The low molecular weight GTP-binding proteins RhoA, RhoB, and RhoC are characterized as specific substrates for the ADP-ribosyltransferase C3 from Clostridium botulinum and are supposed to be involved in the organization of the microfilamental network and transformation. RhoB is known to be immediate-early inducible by growth factors and protein-tyrosine kinases. Since increasing evidence indicates overlapping of growth factor- and UV-induced signal pathways, we studied the effect of UV light and other genotoxic agents on early rhoB transcription. Within 30 min after UV irradiation of NIH3T3 cells, the amount of rhoB mRNA increased 3-4-fold. Elevated rhoB mRNA was accompanied by an increase in RhoB protein, as detected by C3-mediated [32P]ADP-ribosylation. The transcription inhibitor actinomycin D prevented the UV-induced increase in rhoB mRNA and proved rhoB mRNA to be unstable with a half-life of ~20 min. Transcriptional activation of rhoB by UV light was confirmed by run-on analysis. The increase in rhoB mRNA after UV irradiation was prevented by inhibitors of protein kinase A (H9) and C (H7, Gö18). The tyrosine kinase inhibitor genistein did not affect UV induction of rhoB. In addition to UV, N-methyl-N-nitrosourea and the cytosstatic drug cisplatin evoked rhoB response. Cycloheximide was likewise effective in increasing the amount of rhoB mRNA, whereas Bt2cAMP, 12-O-tetradecanoylphorbol-13-acetate, and retinoic acid were without effect. Prior down-regulation of signaling by 12-O-tetradecanoylphorbol-13-acetate and serum pretreatment reduced UV-stimulated rhoB expression. The data indicate that rhoB represents a novel DNA damage-inducible function involved in early steps of signal transduction upon genotoxic stress.

Signaling after mitogenic stimulation of cells has been extensively investigated (1–6). It is characterized by rapid and transient transcriptional activation of genes such as c-fos, fosB, fra-1, c-jun, junB, and NGF1-A, all of which encode DNA-binding proteins (7–13). On the other hand, regulation of cellular responses after exposure to DNA damaging agents is still not well understood. This is particularly true for the early effects caused by DNA damage. Various mammalian gene functions inducible by DNA damaging treatments have been described, such as human collagenase and human plasminogen activator (14, 15), metallothionein (16, 17), p53 (18, 19), EPIF (20), thymidylate synthase (21), the DNA-repair protein O6-methylguanine-DNA-methyltransferase (22, 23), β-polymerase (24, 25), and the proto-oncogenes c-fos and c-jun (26–31). Among these inducible functions, only the proto-oncogenes c-fos and c-jun are transcriptionally activated within minutes after induction of DNA damage. Therefore, these two genes, whose products act as trans-activators by forming the transcription factor AP-1 (26, 32, 33), are referred to as “immediate-early” inducible.

Recently it has been shown that rhoB is immediate-early inducible from growth factors and v-fps (34). Interestingly, the gene product of rhoB belongs to the family of Ras homologous small GTP-binding proteins. The RhoD protein family consists of at least three highly homologous members (RhoA, -B, and -C (35, 36)). RhoA and RhoC are known to be involved in the regulation of the actin cytoskeleton (37–44). In addition, RhoA has been shown to interfere with cell adhesion (45) and transformation (46). Apparently, Rho proteins (RhoA, -B, and -C) have different intracellular locations (47), indicating different physiological functions. Consistent with this is the observation that the expression of rhoB, but not of rhoA or rhoC is rapidly stimulated by growth factors (34). Transcriptional stimulation of rhoB by mitogens appears to differ from other immediate-early genes, like c-fos (34), suggesting a novel pathway for mitogen-induced cellular responses. Interestingly, RhoB has recently been suggested to be involved in cell growth control and Ras-mediated oncogenic transformation (48).

So far, GTP-binding proteins have not been examined as regulators in early signaling after DNA damage, although they could rapidly control adaptive cellular reactions by GTP binding and GTP hydrolysis, thereby changing the activity of various downstream targets. Since the immediate-early genes c-fos and c-jun are not only inducible by mitogens but also by UV light, the question arose whether this would be true for rhoB too. Thus, the recently published data on rhoB regulation (34) prompted us to investigate whether rhoB is part of the early cellular response to DNA damage. Here we show that UV light and other DNA-damaging agents increase RhoB very rapidly by transcriptional activation of the gene, apparently utilizing a pathway not common for previously described early-responsive genes.
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EXPERIMENTAL PROCEDURES

Materials—N-Methyl-N-nitrosourea (MNU) and cisplatin were purchased from Sigma. Protein kinase inhibitors H7 and H9 were obtained from RBI (Research Biochemicals Inc.). Protein kinase G018 was kindly provided by Dr. Schachtele (Goedde, Freiburg, 47). The tyrosine kinase inhibitor genistein was obtained from Sigma.

Cell Culture—Mouse NIH3T3 cells were routinely grown in Dulbecco’s modified Eagle’s medium containing 10% heat-inactivated FCS, 2 mm glutamine, 200 units/ml penicillin, and 100 μM streptomycin. For serum starvation, cells were washed twice with phosphate-buffered saline (PBS) and cultured in the presence of Dulbecco’s modified Eagle’s medium containing 0.5% FCS for 24 h. Before UV treatment (254 nm), medium was removed. All other treatments were performed by adding the drug directly to the medium.

Northern Blot Analysis—After treatment of exponentially growing NIH3T3 cells, medium was removed and cell layer washed twice with ice-cold PBS. Subsequently, cells were lysed onto the plates with guanidinium thiocyanate and total RNA was prepared as described (49). After separation on 1.2% agarose gels, RNA was transferred to Hybond N- membranes overnight (transfer buffer: 50 mm NaOH). Prehybridization was performed in 0.5 x saline-sodium chloride (SSC) (1 x SSC = 0.15 M NaCl, 0.015 M sodium citrate) and 1 x EDTA for 2 h. Hybridization was done overnight in the same solution additionally containing 1% bovine serum albumin and 32P-labeled probe (106 cpm/ml). Filters were washed 2 × 30 min in a solution containing decreasing salt concentrations (2 × SSC (1 × SSC) + 0.5% SDS + 1 mM EDTA). All steps were performed at 65 °C. Rat rhoB-cDNA was kindly provided by Dr. T. Hunter (San Diego, CA), the human rhoA-CDNA by Dr. A. Hall (London, United Kingdom). For hybridization and hybridization analysis we used a 0.9-kilobase EcoRI fragment from the 3′-region of rhoB-CDNA (containing rhoB-specific coding and noncoding sequences). Amounts of total rho mRNAs were determined by hybridization with the whole coding sequence of rhoA-CDNA, which cross-hybrizes to all rho mRNA species because of their high homology (36). The c-fos and GAPDH cDNA hybridization probes were obtained from Dr. H. J. Rahmsdorf (Institute of Genetics, Research Center, Kaisersluehn, Federal Republic of Germany). For quantitation of the data, densitometrical analysis was performed. Relative gene expression was calculated by referring rhoB (rho, c-fos) mRNA to the amount of GAPDH mRNA and by relating to control cells included in each experiment.

2P Labeling of RNA—Run-on experiments were essentially performed as described (28). 5 μg of plasmid DNA containing rhoB-, c-fos-, c-jun-, and GAPDH-cDNA sequences were heat-denatured (10 min, 95 °C) and blotted onto Hybond N- filter using a slot-blot apparatus. Nuclei from UV-irradiated and non-irradiated NIH3T3 cells were incubated in a buffer containing 10 mm Tris-HCl (pH 8.0), 5 mm MgCl2, 300 mm KCl, 0.5 μM of each of dATP, dCTP, and dTTP, and 100 μCi of [32P]GTP (3000 Ci/mM) at 30 °C. Reaction was stopped by Dnase I treatment (20 μg/ml, 5 min, 30 °C) followed by proteinase K digestion (30 min, 42 °C). After phenol/chloroform extraction, 32P-labeled RNA was precipitated by trichloroacetic acid and filtered on BA85 filters (Millipore). After elution from the filters, 32P-labeled RNA was ethanol-precipitated. Hybridization of the blots with [32P]RNA was performed as described (see “Northern Blot Analysis”).

Transient Transfection—A 1.6-kilobase EcoRI fragment from rat rhoB cDNA (27) was cloned both in sense and antisense orientation into the eukaryotic expression vector pSVT7 (gift of Dr. U. Guenther, Freidrich-Mieseler Institute, Basel) and pMAMneo (Clontech), respectively. Transfection of NIH3T3 cells was performed with 20 μg of DNA expression plasmid using the calcium phosphate co-precipitation technique (50). 16 h after transfection, cells were fixed for FITC staining as described below.

ADP-ribosylation—NIH3T3 cells were disrupted by sonication in ice-cold buffer containing 10 mm Tris-HCl (pH 7.4), 1 mm EDTA, 1 mm MgCl2, 0.1 mm phenylmethylsulfonyl fluoride. After centrifugation (10 min, 600 × g, 4 °C), supernatant was used for protein determination according to Bradford (51). ADP-ribosylation of cell lysates was performed as described (52) using tritium-labeled botulinum C3 or botulinum C2 exoenzyme (Rho-specific) ([32P]ADP-ribosylation) or C. botulinum C2 exoenzyme (labeled ADP-ribosylation of G-actin). 20–50 μg of protein from total extracts were incubated for 30 min at 37 °C in buffer containing 20 mm Tris-HCl (pH 7.4), 1 mm EDTA, 1 mm MgCl2, 1 mm dithiothreitol, 10 mm thymidine, 0.2 μM NAD, 0.5 μCi of [32P]NAD, and 0.1 μg of C3 (0.5 μg of C2). Reaction products were then analyzed by one-dimensional SDS-gel electrophoresis and visualized by autoradiography. Alternatively, we used phosphorimaging for quantitative analysis (53). [32P]ADP-ribosylated proteins were detected after exposure of dried gels on Kodak X-Omat films.

RESULTS

To address the question, whether rhoB is inducible by genotoxic agents, NIH3T3 cells were exposed to UV light and rhoB mRNA levels were measured various times after irradiation. As shown in Fig. 1A, a 3- to 4-fold increase in the amount of rhoB mRNA was observed 30 min after UV treatment, as detected by hybridization with a rhoB-specific probe. This hybridization probe mainly consists of the 3′-noncoding region of rat rhoB cDNA and does not cross-hybridize to rhoA or rhoC mRNA. As expected, the amount of c-fos mRNA which was taken as internal standard, was also enhanced upon UV irradiation. Furthermore, rehybridization of the filter was performed with a hybridization probe covering the whole coding region of rhoB. Because of the high homology (~85%) of the diverse Rho species (36), this hybridization probe cross-hybridizes with all rho mRNAs (rhoA, -B, and -C). Using this probe, no UV-induced change in total rho mRNA was observed, indicating that rhoB mRNA most likely represents only a minor portion of total rho mRNA (rhoA, -B, and -C). In contrast to UV, TPA did not increase rhoB mRNA (Fig. 1A). Kinetic analysis of the UV response showed maximal amounts of rhoB mRNA 30 min after irradiation (Fig. 1B). 2 h after exposure to UV, rhoB mRNA returned to basal level again. Increase in rhoB mRNA was already observed with low doses (10 J/m2) of UV (Fig. 1C), exerting only slight toxic effects (90% cell survival). The level of rhoB mRNA was similarly increased after UV treatment of serum-starved or confluent cells, indicating that the UV response of rhoB did not depend on proliferation (not shown). Additionally, we analyzed constitutive and UV-induced rhoB expression on protein level using the specific ADP-ribosylation of Rho proteins by C. botulinum exoenzyme C3 (37–41). Separation of [32P]ADP-ribosylated cell extracts by two-dimensional gel electrophoresis showed that RhoA and RhoC are the major Rho proteins constitutively expressed in NIH3T3 cells (Fig. 2). In contrast, basal amounts of RhoB are very low. 1 h after UV...
Fig. 2. Induction of RhoB protein after UV treatment of NIH3T3 cells. Logarithmically growing NIH3T3 cells were irradiated with 30 J/m² and cells were harvested 1 h later. 50 μg of protein from total cell extracts was ADP-ribosylated by C3 and [32P]ADP-ribosylated proteins were separated by two-dimensional gel electrophoresis. Autoradiograms of dried gels are shown. Control, control extract from non-irradiated cells. Arrows indicate increase in the H^+ gradient. Numbers 1, 2, and 3 (marked with arrows) indicate the position of Rho species RhoA, RhoC, and RhoB, respectively.

Next we studied the effect of the transcription inhibitor actinomycin D on rhoB mRNA. As shown in Fig. 3A, actinomycin D prevented the UV-induced increase in rhoB mRNA, indicating that the rhoB gene was transcriptionally activated upon UV irradiation. This was confirmed by run-on analysis showing transcriptional activation of rhoB within 15 min after UV irradiation (Fig. 3B). To determine the stability of rhoB mRNA, logarithmically growing NIH3T3 cells were treated for various periods of time with actinomycin D. As shown in Fig. 3C, 90 min after actinomycin D addition rhoB mRNA was not longer detectable. Densitometric analysis of the autoradiogram indicated a half-life of rhoB mRNA of ~20 min (Fig. 3D). In contrast to rhoB mRNA, total rho mRNA did not decrease in the presence of actinomycin D (Fig. 3, C and D).

To further analyze the regulation of rhoB expression, we investigated the kinetics of rhoB mRNA increase and its subsequent degradation after treatment with serum or cycloheximide, both of them are well known inducers of c-fos. Serum stimulation of quiescent NIH3T3 cells and cycloheximide treatment of exponentially growing NIH3T3 cells both resulted in a rapid increase in rhoB mRNA. Notably, the level of rhoB mRNA remained enhanced for a longer period of time than c-fos mRNA (Fig. 4, A and B). As already observed after UV treatment, total rho mRNA level did not change after serum stimulation or cycloheximide treatment. Furthermore, neither cycloheximide nor serum influenced the level of the C3-mediated ADP-ribosylation of Rho proteins (not shown).

Since protein kinases interfere with the regulation of the UV-stimulated expression of c-fos (9, 16, 26, 53), we analyzed the involvement of protein kinases in the UV induction of rhoB. NIH3T3 cells were treated with different protein kinase inhibitors before UV irradiation and then the level of rhoB mRNA was assayed. As shown in Table I, the UV-stimulated increase in the amount of rhoB mRNA was blocked after inhibition of protein kinase C by the protein kinase C-inhibitors H7 and G618. Likewise, UV-stimulated expression of the c-fos gene was inhibited by H7 (not shown). Pretreatment of cells with the protein kinase A inhibitor H9 reduced both the basal and the UV-stimulated level of rhoB mRNA (Table I). These data indicate that protein kinases A and C are involved in the UV-
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Effect of protein kinase inhibitors on UV- and serum-stimulated expression of rhoB

| Treatment | Relative rhoB inductiona |
|-----------|--------------------------|
| A         |                         |
| Control   | 1.0                      |
| H7        | 0.6                      |
| UV        | 4.9                      |
| UV + H7   | 0.2                      |
| Control   | 1.0                      |
| G018      | 0.5                      |
| UV        | 2.7                      |
| UV + G018 | 1.0                      |
| Control   | 1.0                      |
| H9        | 0.1                      |
| UV        | 3.3                      |
| UV + H9   | 0.5                      |
| Control   | 1.0                      |
| Gen       | 0.9                      |
| UV        | 3.0                      |
| UV + Gen  | 2.8                      |
| B         |                         |
| Control   | 1.0                      |
| +FCS      | 4.9                      |
| +FCS + H7 | 1.1                      |
| +FCS + G018 | 1.5                   |
| +FCS + H9 | 3.9                      |
| Control   | 1.0                      |
| +FCS      | 4.0                      |
| +FCS + G018 | 1.3                   |

aIncrease in relative rhoB mRNA in treated cells, as compared with untreated control (~1.0-fold). Relative rhoB mRNA was determined in relation to GAPDH mRNA.

TABLE II

TPA- and FCS pretreatment reduce the UV-stimulated expression of rhoB

| Treatment | Relative rhoB inductiona |
|-----------|--------------------------|
| Control   | 1.0                      |
| UV        | 6.2                      |
| TPA       | 1.0                      |
| TPA + UV  | 1.9                      |
| FCS       | 1.4                      |
| FCS + UV  | 3.0                      |

RhoA and RhoC have been shown to interfere with the regulation of the actin cytoskeleton, especially in the organization of growth factor-induced focal adhesions and stress fiber formation (39, 42, 45). So far, the involvement of RhoB in the organization of actin cytoskeleton has not been demonstrated convincingly. Therefore, we were interested to see whether the UV induced increase in RhoB was accompanied by change in actin cytoskeleton. Cells were UV-irradiated and, thereafter, the actin cytoskeleton was fixed and stained by FITC-phalloidin. In a second approach to identify changes of actin cytoskeleton, e.g. depolymerization of F-actin, we used C. botulinum C2 toxin, that ADP-ribosylates specifically monomeric G-actin, but not F-actin. Neither F-actin staining by FITC-phalloidin nor the specific [35P]ADP-ribosylation of G-actin were changed after UV treatment (not shown). Furthermore, no change in F-actin was detectable after transient transfection of rhoB sense and antisense expression vectors followed by FITC staining (not shown). These data indicate that no major alteration (polymerization or depolymerization) of the actin cytoskeleton had occurred after changing RhoB expression. Overall, these findings indicate that RhoB very likely does not play a crucial role in the regulation of actin microfilaments.

DISCUSSION

In this study we have shown that rhoB, encoding a Ras-related GTP-binding protein, is a novel, immediate-early DNA-damage inducible gene. Similar to c-fos, the rhoB gene can be transcriptionally activated by UV light. Other mutagens such as MNU and cisplatin, as well as serum factors and the protein synthesis inhibitor cycloheximide, also evoked rhoB response. However, cycloheximide which apparently interferes with sig-
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The identification of RhoB as an immediate-early gene indicates that RhoB activity is regulated not only by a GTPase cycle but also on the transcriptional level. The high homology between various Rho proteins (RhoA, RhoB, and RhoC) and their characterization as Ras homologous indicates that RhoB is also involved in signal transduction. Rho proteins are believed to participate in the regulation of the actin cytoskeleton (38–44). This was suggested from the results of microinjection experiments with purified and recombinant RhoA and RhoC protein and from the application of Rho inactivating bacterial ADP-ribosyltransferases. However, because RhoB, but not RhoA and RhoC, has been localized on prelysosomal membranes (47) and only RhoB is induced by mitogens (34), the physiological function of RhoB appears to be distinct from that of RhoA and RhoC. This hypothesis is supported by our finding that only RhoB but not the other rho genes behaved as inducible upon treatment with DNA-damaging agents. Furthermore, UV-stimulated rhoB mRNA and protein expression, as well as transient transfection of rhoB expression vectors were not accompanied by changes in actin cytoskeleton. Thus, a major role of RhoB in the formation of actin microfilaments determining cell morphology or adhesion appears to be unlikely. Beside its involvement in cytoskeleton organization, RhoB has additionally been shown to have oncogenic activity (46, 59). In this context it is interesting that very recently RhoB has been suggested to play a role in cell growth regulation and to be necessary for transformation by oncogenic Ras (48).

Summarizing, the GTP-binding protein RhoB which is immediate-early inducible upon genotoxic stress appears to be a candidate for a regulator that directly interferes with early steps of signaling after DNA-damaging treatments. The well known immediate-early inducible proto-oncogenes c-fos and c-jun encode transcription factors that act by trans-activating late responsive genes, some of which may exhibit a protective function (60–63). Another gene product which is involved in cell cycle control and accumulates after UV irradiation is p53 (18, 19). A UV-stimulated increase in p53 is not observed earlier than 3–5 h after UV irradiation (18). Thus, this response appears to occur too late in order to mediate rapid cellular reactions, such as the block of replication that is maximal already 1–2 h after UV irradiation (63). The immediate-early induction of RhoB indicates the existence of a new regulatory pathway which might enable cells to react very rapidly upon induction of DNA damage. It is, to our knowledge, the first evidence for a possible involvement of an inducible GTP-binding protein in the very fast acute response of mammalian cells to environmental stress.

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**Table III**

| Treatment | Induction of rhoB mRNA* |
|-----------|-------------------------|
| UV (log)  | +                      |
| UV (serum starved) | +                     |
| UV (confluent) | +                     |
| MNU  | +                      |
| Cisplatin | +                      |
| Hydroxyurea | +                      |
| H2O2  | 0                      |
| Zn2+  | 0                      |
| FCS   | +                      |
| Dexamethasone | +                   |
| UV medium†  | +                      |
| Ionomycin | 0                      |
| Retinoic acid | 0                      |
| TPA  | 0                      |
| Bt2cAMP | 0                      |
| TPA + Bt2cAMP | 0                     |

* Relative amount of rhoB mRNA in treated cells, as compared with untreated cells. Relative rhoB mRNA was determined in relation to GAPDH mRNA. 0 = no increase (<1.5-fold), + = increase (<2.5-fold), ++ = strong increase (>2.5-fold) of rhoB mRNA.

† Medium of UV-irradiated cells (48 h after irradiation with 30 J/m²).

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**Note:**

Inhibitor genistein whereas UV-mediated rhoB induction was not sensitive. Thus, a regulatory significance of tyrosine kinases in UV response, as deduced from the analysis of the UV induction of c-jun (56), appears to be questionable for rhoB. On the other hand, serum pretreatment reduced the level of a subsequent UV stimulation of rhoB, indicating overlapping of mitogen- and UV-induced signaling of rhoB induction. It is unlikely that the epidermal growth factor receptor is involved in the regulation of rhoB expression by UV because it is activated not earlier than 30–60 min after UV irradiation (57). Cloning of the rhoB gene to analyze its regulatory elements is required in order to clarify its obviously complex regulation. Another kinase recently shown to be activated by UV light is JNK1 kinase (58). The substrate for this kinase has been identified to be c-jun (58). Whether JNK1 kinase and c-jun also interfere with the UV-stimulated expression of rhoB remains to be elucidated.

**Log:** logarithmically growing cells; serum starved, 24 h, 0.5% FCS; confluent 48-h confluent cells; UV, 30 J/m²; MNU, 2 mm; cisplatin, 0.1 µg/ml; hydroxyurea, 2 mm; H2O2, 0.0001%; Zn2+, 50 µM; serum (FCS), 20%; ionomycin, 1 mm; dexamethasone, 2 x 10⁻⁷ M; retinoic acid, 2 x 10⁻⁹ M; Bt2cAMP, 1 mm.
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