Transcription Preferentially Inhibits Nucleotide Excision Repair of the Template DNA Strand in Vitro*

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It has been reported that pyrimidine dimers (pyrimidine\textrightarrow{}pyrimidine) are removed preferentially from actively transcribing genes. Furthermore, the preferential repair is restricted to the transcribed strand of these genes. Currently there is no mechanistic explanation for these phenomena. In this study we investigated the effect of transcription on nucleotide excision repair using defined *Escherichia coli* systems consisting of DNA substrates containing a strong promoter and either (a) a T\textrightarrow{}T at a defined position in the nontranscribed or transcribed strand or (b) photoproducts randomly distributed in both strands, as well as transcription and nucleotide excision repair enzymes. While a T\textrightarrow{}T in the nontranscribed strand had no effect on transcription, a photodimer in the transcribed strand blocked transcription causing RNA polymerase to stall at the T\textrightarrow{}T site. This stalled elongation complex inhibited the excision of the photodimer by (A)BC excinuclease resulting in a net effect of preferential repair of the nontranscribed strand in a mixture containing both substrates. Similarly, when we conducted transcription/repair experiments with a superhelical plasmid no enhanced repair of the transcribed gene was observed compared to nontranscribed regions. We conclude that RNA polymerase stalled at a photodimer does not direct the (A)BC excinuclease to the damaged template strand and therefore cannot account for the strand-specific repair observed in vivo.

Bohr *et al.* (1985) discovered that pyrimidine dimers in the dihydrofolate reductase gene of Chinese hamster ovary cells were removed four to five times more rapidly compared to nontranscribed regions of the chromosome. Mellon *et al.* (1987) demonstrated that this enhanced repair was due almost exclusively to the preferential repair of the transcribed strand. More recently Terleth *et al.* (1989) and Mellon and Hanawalt (1989) showed preferential repair in transcribed genes of *S. cerevisiae* and *Escherichia coli*, respectively. The latter authors measured the rate of repair of pyrimidine dimers in the two strands of the lac operon following UV (254 nm) irradiation. They found that when the operon was actively transcribed, the photodimers were removed five to 10 times more rapidly compared to the nontranscribed strand. As a result, no difference was found in the rate of repair of the two strands in uninduced cells.

Initially, preferential repair of transcribed genes was ascribed to a more open conformation of the transcribed regions of chromatin (see Bohr *et al.*, 1988). However, the discovery that preferential repair is limited to the transcribed strand suggests a more specific coupling mechanism between transcription and repair. A simple model for this coupling proposed by Mellon and Hanawalt (1989) is as follows. RNA polymerase transcribes a gene until it reaches a UV photoproduct; transcription then halts, the RNA polymerase makes a stable elongation complex at the transcriptional stopsite, and this complex constitutes a high affinity site for the nucleotide excision repair proteins which remove the photodimer. A precedent for the targeting of UV photoproducts in DNA for repair by a lesion-bound protein exists. Photolyase, which binds to the pyrimidine dimer-containing strand of the duplex (Husain *et al.*, 1987), stimulates the excision of the photodimer by (A)BC excinuclease, the enzyme that initiates nucleotide excision repair in *E. coli* (Sancar *et al.*, 1984; Sancar and Sancar, 1988; Orren and Sancar, 1989).

The above model is an oversimplification of what takes place in an *E. coli* cell as it neglects the topological effects of transcription (Gamper and Hearst, 1982; Tsoo *et al.*, 1989), transcription factors, the effect of transcription-coupled translation and other phenomena as possible contributors in directing repair enzymes to the transcribed strand. Nevertheless, the simple model outlined above is testable in vitro. In this study we have used either a 137-mer duplex containing a single thymine photodimer located on the nontranscribed or the transcribed strand downstream from a tat promoter, or a UV-irradiated superhelical plasmid containing a tat promoter in a coupled transcription/repair system which includes *E. coli* RNAP, (A)BC excinuclease, and DNA polymerase I and ligase when necessary to study the effect of transcription upon repair. We find that under these in vitro conditions, the template strand is repaired less efficiently than the nontranscribed strand.

**MATERIALS AND METHODS**

**Enzymes and Substrates**

*E. coli* RNA polymerase (factor content not less than 60%) and the RNase inhibitor RNasin were purchased from Promega Biotec (Madison, WI), DNA polymerase I, T4 DNA ligase, restriction enzymes, and DNase I were from Bethesda Research Laboratories, and ribonuclease *Bacillus cereus* and ribonuclease T1 were from Pharmacia LKB Biotechnology Inc. The *E. coli* photolyase was purified as described by Sancar *et al.* (1984) and the UV-A, -B, and -C proteins

1 The abbreviations used are: RNAP, RNA polymerase; TRB, transcription-repair buffer; T\textrightarrow{}T or thymine photodimers, thymine-thymine cyclobutane photodimer; HEPES, 4-(2-hydroxyethyl)-1-piperazinethanesulfonic acid; bp, base pair(s); kb, kilobase(s).

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were purified by the method of Thomas et al. (1985).

Two types of substrates were used. One was a 137-mer duplex which contained a tac promoter and a thymine photodimer (T<>T) in either the transcribed or the nontranscribed strand. The 137-mers labeled with ^32P at the 5' end of the strand with T<>T were constructed by ligation of eight individual oligomers shown in Fig. 1 as described previously for a psoralen-adducted substrate (Shi et al., 1985; Thomas et al., 1987). The central 11-base oligomer, labeled with T<>T, was prepared and purified by the method of Banerjee et al. (1988). The second type of substrate for transcription-repair experiments was superhelical pBR3274 plasmid irradiated with 75 J/m^2 of 254-nm UV light from a QuantaCount monochromer (Phototech Technology, Inc., Princeton, NJ). This UV dose produces about 2 UV photoproducts (cyclobutane dimers and 6-4 photoproducts) per kilobase pair (estimated by incision of cyclobutane dimers and assuming 2-8 PCi of [a-32P]dCTP, UvrA to 1.4 nM, UvrB to 16 nM, UvC to 90 nM, PolII to 200 nM, and T4 DNA ligase to 20 units/ml). Under these conditions we found the UvrA and photoproduct concentrations to be limiting factors, i.e., there was an increase in repair synthesis with higher UvrA concentrations or UV doses (data not shown). The standard TRB contained 37 °C, ATP, GTP, and UTP to 200 μM each, dATP, dGTP, and TTP to 40 μM each, dCTP to 10 μM, including 2-8 μCi of [a-32P]dCTP, UvA to 1.4 nM, UvB to 16 nM, UvC to 90 nM, PolII to 200 nM, and T4 DNA ligase to 20 units/ml. Under these conditions we found the UvrA and photoproduct concentrations to be limiting factors, i.e., there was an increase in repair synthesis with higher UvrA concentrations or UV doses (data not shown). The standard TRB contained 37 °C, ATP, GTP, and UTP to 200 μM each, dATP, dGTP, and TTP to 40 μM each, dCTP to 10 μM, including 2-8 μCi of [a-32P]dCTP, UvA to 1.4 nM, UvB to 16 nM, UvC to 90 nM, PolII to 200 nM, and T4 DNA ligase to 20 units/ml. Under these conditions we found the UvrA and photoproduct concentrations to be limiting factors, i.e., there was an increase in repair synthesis with higher UvrA concentrations or UV doses (data not shown). The standard TRB contained 37 °C, ATP, GTP, and UTP to 200 μM each, dATP, dGTP, and TTP to 40 μM each, dCTP to 10 μM, including 2-8 μCi of [a-32P]dCTP, UvA to 1.4 nM, UvB to 16 nM, UvC to 90 nM, PolII to 200 nM, and T4 DNA ligase to 20 units/ml.

**RESULTS**

We used two types of substrate to investigate the effect of transcription on nucleotide excision repair. One of the substrates was a 137-mer duplex containing a strong tac promoter and a single thymine photodimer in either the nontranscribed or the transcribed strand (Fig. 1). Using this substrate we were able to examine effects of template and non-template lesions on transcription and consequences for repair. The second substrate was a UV-irradiated superhelical plasmid which contained the tac promoter from which a gene (uruC) constituting approximately one third of the plasmid is transcribed (Thomas et al., 1985). This substrate was used to examine, by the repair synthesis assay, whether the transcribed sequences were repaired more efficiently than non-transcribed sequences, and whether transcription had any effect on the rate and extent of repair in the transcribed and nontranscribed regions of the plasmid.

**Transcription and Repair of a Linear Substrate Containing a Thymine Dimer at a Defined Site**

**Transcription**—It has been known for a long time that pyrimidine dimers block transcription in both pro- and eucaryotic cells (Sauerbier and Hercules, 1978). We constructed a synthetic DNA fragment ("T" in Fig. 1) with a strong promoter (tac) and a thymine dimer in vivo by irradiating E. coli with UV light and then cleaving the DNA with an endonuclease. Fig. 2A shows the results of transcription experiments conducted using 137-mers with or without a thymine photodimer. With the dimer-free template transcription continues to the end of the fragment generating transcripts 89 or 90 nucleotides long (see below). When the dimer is present in the transcribed strand transcription terminates at the dimer producing a doublet (see below). Within the sensitivity of our assay (which would detect 0.5–1.0% of the radioactivity of the full length transcripts) we conclude that RNA polymerase did not transcribe past the T<>T. To learn where transcription actually begins and ends with respect to the photodimer, the bands in the doublet which represent truncated transcripts were indi-
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fig. 1. Synthetic transcription-repair substrate. The 137-mers contain the tac promoter (−35 and −10 sequences underlined) and a thymine dimer (T<>T) downstream from the transcriptional initiation sites (positions 48 and 49) in the template (T) and the nontemplate (N) strands. The duplexes were constructed by ligating eight oligonucleotides whose boundaries are indicated by triangles. Transcription starts with UTP at positions 48 or 49 and proceeds in the direction indicated by the arrow.

Effect of Transcription on Excision of T<>T from the Template and Nontemplate Strands by (A)BC Excinuclease—The data presented so far indicate that a T<>T in the transcribed strand blocks transcription resulting in a truncated transcript and a stable elongation complex. Does this complex facilitate the excision of T<>T by (A)BC excinuclease? (A)BC excinuclease hydrolyzes primarily the 8th phosphodiester bond 5' to a T<>T (Sancar and Rupp, 1983). The 137-mers N and T in Fig. 1 (dimer in the nontemplate and template strands, respectively) were labeled with 32P at the 5' end of the damaged strand and digested with ABC excinuclease. The end-labeled digestion product of N runs as a 67-mer on a denaturing gel and digestion of T gives a 56-mer (Fig. 4A). The T substrate is digested with somewhat higher efficiency than N, due to a moderate effect of neighboring sequences on enzyme activity. Incision of both substrates is inhibited at high concentrations of RNAP (Fig. 4A, lanes 8 and 9) due to nonspecific binding of RNAP to DNA (Fig. 3, lanes 4 and 5). The reaction mixtures contained RNAP plus all four rNTPs, the incision of the transcribed strand was inhibited to a greater extent than incision of the nontranscribed strand. The optimal RNAP concentration (13 nM) for producing an elongation complex footprint (Fig. 3, lanes 4 and 8) drastically inhibited the incision of the nontranscribed strand. The optimal RNAP concentration (13 nM) for producing an elongation complex footprint (Fig. 3, lanes 4 and 8) drastically inhibited the incision of T<>T from the transcribed strand (Fig. 4A, compare lanes 8 and 18). The selective inhibition of incision of T<>T in the transcribed strand under transcription conditions was also observed when we measured the kinetics of incision (Fig. 4B). Thus, the data shows that RNAP stalled at a T<>T does not stimulate but actually inhibits the excision of the photoproduc by the excision nuclease.

The selective inhibition of incision of the template lesion was unexpected, yet it was consistently observed under conditions in which stimulation of incision by DNA photolyase was readily demonstrated (Fig. 4). Nevertheless we further examined the possible stimulation of incision of the template dimer by transcription. We reasoned that a low concentration of RNA polymerase might have a subtle enhancing effect on the kinetics of incision that we had not detected. However, the results obtained with the T substrate shown in Fig. 5 indicate that 0.7 nM RNAP did not stimulate incision kinetics while photolyase clearly did. Similarly, varied orders of addition of RNAP and (A)BC excinuclease failed to produce stimulation of incision (Fig. 2A and data not shown).

Photolyase as a Possible Coupling Factor—It has been suggested (Patterson and Chu, 1989) that photolyases function as auxiliary proteins which target lesions for repair by nucleotide excision. Therefore, we conducted further experiments to examine whether more stimulation could be achieved when both RNAP and E. coli DNA photolyase were present in the reaction mixture compared to photolyase alone. Addition of photolyase to a preformed elongation complex fails to relieve the inhibitory effect of RNAP and addition of RNAP to photolyase-T<>T complexes interferes with the stimulatory...
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**Fig. 2.** A, effect of T<>T in the template strand on transcription. 5' terminally labeled synthetic 137-mer ("T" in Fig. 1) labeled at the 5' end of the strand containing the central 11-mer without (lanes 1 and 2) or with T<>T (lanes 3-7) was used as substrate for RNAP and/or (A)BC excinuclease as indicated and the reaction products were analyzed on an 8% polyacrylamide sequencing gel. Transcripts were labeled at the 5' end with [α-32P]UTP. The positions of the full length DNA fragment (137), the DNA band generated by incision 7 bases 5' to T<>T (56) and the full length transcripts (89 and 90) and truncated transcripts (25 and 26) are marked. In lanes 4-6 the DNA was digested for 8 min with (A)BC excinuclease in the presence of rNTPs. In lane 5 the DNA was preincubated with RNAP for 5 min before addition of (A)BC excision nuclease. In lane 6, the DNA was preincubated with UvrA and UvrB for 5 min before adding RNAP and UvrC. Lane 8 is from a separate experiment in which the 137-mer containing T<>T in the non-template strand ("N" in Fig. 1) was transcribed. B, the transcription stopsite in relation to the dimer. 5'-end-labeled 25- and 26-base transcripts (lanes 1-2 and 5-6, respectively) formed in the presence of T<>T (as in A) were excised separately from gels, purified, and sequenced by using the enzymatic method of Donis-Keller et al. (1977). The RNA ladders in lanes 3 and 4 were generated from alkaline hydrolysis of full length transcripts. Note that even though our data shows, unambiguously, that a nucleotide is inserted across from the 3' T residue, we have not actually demonstrated that the inserted nucleotide is dAMP.

**Transcription and Repair of Supercoiled, UV-irradiated DNA**

Transcription causes drastic topological changes in DNA (Gamper and Hearst, 1982; Tsao *et al.*, 1989). It is conceivable that these topological features of transcribed DNA would be associated with the enhanced rate of repair observed in *vivo*. Along these lines Pu *et al.* (1989) recently reported that the rate of incision by (A)BC excinuclease of N-methylmitomycin cross-linked DNA was about 20-fold higher in supercoiled molecules than with relaxed molecules.

To investigate the effect of transcription on repair of superhelical DNA we used pDR3274, which was modified by irradiation with 254-nm light. Repair was measured as the UV-dependent formation of [α-32P]dGTP-labeled repair patches synthesized by the combined action of ABC excinuclease, DNA PolI, and DNA ligase. pDR3274 was selected because it contains a very strongly transcribed UvrC gene (Mulligan *et al.*, 1985; Thomas *et al.*, 1985) and the other genes are either inactive (cam) or weakly transcribed (tet, method of Donis-Keller *et al.* (1977). The RNA ladders in lanes 3 and 4 were generated from alkaline hydrolysis of full length transcripts. Note that even though our data shows, unambiguously, that a nucleotide is inserted across from the 3' T residue, we have not actually demonstrated that the inserted nucleotide is dAMP.
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Bertrand-Burggraf and colleagues (1984). By separating the UvrC gene from silent regions of the plasmid after the repair synthesis reactions, we could compare repair in the UvrC gene with repair in transcriptionally inactive regions.

We first examined transcription of pDR3274 by examining transcripts that were internally labeled with [α-32P]UTP on a 6.5% sequencing gel (data not shown). pDR3274 has three transcriptional units: tac-UvrC (1800 bp), tet (1200 bp), and RNA-I (108 bp). With supercoiled template we established that transcription of this plasmid generates primarily tac-RNA-I (108 bp). With supercoiled template we established that transcription of this plasmid generates primarily tac-UvrC mRNA. We optimized the transcription conditions and found that with 200 nM RNAP the rate of transcription was about 3600 nucleotides plasmid−1 min−1 which corresponds to approximately two transcripts min−1 assuming that all the RNA synthesis was from the tac promoter. This value is reasonably close to the 2.3 transcripts min−1 for the tac operon in vivo under inducing conditions (Lewin, 1987).

We used UV-irradiated pDR3274 in our transcription-repair experiments. Production of full length tac-uvrC transcripts is inhibited in a dose-dependent manner, the transcription level being reduced to approximately e−1 (data not shown) at a dose of 30 J/m2 which is expected to produce about one dimer per strand of the transcription unit. We carried out repair synthesis with DNA irradiated with 75 J/m2, which produces about four dimers per uvrC gene. The reaction mixtures contained a full complement of transcription, repair, and resynthesis proteins in addition to the necessary cofactors and [α-32P]dCTP as a tracer to measure repair synthesis. We used reaction-limiting amounts of the (A)BC excinuclease subunits so that repair synthesis was clearly detectable above the background yet far below saturating in order to detect any stimulatory effect of transcription. Following the transcription-repair reaction, the plasmid was digested with EcoRI, HindIII, and BglII to produce: (a) an ~1.2-kb EcoRI-HindIII fragment which is not transcribed; (b) an ~2.1-kb EcoRI-BglII fragment containing the strongly transcribed UvrC gene; and (c) an ~2.9-kb BglII-HindIII fragment containing the tet gene and the origin of replication.

The fragments were separated on agarose gels which were stained, photographed, and autoradiographed. The results (Fig. 6 and Table I) indicate that the transcribed region of the plasmid is not repaired more efficiently than the nontranscribed regions. With similar concentrations of UV-irradiated DNA and Uvr proteins, the stimulatory effect of photolyase on repair synthesis was easily detectable (Sibghat-Ullah and Sancar, 1990). We considered the possibility that the topological consequences of transcription might be transmitted through the whole plasmid resulting in enhanced repair throughout, thus obscuring gene-specific repair. However, when a comparison is made between repair of all three fragments with or without transcription, no difference is seen (Fig. 6 and Table I). In fact, considering the results obtained with linear DNA one might expect preferential inhibition of repair synthesis of the transcribed sequence when compared to either of the other fragments. While there appears to be a trend in that direction (Table I) the differences are not statistically significant (p < 0.005, Student’s t tests). A plau-
sible explanation for this lack of inhibition is that the inhibitory effect is diluted out because half of the photoproducts are in the nontranscribed strand (repair not inhibited) and stalling of RNAP at the first photoproduct in the template would not inhibit the repair of photoproducts downstream. Attempts to demonstrate an inhibitory effect with DNA containing on the average one T<>T per 60 base pairs would not inhibit the repair of photoproducts downstream. Due to the different sizes of the DNA fragments containing photoproducts, attempts to demonstrate an inhibitory effect could not be made.

From a biological standpoint, for example, it has been suggested (Bohr et al., 1985) that preferential repair of actively transcribed genes may explain the rodent cell paradox: rodent cells which are as resistant to UV as human cells remove only 20% of cyclobutane pyrimidine dimers from bulk DNA in 24 h compared to human cells which remove more than 80% within the same period. However, if the comparison is made at the gene level both cell types remove 80% of the photodimers. The implications are that cells need only to repair genes which control essential functions and that photodimers do not constitute an absolute block to replication and thus can be tolerated. Evidence exists for tolerance mechanisms at the replicational level in both pro- and eucaryotes (Piette and Hearst, 1983; Yang et al., 1982; Vos and Hanawalt, 1987) and such a model makes teleological sense.

In contrast, a comprehensive biochemical model which takes into account the known properties of the transcription apparatus and the nucleotide excision repair system has been difficult to formulate. Three general concepts have been considered: (a) the open conformation of chromatin in transcribed regions may increase accessibility to the repair enzyme; (b) RNAP stalled at a photodimer facilitates the binding and rate of repair by the subunits of the excision repair enzyme in a manner analogous to that of E. coli photolyase (Sancar et al., 1984) and perhaps yeast photolyase (Sancar and Smith, 1989); (c) the unique topology created at the transcription site by overwinding of DNA in front of the transcription complex and underwinding behind it (Tsao et al., 1989) generates a structure with higher affinity for the repair enzyme. Although the open chromatin structure in both pro- and eucaryotes in transcribed regions may make DNA damage more accessible to repair enzymes, it cannot account for the strand selectivity of the excision nucleases unless the template strand is in some way made more accessible than the non-template strand in the open chromatin structure.

Fig. 6. Effect of transcription on repair of superhelical plasmid by (A)BC excinuclease as measured by repair synthesis. Repair synthesis reactions (with DNA polymerase I and DNA ligase) were conducted with undamaged or damaged (75 J/m²) pDR3274, rNTPs, and RNAP (150 nm) as indicated. The incubation with (A)BC excinuclease and DNA pol plus ligase was for 8 min with 2-8 µCi (α-32P)dCTP per reaction as the tracer. Following transcription-repair the DNA was digested with EcoRI, BglII, and HindIII and separated on a 1.2% agarose gel. Top, photograph of ethidium bromide-stained gel; bottom, autoradiograph of the same gel. The bands which carry the strongly transcribed uvrC, weakly transcribed tet, and nontranscribed (promoterless) cam gene are indicated.

Table I

Effect of transcription on repair synthesis in defined regions of pDR3274

| Fragment | Repair synthesis experiments |
|----------|-----------------------------|
|          | −RNA polymerase | +RNA polymerase | +RNAP/−RNAP |
|          | 1 | 2 | 3 | 1 | 2 | 3 |
|          | cpm | cpm | cpm | cpm | cpm |
| tet      | 86(30)* | 61(21) | 212(73) | 71(25) | 79(27) | 232(80) | 1.07 ± 0.13 |
| uvrC     | 61(29) | 36(17) | 262(125) | 37(18) | 50(24) | 227(108) | 0.97 ± 0.23 |
| cam      | 28(24) | 18(15) | 129(107) | 29(24) | 29(24) | 91(76) | 1.10 ± 0.26 |

*Values in parentheses are cpm per kilobase pair (cam, 1.2; uvrC, 2.1; tet, 2.9 kb). +RNAP/−RNAP represents the ratios of cpm per kilobase pair; the means and standard errors of ratios obtained in the three experiments are given.
that a thymine dimer is a strong block to transcription; this has long been inferred but never proven conclusively in either pro- or eucaryotes (Sauerbier and Hercules, 1978). We show that RNAP transcribes up to and including the nucleotide across from the 3' T of the dimer in about 90% of cases (Fig. 2) and stops at the nucleotide across from the 5' T of the dimer in 10% of the molecules (data not shown). For comparison, Shi et al. (1988) found that with psoralen-cross-linked and -monoadducted templates, E. coli RNAP stopped at the last base before the adducted T residue. Transcription up to and including the 3' T of the dimer is one base further than the stopsite of DNA polymerase I at a dimer reported by Moore and Strauss (1978), although a more recent report has shown that PolI can insert dAMP across from the 3' T (Taylor and O'Day, 1990).

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TRANSCRIPTION INHIBITS REPAIR