at 8.5 eV due to DEA to DNA is observed. This result provides further evidence that the ¹⁶O⁻ ESD signals in Fig. 1(a) at 125 and 55 K arise essentially from DEA to the adsorbed O₂. However, when ¹⁸O₂ is used, an ¹⁸OH⁻ signal of about the same magnitude and same line shape as that of ¹⁶OH⁻ in Fig. 1(b) is seen, indicating that oxygen does not simply enhance reaction 2, but participates in the DEA process. The latter is therefore expected to arise from a site, where O₂ is strongly bound to a DNA subunit. Complexes of H₂O with DNA have previously been identified via the appearance of new structures in the ESD yield of H⁻ from oligonucleotide films when they are hydrated at cryogenic temperatures.¹¹ Later theoretical and experimental work¹² suggests that the presence of structural water in the major grooves of DNA strongly perturbs

electron diffraction within DNA and that H₂O:DNA states involving core-excited resonances based on those of H₂O likely contribute to LEE induced strand break damage.¹²

If indeed the ¹⁸OH⁻ signal arises from ¹⁸O₂ strongly bound physically or chemically to DNA, it might be expected to exhibit a quite different behaviour as a function of the sample temperature than that of the ¹⁸O⁻ signal. Fig. 2 displays the magnitude of the ¹⁸O⁻ and ¹⁸OH⁻ yields integrated from 2 to 18 eV under different conditions. In these experiments, the samples were first cooled to 50 K and at time $t = 0$, the film was exposed to 0.75 ML of ¹⁸O₂. The presence of O₂ at the surface and its diffusion into the film causes the ¹⁸O⁻ and ¹⁸OH⁻ yields to increase. Subsequent exposure of 0.75 ML of ¹⁸O₂, 10, 20 and 30 min later and 1.25 ML at $t = 40$ min, have a similar effect. At $t = 50$ min, the cryostat is allowed to warm up to 250 K. As the physisorbed ¹⁸O₂ molecules leave the sample with rising temperature, the ¹⁸O⁻ yield decreases and essentially vanishes at 250 K. The ¹⁸OH⁻ yield has the opposite behaviour with increasing temperature: it keeps increasing as the sample temperature is raised from 50 to 250 K. Thus, O₂ molecules responsible for ¹⁸OH⁻ signal, not only do not leave the sample, but on the contrary, their amount increases with rising temperature. These oxygen molecules or species must necessarily in some way be strongly attached to DNA to resist desorption. The binding energy must be at least larger than the mean thermal energy of 23 meV at 250 K (i.e., larger than that of typical van der Waals potentials). Possibly, as the weakly bound ¹⁸O₂ molecules leave their sites with increasing temperature, they further interact with the strong DNA binding sites and stabilize at these sites, thus increasing the number of strongly bound ¹⁸O₂, and consequently, the ¹⁸OH⁻ signal. In other measurements (not shown), the yields of OH⁻ were observed to decrease at temperatures greater than 250 K.

Evidence on the origins of the O₂-DNA binding is obtained in a further sequence of experiments, the results of which are presented in Figures 3 and 4. SAM-DNA films were cooled to 50 K and irradiated with LEEs for pre-determined periods of time. During this irradiation period (termed “*pre-irradiation*”) the ~2 nA incident current was scanned repeatedly from 0 to 18 eV incident electron energy (energy step size: 0.1 eV; dwell time: 0.25 s, 45 s per sweep). The number of electrons incident during a pre-irradiation of 5 mins is approximately 40% of that delivered during a single yield function measurement of the type used to obtain Figures 1 and 2. Samples were then exposed to a 3.5 ML equivalent dose of ¹⁸O₂ before their temperature was increased to 150 K. The time delay between pre-irradiation and dosing with O₂ is of the order of 2 min. It takes approximately 15 minutes to increase the temperature to 150 K. Anion yield functions were then measured for ¹⁸O⁻ and ¹⁸OH⁻ at 150 K; examples are presented in Fig. 3. It can be seen that the *desorption yields of O⁻ and OH⁻ from films that have not been pre-exposed to LEEs are essentially zero and that these yields increase in intensity with the duration of electron exposure*. Specifically, it is apparent from Fig. 4 that the incident-energy integrated yields (0–18 eV) of these two anions, increase linearly with the length of the pre-irradiation period. We note also, that the yield functions for ¹⁸O⁻ and ¹⁸OH⁻ desorption from irradiated films are very similar to those seen from ¹⁶O₂ dosed films (Fig. 1). *It thus appears that the stabilization of O₂ and the persistence of both the O⁻ and OH⁻ signals to elevated temperatures requires pre-irradiation and so most probably, involves the binding of O₂ to a DNA damaged site.*

Reactions between O_2 and DNA underlie the “Oxygen Fixation Hypothesis”, which has been advanced to explain the radio-sensitization properties of O_2 .¹³ Free radicals “R $\cdot$ ” are expected to be generated in DNA by the interactions of ionizing radiation. In the cell, and in the absence of O_2 , such damage can be reduced by reactions with SH groups.³⁶ However, the formation of ROO $\cdot$, organic peroxides by reaction of free radicals with O_2 , is an irreversible process which is said to “fix” or make permanent the R $\cdot$ lesions. It has been demonstrated in living systems that for fixation to occur, O_2 must be present or administered within several 10^{-3} s of exposure to ionising radiation.¹³

The appearance at high temperature of the $^{18}O^-$ signal in Figures 3 and 4, but only after pre-irradiation of DNA, suggests that the $^{16}O^-$ desorption seen in Fig. 1 is not due to the diffusive trapping of O_2 within the DNA layer, but depends on the existence of electron induced damage in the film and likely derives from DEA to organic peroxides rather than to intact O_2 molecules as earlier postulated. Significantly, peroxy radicals are reported to abstract hydrogen atoms from thymine bases and from deoxyribose to form ROOH moieties.^{37,38} Such damage sites would serve as plausible sources for OH $^-$ desorption under electron impact. Moreover, since it has been observed that radiation-induced radicals in DNA and related molecules, that form and trap at cryogenic temperatures, can combine upon heating,³⁹ it is interesting to speculate that the decrease in O^- signal and increase in OH $^-$ desorption observed with increasing film temperature in Fig. 2, may correspond to the thermally stimulated conversion of ROO $\cdot$ lesions into ROOH.

The data obtained in Figures 3 and 4 were obtained using electrons having energies between 0 and 18 eV. Measurements are proceeding to determine the energetic threshold of the electron damage process which should better identify the fundamental mechanism (e.g., DEA, ionization, electronic excitation, etc.) responsible for the formation of R $\cdot$ damage.

CONCLUSION

These data reveal that under UHV conditions, O_2 forms adducts with DNA, by reacting with electron induced DNA radicals. These adducts are very likely the peroxy radical ROO $\cdot$ and ROOH. Even at low temperatures (e.g., 50 K) electron attachment to these adducts contribute to the ESD yield of O^- and OH $^-$. Increasing film temperature causes unreacted O_2 , to evaporate and diffuse through the film and to increase the number of ROOH adducts. This results in an increase in the ESD yields of OH $^-$ and persistence of the O^- signal. These yields thus appear to arise from a mechanism much like “*Oxygen Fixation*”¹³ which is important for the O_2 radio-sensitization process in radiobiology. The present data therefore indicate that LEEs produced in copious quantities in cells, irradiated with ionizing radiation, may produce a type of damage that can be “fixed” by oxygen.

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FIG. 1.

(a) Yield functions of $^{16}\text{O}^-$ desorption from a self-assembled monolayer of DNA (SAM-DNA) and SAM-DNA dosed with an additional ~ 0.75 ML (equivalent) of $^{16}\text{O}_2$ at each indicated temperature. (b) Yield functions of OH^- desorption recorded under the same conditions as in (a). (c) Details of resonance structure seen in the O^- yield functions recorded at 165 K. The experimental data obtained by adding O_2 (squares) can be reproduced (dashed line) by adding a scaled version of the data obtained at 55 K (dotted line) to the signal recorded for the pure SAM-DNA. The vertical arrow denotes the energy of the $^2\Pi_u$ state O_2^- .

FIG. 2.

Time variation of energy-integrated (a) $^{18}\text{O}^-$ and (b) $^{18}\text{OH}^-$ desorption yields from a SAM-DNA sample as it is cooled to 50 K, dosed repeatedly with $^{18}\text{O}_2$, and then heated to 250 K. The Y-axis represents the result of integrating anion yield functions over the range 2–18 eV.

FIG. 3.

Anion yield functions for a) $^{18}\text{O}^-$ and b) $^{18}\text{OH}^-$ from SAM-DNA measured at 150 K after dosing with 3.5 ML of $^{18}\text{O}_2$ at 50 K. Yield functions were obtained with an incident current of ~ 2 nA, an energy step-size of 0.5 eV and 50,000 electrons pulses per point. The SAM-DNA films were irradiated with 0–18 eV electrons (at an incident current of 2 nA) for the indicated periods prior to their exposure to $^{18}\text{O}_2$.

FIG. 4. Variation of the energy integrated yields of a) $^{18}\text{O}^-$ and b) $^{18}\text{OH}^-$ data of Fig. 3 with period of electron pre-irradiation ($0 \text{ eV} < E_i < 18 \text{ eV}$) prior to exposure to $^{18}\text{O}_2$.