We investigated the intracellular events involved in the 3,3',5-triiodo-L-thyronine (T₃)-induced accumulation in acetylcholinesterase (AChE) activity in neuroblastoma cells (neuro-2a) that overexpress the human thyroid receptor β1 (hTRβ1). Treatment of these cells with T₃ increased AChE activity and its mRNAs after a lag period of 24-48 h, and these levels increased through stabilization of the transcripts by T₃. T₃ had no effect on the transcriptional rate or processing of AChE transcripts. The protein kinase inhibitor H7 inhibited T₃-induced accumulation in AChE activity and its mRNAs, whereas okadaic acid (a potent inhibitor of phosphatases 1 and 2A) potentiated the effect of T₃. Okadaic acid and H7 have no effect on the binding of hTRβ1 to T₃ or the transcriptional rate of the AChE gene. Finally, treatment of cells with T₃ stimulated cytosolic serine/threonine, but not tyrosine kinase, activities. The time course analysis reveals that the increase in serine/threonine activity precedes the effect of T₃ on AChE mRNAs. These results suggest that activation of a serine/threonine protein kinase pathway might be a link between nuclear thyroid hormone receptor activation and stabilization of AChE mRNA.

Acetylcholinesterase (AChE; EC 3.1.1.7) is responsible for the hydrolysis of acetylcholine at peripheral and central cholinergic synapses. This enzyme is encoded by a single gene in those vertebrate species examined to date, and the structural diversity in gene products arises from alternative mRNA processing and post-translational associations of catalytic and structural subunits (1–5).

Several lines of evidence suggest that thyroid hormones are involved in regulating AChE activity in neural cells: AChE activity is decreased in the brains of neonate thyroidectomized rats (6–8), and triiodo-L-thyronine (T₃) increases AChE activity in primary neuronal cultures initiated from fetal rat brains (9). Much less is known about the molecular basis for hormone control. It is widely accepted that thyroid hormones exert their actions via nuclear receptors, which regulate the transcription of thyroid hormone-responsive genes. Three forms of thyroid receptors have been identified in neural cells: TRα1, TRβ1, and TRβ2 (for review, see Ref. 10). Recently, we showed that T₃ increases the activity of AChE and its mRNAs in neuro-2a cells, which specifically overexpress human thyroid receptor β1 (hTRβ1), indicating that the control of AChE gene expression by T₃ occurs at a pretranslational step (11). Furthermore, the lag time of several hours following hormone administration suggests that this effect is likely to be indirect, mediated by unknown factors.

Phosphorylation events regulate AChE activity. 1-(5-Isoquinolylsulfonyl)-2-methylpiperazine (H7), a potent inhibitor of protein kinase, stimulates AChE activity in neuro-2a cells (12). Phosphorylation events also participate in thyroid hormone action. Continued protein phosphorylation is required for stimulation of transcription of the lipogenic genes by T₃ (13), and transient transfection assays showed that phosphorylation of TR α isoforms increases their transcriptional activity (14, 15). Phosphorylation of the TRs could lead to a change in either hormone binding or DNA binding of the receptor (16). Another possibility is that the target for the phosphorylation events may be one or more of the auxiliary proteins or coactivators that are important in T₃-dependent transcription (17, 18). Alternatively, phosphorylation could mediate the post-transcriptional effects of thyroid hormones. Thyroid hormones regulate the turnover of numerous mRNAs (19–23). Most of these effects are typically blocked by inhibitors of protein synthesis, suggesting the possible involvement of one or more components that influence the degradative pathway for specific mRNAs. Phosphorylation events also participate in the control of mRNA turnover. The turnover of several mRNAs is regulated by agents that activate the protein kinase C or A pathway either directly (such as phorbol esters or cAMP) or indirectly (such as via receptor activation) (24–31). It is therefore possible that activation of a protein kinase pathway might be the link between thyroid receptor activation and mRNA turnover. However, there is no example of such a mechanism for thyroid hormones.

In the present study, we investigated the mechanism of regulation of AChE gene expression by T₃ using neuro-2a cells that overexpress hTRβ1 as a model. Our results suggest that activation of a serine/threonine protein kinase pathway may be the link between thyroid receptor activation and mRNA turnover. However, there is no example of such a mechanism for thyroid hormones.

**EXPERIMENTAL PROCEDURES**

**Tissue Culture—Neuro-2a cells that overexpress hTRβ1 were cultured in Dulbecco’s modified Eagle’s medium and F-12 in a 1:1 (v/v) ratio, supplemented with 10% fetal calf serum as described previously (11). The serum was depleted of thyroid hormones according to the procedure of Samuels et al. (32). Cells were grown at 37 °C in a humidified atmosphere of 5% CO₂, 95% air.**

**RNA Preparation and Northern Blot Hybridization—Total RNA was prepared by the guanidinium thiocyanate procedure (33), and poly(A)⁺ mRNA was prepared as described by Hartmann et al. (34). RNA concentrations were determined spectrophotometrically by measuring the A₂₆₀.**

**DNA Analysis—**For Northern blot, 6 μg of poly(A)⁺ mRNA or 15 μg of total RNA was separated on 1.5% agarose gels containing 0.66 M formaldehyde and transferred to nitrocellulose membranes as described (11).
Specific oligonucleotides (nucleotides 1779–1824 and 1936–1979 of mouse AChE cDNA) were synthesized, 3'-end-labeled, and used to probe endogenous AChE mRNAs as described (11). The recovery of RNAs and poly(A)+ was determined by hybridization with either a 3'-end-labeled 18 S rRNA-specific oligonucleotide (35) or a 32P-labeled cDNA cyclphilin probe, an unrelated gene constitutively expressed in cells (36), respectively. All values were normalized to cyclophilin or 18 S rRNA signal. Quantitation of relative RNA levels of autoradiograms was determined by densitometry (Biogame-Visage 110S from Millipore Corp., Ann Arbor, MI).

Nuclear Run-on Transcription Analysis—Approximately 1 x 107 cells were washed three times in ice-cold phosphate-buffered saline, collected in 1 ml of the same buffer, and centrifuged for 10 min at 500 x g. Cells were resuspended in lysis buffer (10 mM Tris-HCl, pH 7.4, 3 mM CaCl2, and 2 mM MgCl2) containing 0.5% Nonidet P-40. Nuclei were prepared as described previously (37) and resuspended in 200 μl of 50 mM Tris-HCl, pH 7.4, 40% glycerol, 5 mM MgCl2, and 0.1 mM EDTA, frozen in liquid nitrogen, and stored at –70 °C.

Transcription assays were performed essentially as described (38). Nuclei were resuspended in buffer containing 250 μCi of [α-32P]UTP (Amersham Corp.) and 1 mM each of ATP, CTP, and GTP and 5 mM dithiothreitol. For experiments with α-amanitin (from Sigma), α-amanitin (2 μg/ml) was added to medium prior to initiating synthesis. After incubation at 30 °C for 30 min, the radiolabeled RNAs were isolated by ethanol precipitation after DNase I and proteinase K treatment. Total incorporation of [α-32P]UTP into RNAs was 3–6 x 106 cpm/107 nuclei.

The radiolabeled RNAs were hybridized to dot blots containing 5 μg of plasmid DNA. Plasmids containing the 2.1-kb AChE insert and a control plasmid (pcDNA3) were linearized with PstI and HindIII. In previous experiments, we showed that the transcription factor NGFI-A (39) (also known as zif268 (40), Krox-24 (41), Erg-1 (42), T528 (43), and CEF-5 (44)) is transcriptionally regulated by T3 in these cells (2). Thus, a plasmid containing the 3.2-kb NGFI-A insert (from ATCC) was used as positive control. 2 x 106 cpm were hybridized to the blots in 10 mM Tris-HCl, pH 7.4, 0.2% SDS, 10 mM EDTA, 300 mM NaCl, 1 x Denhardt’s solution, and 200 mg/ml Escherichia coli tRNA. Blots were incubated for 36 h at 65 °C, washed in 2 x SSC and 0.1% SDS at room temperature, and then washed at high stringency (0.2 x SSC and 0.1% SDS at 65 °C) for 1 h. 5,6-Dichloro-1-β-d-ribofuransylbenzimidazol (DRB) Inhibition of Transcription—Neuro-2a cells were maintained for 3 days in cultured media with and without T3 (30 nm). On day 3, cells were extensively washed with fresh media and treated for the specified times with 20 μg/ml DRB, which was dissolved (4 mg/ml) in ethanol and diluted 1/200 in cultured media. The same volume of ethanol was added to controls. Total RNA was isolated and used for Northern blot. Blots were normalized to the 18 S rRNA.

Determination of AChE Activity—AChE activity was determined by colorimetrically measuring the rate of hydrolysis of acetylthiocholine (45). Protein determinations were performed by the method of Lowry et al. (46).

Determination of T3 Binding Capacity—Cells were grown for 24 h in the absence or presence of either H7 (25 μM) or okadaic acid (OA) (10 μM). Then 1106 cpm–labeled T3 was added to the cultures, and cells were further incubated for 3 h at 37 °C. After incubation, the nuclei were isolated, and the radioactivity was determined as described (37). Non-specific binding was determined by incubating cells with a 1000-fold excess of nonradioactive T3. DNA was assayed by the method of Burton (47).

Measurement of Kinase Activity—Cells grown in the presence or absence of T3 (30 nm) were homogenized in 10 mM Tris-HCl, pH 7.4, 15 mM MgCl2, 10 mM EDTA, 1 mM dithiothreitol, 17 μg/ml phenylmethylsulfonyl fluoride, 5 μg/ml leupeptin, 5 μg/ml antipain, and 5 μg/ml pepstatin A. Homogenates were centrifuged at 10,000 x g for 10 min at 4 °C. The soluble fraction was centrifuged at 100,000 x g for 60 min, and the resulting supernatants were stored at –80 °C. Aliquots (5 μg of protein) were mixed with 50 μl Hepses buffer, pH 7.4, containing 40 mM MgCl2, and 30 μg of myelin basic protein (MBP) or 75 μg of a copolymer glutamic acid and tyrosine (41), ptdy(Glu/Tyr) (48). The reaction was initiated by adding [γ-32P]ATP (final concentration 25 μM, 32 μCi/μmol). After 30 min, the reaction was stopped by spotting aliquots on chromatography paper squares (Whatman No. 3MM, 2 x 2), which were then placed in a 10% trichloroacetic acid bath containing 10 mM of sodium pyrophosphate for 60 min at 4 °C. The squares were washed twice for 30 min at room temperature, rinsed with 100% ethanol, and air-dried. Radioactivity was determined by liquid scintillation counting. Incubation in the absence of substrate was used as control. Control values were subtracted from the total counts.

RESULTS

AChE Activity and mRNA Levels Are Induced by T3—Neuroblastoma cells that overexpress the thyroid receptor β were used to analyze the effect of T3 on AChE gene expression. The time course analysis of the effect of T3 on AChE activity is shown in Fig. 1 (upper panel). There was a significant increase in AChE activity after 24 h of treatment with 30 nm T3 to 180% of the control value, with a maximal effect occurring after 48 h of treatment. The increase in AChE activity was paralleled by an increase in AChE mRNA, as determined by Northern analysis (Fig. 1, lower panel). Site-specific oligonucleotides recognized a major 2.4-kb mRNA species and a minor 3.2-kb mRNA species, which are the expected size of mouse AChE mRNAs (11, 49). Treatment of cells with T3 (30 nm) significantly increased AChE mRNA after 24 h of treatment; maximal increase occurred after 48 h of treatment. The ratio of the 2.4/
3.2-kb AChE mRNA was not significantly changed by T3 treatment (not shown). Because the 2.4- and 3.2-kb mRNAs are alternatively spliced AChE mRNAs (49, 50), our finding indicates that T3 had no effect on the process that altered the size of AChE mRNAs.

T3 Stabilizes AChE mRNA—To determine whether the T3-induced accumulation in AChE mRNA resulted from an increased rate of transcription, we performed nuclear run-on measurements with nuclei isolated from neuro-2a cells maintained for 1 and 24 h in the presence of T3. Fig. 2 shows densitometric analysis of the data from two different experiments normalized to the cyclophilin signal. The transcription rate for NGFI-A gene showed a 2-fold increase (633 ± 41 and 1384 ± 260 arbitrary units, for control and T3-treated cells, respectively). The enhancement of the rate of transcription for NGFI-A gene was in accord with the changes in mRNA levels detected by Northern blot (not shown). No change was detected in the transcriptional rate for c-jun gene or the AChE gene in cells treated for 24 h with T3. The AChE signal is specific since it was completely abolished by α-amanitin at a concentration (2 μg/ml) that specifically inhibited RNA polymerase II (38) (Fig. 2, upper panel). Similar results were obtained with cells treated for 1 h with T3 (not shown), indicating that the effect of T3 on AChE mRNAs is exerted at a post-transcriptional step.

The adenosine analog DRB specifically inhibits RNA polymerase II (51, 52), and its capacity to block the transcription of AChE gene has rendered it useful in determining the half-life of AChE mRNAs (53). In the absence of T3, the AChE mRNA level decayed significantly after cells were treated with DRB (Fig. 3). The calculated AChE mRNA half-life is about 4–5 h, which is in agreement with the AChE mRNA half-life reported in muscle cells (53). In cells pretreated with T3 for 72 h, no decrease in AChE mRNA levels was evident over the next 24 h, suggesting the presence of a very stable mRNA following a global block of transcription (Fig. 3). Furthermore, the addition of OA (10 nM) prevented the decrease in AChE mRNA levels observed 48 h after DRB treatment, suggesting that OA further stabilizes the mRNAs. The addition of DRB 15 min before treatment with T3 completely inhibited T3’s ability to increase AChE mRNA levels (Fig. 4).

Phosphorylation Events Mediate the T3-induced Accumulation of AChE Activity and Its mRNA—OA, a selective inhibitor of serine/threonine protein phosphatases 1 and 2A (54), was used to determine whether phosphorylation on serine/threonine residues is involved in T3-induced AChE activity. Treatment of cells with OA for 24 h increased the activity of AChE in a dose-dependent manner in cultures grown in the absence of T3 (Fig. 5, upper panel). The simultaneous addition of T3 (30 nM) and OA (5–20 nM) to the culture caused a synergistic effect. At 5 nM, OA had no significant effect on AChE activity in the absence of T3, whereas it potentiated the effect of T3 on AChE activity. These results suggest that T3 regulated a protein kinase that increases phosphorylation on serine/threonine res-
To test this possibility, we examined the effect of protein kinase inhibitors (H7, calphostin C, and N-(2-guanidinoethyl)-5-isoquinolinesulfonamide (HA1004)) on the T3-induced accumulation in AChE activity. H7 (5–50 μM) had no effect on AChE activity in cells grown in the absence of T3 (not shown). The increase in AChE activity caused by the addition of T3 for 24 h was inhibited by 90% when H7 (50 μM) was added for the same period of time as T3 (Fig. 5, lower panel). Fifty percent of maximal inhibition was achieved at about 10–15 μM H7. On the other hand, calphostin C (100–400 nM, a specific inhibitor of protein kinase C) and HA1004 (a specific inhibitor of protein kinase A) had no effect on the T3-induced accumulation of AChE activity (Table I). Furthermore, treatment of cells with various activators (8-bromo-cAMP, phorbol 12-myristate 13-acetate) did not affect basal AChE activity or T3-induced accumulation in AChE activity (Table I).

To determine whether phosphorylation mediates the effect of T3 on AChE mRNA, we examined the effect of H7 and OA on the T3-induced accumulation of AChE mRNA. H7 (20 μM), added coincidentally with T3, caused an almost 90% inhibition of the T3-induced accumulation of AChE mRNA after 24 h, whereas treatment of cells with OA for 24 h potentiated the effect of T3 on AChE mRNA levels (not shown). A transcriptional effect of H7 and OA on AChE gene was excluded by run-on transcriptional analysis (not shown).

To determine whether T3 activates a protein kinase pathway, serine/threonine and tyrosine kinase activities were measured in the cytosolic fractions prepared from treated and untreated cells using MBP and poly(Glu/Tyr) as substrates, respectively. As shown in Table II, treatment of cells with T3 (30 nM) induced a 1.5- and 2.1-fold increase in serine/threonine protein kinase activities after 16 and 24 h of treatment, respectively. Phosphoamino acid analysis showed that MBP was phosphorylated predominantly on serine and, to a lesser extent, on threonine residues (not shown). No significant effect of T3 was observed on phosphorylation of poly(Glu/Tyr) (183 ± 23 and 190 ± 15 for untreated and cells treated for 24 h with T3, respectively). Furthermore, treatment of cells with genistein, a specific inhibitor of tyrosine kinase, had no effect on the T3-induced serine/threonine protein kinase activity (not shown), indicating that the effect of T3 on serine/threonine protein activity is not mediated by a tyrosine kinase.

T3 Binding Capacity—To exclude an effect of H7 and OA on the binding of T3 to its receptors, H7 and OA were added for 24 h to neuro-2a cells and then T3 binding capacity was measured in nuclei isolated from intact cells. H7 (25 μM) and OA (10 nM) had no effect on T3 binding capacity (control, 1967 ± 221; H7, 1966 ± 548; OA, 1742 ± 200 fmol/mg DNA; mean ± S.D., n = 3), indicating that the effect of H7 and OA was not caused by a change in T3 binding to its receptors.

FIG. 4. DRB inhibits the T3-induced accumulation of AChE mRNA. Neuro-2a cells were cultured for 3 days in the absence of T3. At day 3, DRB (20 μg/ml) was added to the medium 15 min before treatment of cells with 30 nM T3. The cells were harvested after an additional 48 h and total RNA isolated and analyzed as described in the legend to Fig. 3.

FIG. 5. Effect of H7 and OA on the T3-induced accumulation in AChE activity. Neuro-2a cells were cultured for 3 days in the absence of T3. On day 3, the medium was changed to one of the same composition plus or minus T3 (30 nM) and (or) either H7 or OA at the indicated concentration. AChE activity was determined 48 h latter. Upper panel, effect of okadaic acid on AChE activity; open bars, control; dashed bars, T3-treated cells. Lower panel, effect of H7 on the T3-induced accumulation in AChE activity. Results are expressed as nmol of AChE hydrolyzed per min/mg of protein and represent the mean ± S.E. of three separate experiments.

DISCUSSION

In neuro-2a cells that overexpress hTRβ1, we previously showed that the effect of T3 on AChE gene expression is independent of its antiproliferative effect, indicating that both effects are regulated by distinct mechanisms (11).

We show here that the T3-induced accumulation in AChE activity and its mRNA result from stabilization of the mRNAs. No effect of T3 was observed on the transcriptional rate or processing of AChE mRNA. Although we previously reported that the increase in 3.2-kb mRNAs was much greater than the increase of 2.4-kb mRNAs after T3 treatment (11), this result was not confirmed in the present study, in which we analyzed a larger number of Northern blots. It is likely that nuclear thyroid receptors indirectly participate in regulated AChE
mRNA turnover, because they are required for the transcriptional regulation of one or more components that influence the degradative pathway of AChE mRNAs. Consistent with this point is the suppression of the T3-induced accumulation in AChE mRNAs after a global block of transcription. The protein kinase inhibitor H7 blocks the ability of T3 to increase AChE activity and its mRNAs. H7 had no effect on AChE activity, AChE mRNA levels, or the rate of AChE gene transcription in the absence of T3 which suggests that H7 selectively inhibits the increase in AChE mRNA induced by T3. At 500 nM, however, H7 increases AChE activity in neuro-2a cells (12). Isooxindolium sulfonamide derivatives vary with respect to the affinity with which they bind to different protein kinases (55). In our study, half-maximal inhibition of the activity of AChE enzyme occurs at about 10–15 μM H7. This concentration of H7 is slightly higher than the Ki values of protein kinase C (6 μM). It is likely that at higher concentration, H7 may inhibit other protein kinases that are also involved in the regulation of AChE enzyme activity. In contrast, OA potentiated the effect of T3 on AChE activity and its mRNA. The finding that OA had no effect on the transcription of the AChE gene indicates that OA selectively potentiates the effect of T3 at a post-transcriptional step. This effect was observed at a concentration of 5 nm OA, which is in the range of the concentration necessary to inhibit protein phosphatases 1 and 2A (0.1–10 nm) (54). At this concentration, OA does not inhibit protein phosphatase 2B and 2C, protein-tyrosine phosphatases, acid and alkaline phosphatase (54), indicating that OA selectively inhibits serine/threonine phosphatases 1 and 2A. Furthermore, we showed that T3 stimulated cytosolic serine/threonine but not protein tyrosine kinase activities. The time course analysis revealed that the effect of T3 on serine/threonine protein kinase activity precedes its effect on AChE mRNAs, supporting the hypothesis that activation of a serine/threonine protein kinase pathway might be the intermediate between thyroid receptor activation and stabilization of the AChE mRNA. Attempts were made to identify the serine/threonine kinase(s). However, our study suggests that the endogenous serine/threonine kinase(s) involved in the stabilization of AChE mRNAs did not have specificities similar to those of cyclic AMP-dependent protein kinase, protein kinase C, phosphorylase kinase, glycogen synthetase kinase 3, and casein kinases I and II.

Our working hypothesis is that T3 activates a serine/threonine protein kinase, which in turn causes the stabilization of AChE mRNAs. The mechanisms by which protein phosphorylation controls the stability of AChE mRNA are unknown. The binding activity of an inducible stabilizing factor that recognizes the AU-rich motif in the 3′-untranslated region of granulocyte-macrophage colony-stimulating factor is dependent on its phosphorylation state (56). It is therefore possible that phosphorylation of cytosolic turnover factor(s) could influence their binding activity to specific sequence in AChE mRNAs, which in turn causes the stabilization of AChE transcripts.

In conclusion, the present study provides the first evidence that activation of the protein kinase pathway might be the linkage between nuclear thyroid receptor activation and a post-transcriptional effect of thyroid hormones.

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