Reverse Gyrase Recruitment to DNA after UV Light Irradiation in *Sulfolobus solfataricus*

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Induction of DNA damage triggers a complex biological response concerning not only repair systems but also virtually every cell function. DNA topoisomerases regulate the level of DNA supercoiling in all DNA transactions. Reverse gyrase is a peculiar DNA topoisomerase, specific to hyperthermophilic microorganisms, which contains a helicase and a topoisomerase IA domain that has the unique ability to introduce positive supercoiling into DNA molecules. We show here that reverse gyrase of the archaeal *Sulfolobus solfataricus* is mobilized to DNA in vivo after UV irradiation. The enzyme, either purified or in cell extracts, forms stable covalent complexes with UV-damaged DNA in vitro. We also show that the reverse gyrase translocation to DNA in vivo and the stabilization of covalent complexes in vitro are specific effects of UV light irradiation and do not occur with the intercalating agent actinomycin D. Our results suggest that reverse gyrase might participate, directly or indirectly, in the cell response to UV light-induced DNA damage. This is the first direct evidence of the recruitment of a topoisomerase IA enzyme to DNA after the induction of DNA damage. The interaction between helicase and topoisomerase activities has been previously proposed to facilitate aspects of DNA replication or recombination in both Bacteria and Eukarya. Our results suggest a general role of the association of such activities in maintaining genome integrity and a mutual effect of DNA topology and repair.

In all living cells the induction of DNA damage activates an extremely complex network of events involving every cell process from DNA replication and transcription to cell division, protein synthesis and degradation, and, eventually, cell death (1). A major challenge in cell biology is elucidating how these events are regulated and connected.

Archaea are helpful model systems for studying pathways of DNA transactions (replication, transcription, recombination, and repair) that are thought of as simplified and ancient versions of the eukaryal ones (reviewed in Refs. 2 and 3). However, to date the response to DNA damage has been poorly investigated in Archaea.

 Genome sequencing has revealed the presence of archaeal genes homologous to components of the eukaryal nucleotide excision repair pathway, which is involved in the repair of UV-induced DNA lesions (4, 5). We have shown previously that the exposure of *Sulfolobus solfataricus* to UV light and the intercalating agent actinomycin D elicits a DNA damage response with features essentially conserved in all three domains of life, including growth arrest and transcriptional induction of nucleotide excision repair genes. UV light and actinomycin D also regulate the genes encoding two DNA-binding proteins affecting DNA conformation, namely Sul7d and Smj12 (6). Sul7d is an abundant chromosomal protein that induces DNA bending, compaction, and negative supercoiling (7). In contrast, Smj12 induces positive supercoiling (8). Although the reasons for this regulation are currently only matters of speculation, these results support the notion that DNA damage and repair are linked intimately with the structural and dynamic properties of chromatin (9) and prompted us to investigate the involvement of DNA topoisomerases in maintaining genome integrity in *S. solfataricus*.

Three topoisomerases have been characterized in *S. solfataricus* as follows: (i) a type II enzyme called Topo IV, which is homologous to Spo11, the initiator of recombination in eukaryotes (10); (ii) a type III topoisomerase (11); and (iii) reverse gyrase. This latter is a peculiar enzyme, specific to thermophilic microorganisms, that induces positive supercoiling (reviewed in Refs. 12 and 13). Consistent with the presence of this enzyme, the DNA of hyperthermophiles is relaxed or positively supercoiled in vivo (14) in contrast to that of mesophiles, which is negatively supercoiled. Positive supercoiling is considered a mechanism of adaptation to high temperature, and, indeed, reverse gyrase is the only hyperthermophile-specific gene (15). The enzyme contains a N-terminal helicase and a C-terminal Topo IA-type domain that cooperate to introduce positive supercoiling in an ATP-dependent reaction (16, 17).

Although reverse gyrase is unique in its activity, type IA topoisomerases are essential enzymes ubiquitous in Bacteria, Archaea, and Eukarya. Moreover, there are multiple examples of interaction between helicase and topoisomerase activities that are involved in different aspects of DNA metabolism (18). In particular, type IA Topo III enzymes associate physically and functionally with helicases of the RecQ family, forming an evolutionary conserved complex that has essential functions in the maintenance of genome stability (19–22). Genetic defects in human homologs of the RecQ helicase are associated with the Bloom and Werner syndromes, which are characterized by high incidences of chromosomal rearrangements and cancer.

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‡‡The abbreviations used are: Topo, topoisomerase; CCA, covalent complex assay; MES, 4-morpholinopentane sulfonic acid.
tracts were preincubated for 10 min. at 70 °C. Radioactivity was determined with a Storm PhosphorImager (Amersham Biosciences).

were denatured and loaded on 6% SDS-polyacrylamide gels. Radioactivity
quickly chilling on ice and adding SDS-PAGE sample buffer. Samples
were protected from UV light irradiation. Aliquots of irradiated and
controls were mock-treated following exactly the same procedure but
not irradiated.

Protocol for the preparation and fractionation of cell extracts was set up; briefly, cells were lysed by the addition of a nonionic detergent while taking care to minimize the times of manipulation and the mechanical shearing. The lysate was centrifuged to obtain a supernatant and a pellet. The majority of the total cellular protein remained in the supernatant as soluble pellet, whereas the pellet contained genomic DNA with tightly associated chromosomal proteins as well as cell debris (data not shown). We analyzed the amount and distribution of reverse gyrase in cell extracts by Western blotting using a polyclonal antibody directed against the protein purified from S. shibatae (25) (Fig. 1). In total cell extracts, the level of reverse gyrase was not affected by UV irradiation. However, the distribution of the protein between supernatants and pellets was dramatically different in controls and UV light-irradiated cells; in controls the protein was found in the soluble fraction but was almost absent in the pellet. Two hours after UV exposure, the protein was reduced in supernatants in a UV light dose-dependent manner, and consistently, it increased in pellets. As a control, we probed the same filter with an antibody directed against the single-stranded binding protein Ssb (27) (Fig. 1B); the protein was equally distributed between supernatants and pellets, and neither the amount or the distribution were affected by UV light irradiation. Reverse gyrase was quantified in multiple independent experiments (Fig. 1C). Whereas in controls 90% of the protein was found in the soluble and 10% in the insoluble fraction, at the highest UV dose used the ratio was reversed, with ~40% of the protein in the supernatant and 60% in the chromatin fraction.

These results showed that, in control cells, reverse gyrase is present almost exclusively in the soluble fraction, suggesting either that it is mostly cytoplasmic or that it is associated with the chromatin less tightly than are Ssb and the other DNA-binding proteins that were copurified in significant amounts with our pellet fraction (Fig. 1B and data not shown).

If total cell extracts were first subjected to digestion with DNase I and then fractionated, most of the reverse gyrase present in the pellet was solubilized, and the distribution of the protein in UV light-irradiated cells became similar to that in controls (Fig. 1D). We conclude that UV light radiation does not affect the global reverse gyrase amount but induces its translocation from the supernatant to the chromatin fraction, where it is associated with genomic DNA.

Effects of UV Exposure on Reverse Gyrase Activity—To test whether the change in the fractionation properties of reverse gyrase following UV irradiation is associated with changes in its activity, we assayed positive supercoiling activity in cell extracts. We used two-dimensional agarose gel electrophoresis to separate positive and negative topoisomers (Fig. 2A). When a highly negatively supercoiled DNA plasmid (Fig. 2B) was incubated at 70 °C with total cell extracts, highly positive topoisomers were produced; such activity was unchanged in extracts prepared 2 h after exposure to UV light.

**RESEARCH**

**Effect of UV Radiation on the Amount and Intracellular Localization of Reverse Gyrase—Exponential S. solfataricus cultures grown at 80 °C were UV light-irradiated at room temperature with two different UV fluences that were shown previously to elicit a transcriptional response without inducing massive cell death (6). Aliquots of cultures were withdrawn after different durations of recovery at 80 °C and analyzed. Because the cultures undergo rapid temperature changes during the experiment and it has been shown that DNA topology is affected by temperature shock in Sulfolobales (26), in each experiment an aliquot of the culture was routinely mock-treated exactly in the same way as samples, but not irradiated.

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regardless of the dose used (data not shown). However, the distribution of the activity between supernatants and pellets was different in controls and UV light-irradiated cells (Fig. 2C); in controls, activity was found almost all in the supernatants but was barely detectable in pellets. In soluble extracts prepared 2 h after exposure to 200 J/m², the activity was reduced, and the reduction was much more pronounced at 650 J/m². Consistently, the opposite was found in pellets; positive supercoiling activity increased in UV light-irradiated cells in a dose-dependent manner.

A quantitative comparison of reverse gyrase activity was obtained by calculating the relative amount of total products in each sample (Fig. 2D). Enzyme activity calculated in this way was in good agreement with the distribution of the polypeptide shown in Fig. 1C.

We have shown in Fig. 1D that reverse gyrase can be solubilized from pellets by digestion with DNase I. In a similar experiment, we tested whether positive supercoiling activity could also be rescued in supernatants of UV light-irradiated cells (Fig. 3A). Because DNase I treatment would digest the substrate during the assay, extracts were prepared from aliquots of the same culture and either treated in the usual manner (Fig. 3A, lanes 1 and 2) or sonicated before the separation of supernatants and pellets (Fig. 3A, lanes 3 and 4). Because sonication solubilizes most protein, sonicated and non-sonicated extracts cannot be directly compared. Whereas in supernatants prepared from UV light-irradiated cells the reverse gyrase activity was reduced (Fig. 3A, compare lane 2 with lane 1), this difference was no longer observed in sonicated extracts (Fig. 3A, compare lane 4 with lane 3). Indeed, quantitation of results showed that, after sonication, the supernatants of control and UV light-irradiated cells showed similar efficiencies of product formation (Fig. 3B). From these data we concluded that the distribution of positive supercoiling activity was consistent with the distribution of the protein as determined by Western blotting.

Analysis of Reverse Gyrase Cleavage Activity—The results shown in the previous paragraph suggest that the enzyme in pellets of UV-irradiated cells is fully active. However, crude extracts contain, in addition to reverse gyrase, other factors that regulate DNA topology (Topo III and Topo VI, DNA-binding proteins). Therefore, in our assay the observed topology might be the result of different (and opposite) activities. Moreover, positive supercoiling activity itself is a complex reaction (17). The catalytic cycle of topoisomerases can be dissected into four steps as follows: (i) DNA binding; (ii) cleavage; (iii) strand passage; and (iv) religation of the DNA ends (reviewed in Refs. 13 and 28). A covalent DNA-protein complex is formed as a transient intermediate; in particular, type IA topoisomerases form a phosphodiester bond between a DNA 5’-end and the catalytic tyrosine. It has been shown previously that covalent complexes between DNA and purified reverse gyrase can be trapped if reactions are performed in the absence of ATP and quickly blocked with detergents (29) and that the efficiency of covalent complex formation can be used as a measure of the cleavage activity of reverse gyrase (30).

We set up a CCA to evaluate the efficiency of covalent complex formation as an independent direct measure of reverse gyrase activity. We incubated fractionated extracts with end-labeled oligonucleotides under the conditions described (29); complexes were visualized as radioactive protein bands in SDS-PAGE (Fig. 4). Because reverse gyrase shows a certain degree
of sequence preference (29), we first screened several sequences for covalent complex formation (data not shown). We selected RGA, a 35-bp oligonucleotide, which was more effective than the previously characterized SA oligonucleotide (29). When 3'- but not 5'-end-labeled RGA was incubated with purified reverse gyrase, radioactivity was found associated with the protein (which is ~120 kDa), showing that reverse gyrase forms a covalent complex with a DNA 5'-end (Fig. 4B, lanes 2 and 4). A band of similar molecular weight was obtained incubating soluble cell extracts with 3'- but not 5'-end-labeled RGA (Fig. 4B, lanes 1 and 3). If, after complex formation, the reactions were incubated again at 70 °C, the complex disappeared in a few minutes, showing that it corresponds to a rapidly processed reaction intermediate (Fig. 4C).

We then analyzed by CCA the effects of UV light irradiation on covalent complex formation in cell extracts. In soluble fractions, 30 min after irradiation the complex was formed with the same efficiency as in controls, regardless of the UV light dose used. However, 2 h after irradiation it was strongly reduced if 200 J/m² were used and completely absent at 650 J/m² (Fig. 5A). In contrast, complexes were formed more efficiently in pellets of UV light-irradiated cells than in controls (Fig. 5B). Exact quantitation of the efficiency of covalent complex formation was impaired by the transient nature of the intermediate and the lack of any possible internal control; the experiments were repeated several times with five independently prepared extracts and always confirmed that CCA-forming activity was reduced in supernatants and increased in pellets of UV-light-irradiated cells. Taken together, our data strongly suggest that UV irradiation affects the localization but not the activity of reverse gyrase.

Because no relocalization of reverse gyrase was observed until 30 min after UV light irradiation (Fig. 5A), it is possible that some intermediate events are involved in the recruitment of reverse gyrase onto DNA. However, pre-treatment with puromycin, an antibiotic that blocks protein synthesis in S. solfatarius (31), did not prevent the UV light-dependent translocation of the covalent complex-forming activity, thus suggesting that it does not require the synthesis of new protein factors (data not shown).

**Effect of UV Light-induced DNA Lesions on Reverse Gyrase Activity**—It has been shown that in vivo eukaryotic Topo I (a type IB enzyme) is rapidly mobilized to the genome after DNA damage and that in vitro Topo I-DNA covalent complexes are stabilized by DNA-damaging agents, including UV light (32, 33). Moreover, the activity of Micrococcus luteus DNA topoisomerase I (a type IA enzyme) is also inhibited by UV light-induced lesions in vitro (34).

We analyzed the activity of reverse gyrase on DNA substrates exposed previously to UV light in vitro (Fig. 6A). Positive supercoiling activity of the enzyme, either purified or in supernatants, was strongly reduced by the presence of UV lesions; interestingly, the negative substrate was completely processed, but a nicked form accumulated, suggesting that the reaction was blocked after the cleavage and before the ligation step.

We then analyzed directly by CCA the effect of UV lesions on the in vitro formation of reverse gyrase-DNA covalent complexes (Fig. 6B). Complexes were formed more efficiently when irradiated DNA was used as compared with controls. This effect is not sequence-specific, because the same result was obtained with
either RGA or SA. These results indicated that it is the presence of UV lesions that stabilizes the covalent complexes.

Effect of Actinomycin D on Reverse Gyrase Localization and Activity—Actinomycin D is an intercalating drug that induces a dual effect, i.e., direct DNA damage and a block of transcription. We have shown previously that, in *S. solfataricus*, actinomycin D, although blocking general transcription, induces specific transcriptional regulation of the same nucleotide excision repair and chromatin genes that are also regulated by UV light radiation (6), thus suggesting that both UV light- and actinomycin D-induced DNA damage may elicit a similar response and be transduced by the same mechanism.

We tested whether actinomycin D might affect reverse gyrase activity and/or distribution in *vivo*. Exponential cultures were treated with actinomycin D, and extracts were prepared and fractionated as described above. The dose used (10 μg/ml) and the duration of treatment (2 h) were shown previously to induce a DNA damage response in *S. solfataricus* (6). Aliquots of the same culture untreated with the drug were always used as controls. Level and distribution of reverse gyrase (Fig. 7A) and positive supercoiling activity (Fig. 7B) between supernatants and pellets of actinomycin D-treated cultures were similar to those in controls. Therefore actinomycin D, unlike UV light, does not induce translocation of reverse gyrase from the soluble to the insoluble fraction.

We then tested directly the effect of actinomycin D addition on positive supercoiling activity (Fig. 7C). When added to reactions containing purified reverse gyrase or cell extracts, the drug inhibited positive supercoiling activity. However, it did not result in the accumulation of nicked plasmid, as was observed for UV-damaged substrates; rather, the substrate was left negatively supercoiled, suggesting that actinomycin D impairs early step(s) of the reaction before strand cleavage. Indeed, actinomycin D prevented the formation of the reverse gyrase-oligonucleotide covalent complex (Fig. 7D). This result strongly suggests that reverse gyrase inhibition by UV and actinomycin D follows a different mechanism. Actinomycin D inhibits type IB and II topoisomerases; however, whereas at low concentration it stimulates topoisomerase-induced cleavage, at higher concentrations it suppresses enzyme-mediated DNA cleavage (35). To our knowledge, this is the first report of an actinomycin D effect on a type IA topoisomerase.

**DISCUSSION**

Using three independent techniques, we have shown that UV light radiation induces the translocation of reverse gyrase to DNA *in vivo* without affecting its total amount or activity (both strand cleavage and positive supercoiling). *In vitro*, reverse gyrase is blocked after the cleavage step by the presence of UV light-induced lesions. In contrast, actinomycin D, which has effects similar to UV light irradiation on gene expression in *Sulfolobus* (6), does not induce translocation of reverse gyrase *in vivo* or stabilization of covalent complexes *in vitro*, thus showing that those effects are UV light-specific.

Under the conditions used here, *Sulfolobus* genomic DNA contains a high number of cyclobutane pyrimidine dimers (6); although our experiments do not directly prove that this is the mechanism of reverse gyrase recruitment *in vivo*, the simplest
lesions in vitro (34), but its association with DNA in vivo has not been tested.

Actinomycin D is known to induce positive supercoiling of DNA and could inhibit reverse gyrase activity for this reason; however, two lines of evidence suggest that this is not the case. First, ethidium bromide, which also induces positive supercoiling, does not inhibit the enzyme. Third, actinomycin D prevents the cleavage of a linear substrate (Fig. 7D). No data have been reported so far on the effects of actinomycin D on type IA enzymes; the different behavior of reverse gyrase and eukaryal Topo I suggests that the effect of different DNA-damaging agents might be enzyme-specific.

Several hypotheses can be made to explain the biological meaning of our results. Reverse gyrase recruited to DNA might assist repair, either directly or by signaling lesions and/or recalling repair factors. Interestingly, reverse gyrase N-terminal domain contains a RecA-type fold also found in the Escherichia coli nucleotide excision repair protein UvrB, and both proteins show non-processive strand displacement activity (17). One attractive hypothesis is that the N-terminal part of reverse gyrase might work on UV light-irradiated DNA like UvrB.

On the other hand, if UV lesions stabilize covalent complexes between reverse gyrase and DNA, such complexes might amplify the damage, exacerbating the cell response. Indeed, Topo I and Topo II cleavage complexes can be stabilized by enzyme-specific inhibitors, including potent anticancer drugs that induce DNA damage and checkpoint response (39). No such drug is known for reverse gyrase (or other type IA topoisomerases). However, eukaryal Topo I is also blocked at UV light-damaged sites where it has been shown to interact with p53, suggesting a role for Topo I in the decision between repair and apoptosis (37). A third possibility is that reverse gyrase engagement on

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3. A. Napoli and M. Ciaramella, unpublished observations.
DNA is a mechanism to reduce its activity, which might be needed to allow repair. Indeed in E. coli DNA lesions located on positively supercoiled regions are not repaired in vivo (40). However the actual topology of the DNA substrate in vivo during repair reaction is unknown, and studies on the effect of DNA topology on the activity of archaeal repair factors are lacking. Although further studies are required to distinguish among these hypothesis, we have shown that the chromatin-bound reverse gyrase is active, suggesting a functional recruitment of the enzyme rather than an inactivation mechanism.

Although its function in vivo has never been demonstrated, reverse gyrase has been associated with adaptation to high temperature; we have shown that it is involved, directly or indirectly, in DNA damage response in Archaea. Moreover, our results might have more general implications. In several systems, the combination of helicase and topoisomerase activities has been shown to facilitate aspects of DNA replication or recombination by either introducing supercoiling or by helping to resolve replication or recombination intermediates (19–23, 41). Immunolocalization of Topo III in fission yeast and human cells showed that, after the induction of DNA damage, these proteins form distinct nuclear foci that colocalize with the RecQ helicase homologs Rhq1 and BLM, respectively. Foci formation is stimulated by DNA damage (UV light or ionizing radiations) (42, 43). Moreover, in mitotic cells BLM is dephosphorylated and translocated to DNA after γ-ray irradiation; however, whether the chromatin-bound BLM is also associated with Topo III has not been determined (44). Our observations that reverse gyrase (consisting of a helicase and a topoisomerase domain) is translocated to DNA after the induction of DNA damage raises the possibility that helicases like RecQ might be recruited to damaged DNA through their association with topoisomerases.

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REFERENCES

1. Eckardt-Schupp, F., and Klaus, C. (1999) Biochimie 81, 161–171.
2. Bell, S. D., and Jackson, S. P. (2001) Curr. Opin. Microbiol. 4, 208–213.
3. Ciaramella, M., Pisani, F. M., and Rossi, M. (2002) Antonie Leeuwenhoek 81, 85–97.
4. Aravind, L., Walker, D. R., and Koonin, E. V. (1999) Nucleic Acids Res. 27, 1223–1242.
5. White, M. F. (2003) Biochem. Soc. Trans. 31, 690–693.
6. Salerno, V., Napoli, A., White, M. F., Rossi, M., and Ciaramella, M. (2003) Nucleic Acids Res. 31, 6127–6138.
7. Napoli, A., Zivanovic, Y., Bocci, C., Buhler, C., Rossi, M., Forster, P., and Ciaramella, M. (2002) Nucleic Acids Res. 30, 2656–2662.
8. Napoli, A, Kvaratskhelia, M., White, M. F., Rossi, M., and Ciaramella, M. (2001) J. Biol. Chem. 276, 10745–10752.
9. Thoma, F. (1999) EMBO J. 18, 6585–6598.
10. Bergerat, A., de Massey, B., Guedel, D., Varoutas, P. C., Nicolas, A., and Forster, P. (1997) Nature 386, 414–417.
11. Dai, P., Wang, Y., Ye, R., Chen, L., and Huang, L. (2003) J. Bacteriol. 185, 5500–5507.
12. Dutertre, M. (1995) in Nucleic Acids and Molecular Biology (Eckstein, F., and Lilley, D. M. J., eds) Vol. IX, pp. 84–114, Springer-Verlag, Berlin-Heidelberg.
13. Champortal, J. J. (2001) Annu. Rev. Biochem. 70, 369–413.
14. Forster, P., Bergerat, A., and Lopez-Garcia, P. (1996) FEMS Microbiol. Rev. 18, 237–248.
15. Rodriguez, A. C., and Stock, D. (2002) EMBO J. 21, 418–426.
16. Declais, A. C., Marsault, J., Confalonieri, F., de La Tour, C. B., and Duguet, M. (2000) J. Biol. Chem. 275, 19486–19504.
17. Wadsworth, R. I., and White, M. F. (2003) Nucleic Acids Res. 22, 914–920.
18. Wang, J. C. (2002) Nat. Rev. Mol. Cell. Biol. 3, 430–440.
19. Jaxel, C., Duguet, M., and Nadal, M. (1999) Eur. J. Biochem. 266, 103–111.
20. Rodriguez, A. C. (2003) Biochemistry 42, 5993–6004.
21. Jaxel, C., and Bernard, R. (2006) Mol. Microbiol. 10, 225–234.
22. Lanza, A., Tormaletti, S., Redolfi, C., Scavavini, M. C., and Pedrini, A. M. (2006) J. Biol. Chem. 271, 6976–6986.
23. Subramanian, D., Rosenberg, B. S., and Muller, M. T. (1998) Cancer Res. 58, 976–984.
24. Pedrini, A. M., and Ciarrocchi, G. (1983) Proc. Natl. Acad. Sci. U. S. A. 80, 1787–1791.
25. Wasserman, K., Markovits, J., Jaxel, C., Capranico, G., Kohn, K. W., and Pommier, Y. (1999) Mol. Pharmacol. 53, 38–45.
26. Trask, D. K., and Muller, M. T. (1998) Proc. Natl. Acad. Sci. U. S. A. 85, 1417–1421.
27. Mao, Y., Okada, S., Chang, L. S., and Muller, M. T. (2000) Cancer Res. 60, 4538–4543.
28. Mao, Y., Okada, S., Chang, L. S., and Muller, M. T. (2000) Cancer Res. 60, 1115–1126.
29. Pommier, Y., Redon, C. A., Vass, J. A., Sordet, O., Takemura, H., Antongi, S., Meng, L., Liao, Z., Kohlhagen, G., Zhang, H., and Kohn, K. W. (2000) Mutat. Res. 432, 173–203.
30. Park, J. Y., and Ahn, B. (2000) FEBS Lett. 476, 174–178.
31. Bischof, O., Kim, S. H., Irving, J., Beresten, S., Ellis, N. A., and Campisi, J. (2003) J. Cell Sci. 116, 380–389.
32. Jaxel, C., Duguet, M., and Nadal, M. (1999) Eur. J. Biochem. 266, 103–111.
33. Rodriguez, A. C. (2003) Biochemistry 42, 5993–6004.
34. Jaxel, C., and Bernard, R. (2001) Mol. Microbiol. 40, 225–234.
35. Lanza, A., Tormaletti, S., Redolfi, C., Scavavini, M. C., and Pedrini, A. M. (2006) J. Biol. Chem. 271, 6976–6986.
36. Subramanian, D., Rosenberg, B. S., and Muller, M. T. (1998) Cancer Res. 58, 976–984.
37. Pedrini, A. M., and Ciarrocchi, G. (1983) Proc. Natl. Acad. Sci. U. S. A. 80, 1787–1791.
38. Wasserman, K., Markovits, J., Jaxel, C., Capranico, G., Kohn, K. W., and Pommier, Y. (1999) Mol. Pharmacol. 53, 38–45.
39. Trask, D. K., and Muller, M. T. (1998) Proc. Natl. Acad. Sci. U. S. A. 85, 1417–1421.
40. Mao, Y., Okada, S., Chang, L. S., and Muller, M. T. (2000) Cancer Res. 60, 4538–4543.
41. Mao, Y., and Muller, M. T. (2003) DNA Repair 2, 1115–1126.
42. Pommier, Y., Redon, C. A., Vass, J. A., Sordet, O., Takemura, H., Antongi, S., Meng, L., Liao, Z., Kohlhagen, G., Zhang, H., and Kohn, K. W. (2000) Mutat. Res. 432, 173–203.
43. Park, J. Y., and Ahn, B. (2000) FEBS Lett. 476, 174–178.
44. Bischof, O., Kim, S. H., Irving, J., Beresten, S., Ellis, N. A., and Campisi, J. (2003) J. Cell Sci. 116, 380–389.
45. Jaxel, C., Duguet, M., and Nadal, M. (1999) Eur. J. Biochem. 266, 103–111.
46. Rodriguez, A. C. (2003) Biochemistry 42, 5993–6004.
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