Cross-modulation between androgen receptor (AR) and NF-κB/Rel proteins was studied using various androgen- and NF-κB-regulated reporter genes under transient transfection conditions. In COS-1 cells, elevated expression of RelA (p65) repressed AR-mediated transactivation in a dose-dependent manner, whereas NFκB1 (p50), another major member of the NF-κB family, did not influence transactivation. The repression of AR appeared to involve the N-terminal region of the protein between residue 297 and the DNA-binding domain. RelA-mediated transrepression could not be overcome by releasing the amount of AR. Transcriptional interference between RelA and AR was mutual in that cotransfected AR was able to attenuate transactivation by RelA in a dose- and steroid-dependent fashion. An excess of RelA was able to rescue the repression to some extent. Immunological analyses of RelA and AR protein levels indicated that transrepression was not due to reciprocal decrease in their amounts. Neither did AR increase the concentration of IκBα, which can sequester and inactivate RelA. Electrophoretic mobility shift assays using extracts from cotransfected cells and purified recombinant proteins showed that AR and RelA did not significantly influence each other’s DNA binding activity. Nevertheless, protein-protein interaction experiments demonstrated a weak association between AR and RelA. Collectively, these data suggest that the mutual repression in intact cells is due to formation of AR-RelA complexes that are held together by another partner or to competition for a coactivator required for transcription.

Androgen receptor (AR) belongs to the superfamily of ligand-activated transcription factors, the nuclear receptors (1, 2). AR regulates the development, differentiation, and maintenance of male reproductive functions. In addition, androgens are involved in the regulation of other sexually dimorphic processes, ranging from the development of neural tissues to the modulation of immune function. Once bound to androgen, AR acquires a new conformational state, which renders the receptor capable of interacting not only with DNA sequences specified by androgen response elements (AREs) but also with other transcription-regulating proteins. This interaction will eventually result in activation and/or repression of specific gene transcription, depending on the physiological context (3).

The importance of a phenomenon called cross-modulation, unexpected interactions of distinct transcription factors, is illustrated by well documented interactions of AP-1 family members with the nuclear receptors (3–6). Molecular mechanisms that underlie these interactions have mostly remained elusive. Cross-talk between signaling pathways may link processes occurring in different cellular compartments, increase regulatory diversity, and provide opportunities for cell- and tissue-specific responses. AP-1 and NF-κB families of transcription factors regulate an array of gene networks that are induced in response to growth factors, mitogens, tumor promoters, DNA-damaging agents, or oxygen radicals (7, 8). Although NF-κB is a ubiquitous transcription factor, its function plays a central role in cells of the immune system (7). It has been recently reported that one member of the nuclear receptor superfamily, the glucocorticoid receptor (GR), is able to repress NF-κB function probably through a direct physical association that generates transcriptionally inactive complexes (9–11). The transcription properties of AR and GR are in many aspects similar; however, some of their cross-modulatory properties with AP-1 family members are clearly distinguishable (3, 12).

The interleukin-6 (IL-6) gene is one of the many cytokine genes, the regulation of which involves activation of NF-κB (13), and androgens have been reported to inhibit IL-6 production by murine bone marrow-derived stromal cells (14). Additionally, several other genes down-regulated by androgens appear to contain potential NF-κB binding sites in their promoter regions. On the other hand, NF-κB has been implicated in the repression of the AR gene promoter, and an age-dependent increase in NF-κB content in the liver may relate to androgen desensitization of this tissue that occurs during senescence (15). Tumor necrosis factor α, an activator of NF-κB, has been shown to inhibit proliferation of androgen-independent prostate cancer cells, whereas cancer cells devoid of AR were not affected (16), suggesting that NF-κB interferes with the function and/or synthesis of the AR. In the present work, we have investigated cross-modulation between NF-κB and AR by using transactivation assays in intact cells and by examining the possibility that these proteins are physically associated in vitro.

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1 The abbreviations used are: AR, androgen receptor; ARE, androgen response element; CAT, chloramphenicol acetyltransferase; CMV, cytomegalovirus; DBD, DNA-binding domain; EMSA, electrophoretic mobility shift assay; GR, glucocorticoid receptor; GST, glutathione S-transferase; LBD, ligand-binding domain; LUC, luciferase; MMTV, mouse mammary tumor virus; NT, N-terminal region; IL, interleukin; hAR, human AR; rAR, rat AR.

2 J. J. Palvimo, P. Reinikainen, and O. A. Jänne, unpublished observations.
EXPERIMENTAL PROCEDURES

Materials—Human AR (hAR) expression vector (pCMV-hAR) was constructed by replacing the SmaI-KpnI fragment of pCMV-hAR-Qin50 (17) by the respective fragment of pSG5-hAR (18). Rat AR (rAR) deletion mutants rAR641–902, rAR38–296/641–902, and rAR436–408/641–902 were constructed from pSG-RAR using polymerase chain reaction.\(^5\) Expression vectors encoding human RelA (pCMV-p65), NFκB1 (pCMV-p50), and reporter vector pEκBtk-luciferase (LUC) were gifts from Dr. Patrick Baeuerle (Alberts-Ludwig Universita ¨t, Freiburg, Germany) (19). pCMV was obtained from Clontech (Palo Alto, CA). Murine mammary tumor virus promoter LUC construct (pHH-LUC, containing region −203/+105 of the promoter), and pMMTV-CAT containing the MMTV-LTR-driven bacterial chloramphenicol acetyltransferase (CAT) gene was obtained from American Type Culture Collection (ATCC, Rockville, MD). pAREtk-LUC was constructed by inserting the SalI-XhoI fragment of pAREtk-CAT (20) into the polylinker of pGEX-5X-1 vector (Pharmacia Biotech Inc.) (3). pGST-p65Eco encompassing the Rel homology domain of RelA (amino acids 1–282) and pGEX-5X-1 vector (Pharmacia Biotech Inc.) (3). pBH500 (a gift from Dr. Robert J. Matusik, University of Manitoba, Winnipeg, Canada) containing the MMTV-LTR-driven bacterial chloramphenicol acetyltransferase (CAT) gene was obtained from American Type Culture Collection (ATCC, Rockville, MD). pAREtk-LUC was constructed by inserting the SalI-XhoI fragment of pAREtk-CAT (20) into the polylinker of pGEX-5X-1 vector (Pharmacia Biotech Inc.) (3). pGST-p65Eco encompassing the Rel homology domain of RelA (amino acids 1–282) and pGEX-5X-1 vector (Pharmacia Biotech Inc.) (3). Fusion proteins were expressed in JM109 cells and purified (3). [\(^{35}S\)]methionine labeled RelA and NFκB1 proteins was carried out as described (c.f. Fig. 1). Additional deletion of amino acids from 296 to 408 generated a receptor form (rARΔ46–408/Δ641–902 mutant) that retained approximately 50% of the activity of the wild-type rAR in the presence of androgen but that was no longer significantly affected by body. After a 30-min incubation at 22 °C, the cross-linking agent dithio- bis[succinimidylpropionate] was added to a final concentration of 0.1%, and the incubation continued for additional 30 min, essentially under the conditions described by Stein et al. (25). Protein–protein affinity chromatography using GST-NT, GST-DDB, and GST-LBD with [\(^{35}S\)]methionine-labeled RelA and NFκB1 proteins was carried out as described (25), except that incubations were performed at 22 °C and washed at 4 °C. Triton X-100 was omitted from buffers, and the washing buffer contained 200 mM NaCl.

RESULTS

Repression of Androgen Receptor by RelA—Transcriptional cross-modulation between AR and NFκB was studied in COS-1 cells, which are devoid of endogenous androgen receptor. The cells were cotransfected with an AR expression vector and increasing amounts of RelA expression plasmid, together with an androgen-regulated LUC reporter gene driven by two ARESs in front of the thymidine kinase promoter (pAREtk-LUC). Under these conditions, 10 nM testosterone in culture medium increased LUC activity 10-fold over that without androgen (Fig. 1A). Cotransfection of increasing amounts of RelA expression plasmid (pCMV-p65) brought about a consistent, dose-dependent decrease in androgen-induced transactivation. Already low amounts of pCMV-p65 (0.1 μg/10 cm plate) decreased the transactivation by AR over 50%, and higher amounts of RelA (1–2 μg/plate) almost completely blocked the effect of testosterone. RelA (1 μg/plate) influenced only marginally basal LUC activity in the absence of testosterone (data not shown). Interestingly, NFκB1, which is in many aspects homologous to RelA but lacks the transcriptional domain (19), was not able to repress AR-mediated reporter gene activation. Comparable results were obtained with other reporter constructs transactivated by the AR, such as pHH-LUC containing the region −203/+105 of the MMTV promoter (Fig. 1B), the rat probasin gene proximal promoter (Fig. 1C) or pMMTV-CAT (Fig. 1D), indicating that the effect of RelA on AR function was not a feature peculiar to artificial tk-based promoters. Moreover, the function of an AR expression vector driven by the SV40 promoter (pSG5-rAR) instead of the CMV promoter was repressed by RelA in a similar fashion in CV-1 cells, verifying that transrepression by RelA was not due to inhibition of CMV-driven expression vectors or specific for transformed COS-1 cells (Fig. 1D).

When both RelA and NFκB1 (in equal amounts) were transfected together, the effect on AR-mediated transactivation was similar to that of RelA alone (Fig. 1A). The repression by RelA could not be overcome by the addition of excess AR, suggesting that the two proteins competed for coactivator(s) present in limiting amounts in the cell, rather than putative AR-RelA complexes being incapable of transactivation and that the affinity of RelA for this plausible coactivator is higher than that of AR. An alternative explanation is that RelA induces expression of an unknown repressor of AR function, even though there is currently no experimental evidence for the presence of such a repressor.

Domains of AR Mediating the Repression by RelA—The effect of RelA on transactivation mediated by a few mutated receptor forms was examined in CV-1 cells to delineate domains of AR involved in the repression. A receptor mutant devoid of the LBD (rARΔ641–902 mutant) and a form that lacked both LBD and N-terminal amino acids 38–296 (rAR38–296) was tested in transactivation of the reporter construct together with increasing amounts of rAR in CV-1 cells. The repression by RelA was not due to inhibition of CMV-driven expression vectors or specific for transformed COS-1 cells (Fig. 1D).

Electrophoretic Mobility Shift Assay—EMSA with purified recombinant proteins and preparation of whole cell extracts for EMSA were carried out as described previously (3, 22, 24). Nuclei were prepared by homogenization in 10 mM Hepes-KOH (pH 7.5), 420 mM NaCl, 25% (v/v) glycerol, 1.5 mM MgCl\(_2\), 0.2 mM EDTA, 1 mM dithiothreitol, 1 mM phenylmethylsulfonyl fluoride, 10 μg/ml leupeptin, and 4 μg/ml trasylool.

Immunoblotting, Coimmunoprecipitation, and Protein–Protein Interaction Experiments—Whole cell extracts from COS-1 cells were resolved by electrophoresis on polyacrylamide gels under denaturing conditions, proteins transferred onto Immobilon-P membrane (Millipore, Bedford, MA) and processed as described previously (20). Coimmunoprecipitation of in vitro translated proteins (RelA in wheat germ lysate and AR in rabbit reticulocyte lysate) was performed using AR333 antibody\(^6\) raised against purified full-length rAR or a RelA-specific anti-
adding empty pCMV DNA when needed. The total amount of DNA per dish was kept constant by mean internal control gene activities were determined. CAT activities are or plus signs. After a 30-h culture, the cells were harvested, and reporter gene (LUC or CAT) and internal control gene (β-galactosidase) activities were determined. Reporter gene activities are expressed relative to that of AR expression plasmid (1 μg) in the presence of testosterone (100%), and the mean ± S.E. values of three independent experiments are given in percentages. Transactivation by AR in the presence of androgen was 10-, 15-, 60-, and 100-fold in A, B, C, and D, respectively. The total amount of DNA per dish was kept constant by adding empty pCMV DNA when appropriate.

Regions of AR involved in mediating transrepression by RelA. CV-1 cells were transfected with 5 μg of pARE2tk-LUC (Ref. 3; the cryptic AP-1 site is deleted from the vector backbone) and 1 μg of the indicated rat AR expression vectors in the presence (open bars) and the absence (solid bars) of 1 μg of RelA expression plasmid. The cells received 18 h later fresh medium that contained 10 nM testosterone. After a 30-h culture, the cells were harvested, and reporter gene and internal control gene activities were determined. CAT activities are expressed relative to that of rAR expression plasmid (100%), and the mean ± S.E. values of three independent experiments are given in percentages. The total amount of DNA per dish was kept constant by adding empty pCMV DNA when needed. 

Overexpression of RelA attenuates androgen-induced transactivation. A, COS-1 cells were transfected with pARE2tk-LUC (5 μg) and indicated amounts (in μg) of hAR, RelA, and NF-κB1 expression vectors. B, pHH-LUC was used as the reporter in COS-1 cells. C, pPPB1tk-LUC was used as the reporter in COS-1 cells. D, CV-1 cells were transfected with pMMTV-CAT and a rat AR expression plasmid (pSG5-rAR) was used. In all experiments, the cells received 18 h later fresh medium with vehicle or 10 nM testosterone (T) as depicted by minus or plus signs. After a 30-h culture, the cells were harvested, and reporter gene (LUC or CAT) and internal control gene (β-galactosidase) activities were determined. 

**Analysis of RelA, AR, and 1xBo Protein Levels**—One possible explanation for the above findings is that expressed AR and RelA proteins decrease each other’s cellular concentration in COS-1 cells. Overexpression of RelA increased the level of immunoreactive AR, both in whole cell extracts and nuclear extracts (Fig. 4, upper panel, lanes 2 and 3). This is likely due to activation of the CMV promoter by RelA, because CMV promoter-driven β-galactosidase activity (from pCMVβ reporter vector) was also increased 2-fold by overexpression of RelA. Immunoblotting analysis of whole cell and nuclear extracts revealed that immunoreactive RelA protein was not decreased by cotransfected AR (Fig. 4, middle panel, lanes 2 and 3). If anything, the amount of RelA was increased by coexpression of AR, especially in nuclear extracts. Because AR and RelA expression vectors were driven by the CMV promoter and were affected in a similar fashion, RelA responsiveness of these promoters should not complicate interpretation of our data, which is that the mutual transrepression is not due to decreased levels of RelA and AR proteins.

It has been reported that in addition to direct interaction between GR and NF-κB, glucocorticoids are also capable of inducing the 1xBo gene (26, 27), the product of which is an inhibitor of NF-κB. Immunoblotting experiments revealed that ligand-free or ligand-occupied receptors do not influence markedly the concentration of 1xBo protein in COS-1 cells (Fig. 4, bottom panel, lanes 2 and 3). By contrast, cells transfected with RelA had a substantially elevated 1xBo content, especially in whole cell extracts (Fig. 4, bottom panel, lanes 1–3).

**Binding of AR and RelA to Their Cognate DNA Elements**—Transrepression of AR function could derive from its altered DNA binding activity for the NF-κB element. Transfection of COS-1 cells with RelA resulted in the formation of a new NF-κB element-binding complex that migrated somewhat faster than other main complexes present in mock- or AR-transfected cells (Fig. 5A and B, lanes 1–3). Combined expression of RelA and AR did not result in decreased amount of this complex or other complexes either in whole cell extracts or nuclear extracts (Fig. 5, A and B, lanes 2 and 3; cf. lane 1); rather, cotransfection of AR increased NF-κB binding activity in whole cell extracts (Fig. 5A, lanes 2 and 3).
identification of DNA-protein complexes specific for cells transfected with RelA. Several protein complexes were bound to NF-κB element in a specific manner in that they were abolished by excess of cold NF-κB oligomer but not by an AP-1 oligomer (Fig. 5C). A RelA-specific antibody supershifted only the complex that appeared upon transfection with the RelA expression plasmid (Fig. 5C, lane 10), whereas a nonspecific antibody did not have the same effect (Fig. 5C, lane 9). The nature of the other proteins interacting with NF-κB element was not investigated.

We also examined the ability of purified full-length AR and its DBD to interfere with DNA binding of the Rel homology domain of RelA. Incubations were performed under the buffer conditions that we have previously used to demonstrate inhibition of c-Jun/AP-1 element interaction by AR (3). Preincubation of increasing amounts of AR or AR-DBD with RelA decreased slightly DNA binding of the latter protein (Fig. 6, lanes 3–8); this decrease was maximally 25% with the highest amount of rAR protein used. The Rel homology domain did not modify binding of full-length AR or AR-DBD to AREs (Fig. 6, lanes 10–12 and 14–16), further testifying for a lack of significant interaction between RelA and AR proteins. The same result was obtained when in vitro translated full-length AR and RelA proteins were used (data not shown). It is also worth emphasizing that RelA was not able to bind to a consensus ARE or vice versa; AR did not recognize the NF-κB element (Fig. 6, lanes 2, 9, and 13).

Analysis of Protein-Protein Interaction—A direct interaction between AR and RelA is still a plausible mechanism to explain our findings. To examine whether RelA and AR indeed associate in the absence of specific DNA elements, as suggested for the GR (9–11), protein-protein affinity chromatography and communoprecipitation experiments were performed. [35S-Labeled RelA or NFκB1 proteins were tested for their ability to bind to purified GST fusion proteins containing various domains of AR. GST-NT, GST-DBD, and GST-LBD were immobilized onto glutathione-Sepharose matrix, incubated separately with labeled RelA and NFκB1, and bound proteins were analyzed by polyacrylamide electrophoresis under denaturing conditions. The DNA-binding domain of AR showed weak association with both RelA and NFκB1 (Fig. 7), but only low amounts (0.1%) of the input proteins were recovered as complexes. NFκB1 that had no effect in cotransfection assays bound somewhat better to GST-DBD than RelA, whereas RelA but not NFκB1 showed weak association with GST-NT. No binding of a control protein, [35S-labeled luciferase, was observed to any of the GST matrices used in these experiments.5 We could not detect specific association between AR and RelA by communoprecipitation, even in the presence of the protein cross-linking agent dithiothreitol(succinimidylpropionate).5 Taken together, these results question the formation of stable complexes between RelA and AR in vitro but do not necessarily exclude the possibility that some AR-RelA interactions occur in intact cells.

**DISCUSSION**

We have shown in the present work for the first time that AR-mediated transactivation can be markedly repressed by another transcription factor, RelA. Our data on transrepression between RelA and AR are similar to those recently re-
coexpression of AR with RelA does not decrease cellular NF-kB element binding activity. EMSA experiments were performed using the protein samples corresponding to extracts in Fig. 4. A, aliquots of whole cell extracts (15 μg) were incubated with 32P-labeled NF-κB element oligomer in the presence or the absence of 100-fold molar excess of competing cold oligomer as depicted by plus and minus signs (verifying the specificity of DNA-protein complexes) and electrophoresed on 4% polyacrylamide gels under the conditions described previously (24). B, aliquots of nuclear extracts (10 μg). Lane 1, RelA alone in the presence of testosterone; lane 2, RelA and hAR in the absence of testosterone; lane 3, RelA and hAR in the presence of testosterone; lane 4, hAR alone in the presence of testosterone; and lane 5, empty pCMV vector. C, identification of DNA-protein complexes specific for RelA-transfected cells. Lanes 1–5, nuclear proteins from cells transfected with pCMV-hAR; lanes 6–10, nuclear proteins from cells transfected with the RelA expression vector pCMV-p65. The extracts (10 μg of protein) were incubated in the presence or the absence of 100-fold molar excess of AP-1 or NF-κB oligomers or RelA-specific antibody (0.5 μg) or control IgG as depicted by plus and minus signs.

FIG. 5. Analysis of RelA and AR interaction by EMSA. Purified GST-p65Eco protein encompassing the Rel homology domain (20 nM, lanes 1–5), full-length rAR (20 μM, lanes 6–12), or histidine-tagged H6-DBD of hAR (80 nM, lanes 13–16) were preincubated for 1 h at 4 °C with the following additions: rAR storage buffer alone (lane 3); 8 and 40 nM rAR (lanes 4 and 5); H6-DBD storage buffer alone (lane 6); 400 and 1600 nM H6-DBD (lanes 7 and 8); GST-p65Eco storage buffer alone (lanes 10 and 14); and 10 and 100 nM GST-p65Eco (lanes 11, 12, 15, and 16). After the preincubation, a labeled NF-κB element (lanes 1, 3–9, and 13) or ARE (lanes 2, 10–12, and 14–16) were added, and the incubation continued for 1 h. Protein-DNA complexes were separated from free DNA by EMSA (3). Origins of the fast-migrating complexes in lanes 2, 7, and 8 are not known.

FIG. 6. Interaction of RelA and NFκB1 with various functional domains of AR. In vitro translated 35S-labeled RelA was incubated with the following fusion proteins: GST alone (lane 2), GST-DBD (lane 3), GST-LBD in the presence of 100 nM testosterone (lane 4), and GST-NT (lane 5). The fusion proteins were adsorbed to glutathione-Sepharose, mixed with labeled RelA protein, and incubated, and the matrix was washed essentially as described by Stein et al. (28). Lanes 7–10 show results of identical experiments in which 35S-labeled NFκB1 protein was incubated with the following reagents: GST alone (lane 7), GST-DBD (lane 8), GST-LBD (lane 9), and GST-NT (lane 10).

Androgen Receptor-RelA Interference

Androgen and that regions other than LBD of AR appear to participate in transrepression.

Direct interaction is not the only mechanism for the GR-mediated repression of NF-κB, because glucocorticoids also induce expression of the IκBα gene (26, 27), the product of which is an inhibitor of NF-κB. Increased levels of IκBα, in turn, reduce the amount of active NF-κB that is able to translocate to the nucleus. In contrast to the situation with GR, our results indicate that AR does not affect IκBα protein level, and therefore, the mechanism of AR-mediated repression should not involve inhibition of RelA translocation to nucleus, a fact that was also verified by immunoblotting experiments. The observation that RelA itself increased IκBα levels is in accord with the recent reports showing the existence of an autoregulatory loop between RelA and IκBα (30, 31).

Although RelA and NFκB1 share a conserved Rel homology domain needed for DNA binding and dimerization, only RelA was capable of repressing AR-mediated gene activation. The C-terminal parts of RelA and NFκB1 differ significantly from each other (7, 8, 19). RelA contains a potent activation domain in its C terminus, which enables it to transactivate as both homo- and heterodimers, whereas NFκB1 is devoid of transcription activation domain and thus inactive as a homodimer.
The transactivation domain of RelA has been recently shown to contact some general transcription factors, such as TFIIB and TATA-binding protein, and functionally interact with them (32, 33). However, overexpression of TATA-binding protein in COS-1 cells does not affect androgen-induced transactivation or rescue the repression of AR by RelA. In addition to the general transcription factors, coactivators such as PC1 have been reported to activate RelA-dependent transcription (33). In view of this, it tempting to speculate that mutual repression of transactivation by AR and RelA is due to their competition for PC1-like coactivators that are utilized by both transcription activators. Alternatively, a coactivator may mediate or stabilize complex formation between RelA and AR in intact cells. This latter alternative is supported by our findings (Fig. 3B) that mutual transcriptional interference also occurred when 10-fold lower amounts of RelA and AR expression plasmids were used in transfections.

Our findings offer a mechanistic explanation for the AR-mediated down-regulation of IL-6 gene expression (14), which is inhibition of transcriptional activation of this gene by NF-κB. In the case of estrogen receptor, transcription factor C/EBPβ is also involved in the repression of the IL-6 gene (21). It remains to be elucidated whether C/EBPβ plays a role in the transrepression of this gene by AR. NF-κB has been implicated in the negative regulation of the rat AR gene promoter (15). As a consequence, the age-dependent increase in NF-κB activity could, at least in part, explain androgen desensitization of the liver of aged rats owing to a decreased receptor gene transcription (15). Our results offer an alternative mechanism for attenuated androgen responsiveness with increasing age, in that age-related elevation in RelA (NF-κB) content could interfere with AR-mediated transactivation events at large, rather than being specific for the AR gene promoter. Finally, the results from this work add another piece of evidence to the notion that down-regulation of gene expression by the androgen receptor occurs via mechanisms other than those involving “negative” DNA elements.

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