of thyroid hormone are thought to be initiated by the selective binding of 3,5,3'-triiodothyronine (T3) to these receptors, the inability of the adult brain to respond to thyroid hormone remains poorly understood.

In most animals the bioactive form of thyroid hormone, T3, is derived from the hormone-catalyzed 5'-deiodination of T4, the major secretory product of the thyroid gland (6). This enzyme-catalyzed bioactivation of thyroid hormone accounts for >75% of the T3 found in the circulation (7, 8). The brain differs from other hormone-responsive tissues in that it relies almost exclusively on intracellular conversion of T3 to T2 rather than the circulation, as its source of bioactive T3 (6-8). Interestingly, the activity of type II iodothyronine 5'-deiodinase, the brain enzyme catalyzing this reaction, shows rapid 5- to 10-fold increases after thyroidecotomy and equally rapid decrements with hormone replacement (9-11). This ability of the brain to modulate type II iodothyronine 5'-deiodinase content and thus intracellular T3 production appears to be a homeostatic mechanism to preserve intracerebral T3 levels within narrow limits and is likely to play a role in preventing pathophysiological changes in cerebrocortical metabolism in hyper- or hypothyroidism (10).

The mechanism(s) by which thyroid hormone regulates levels of this plasma membrane-bound enzyme in the brain have been studied both in vivo and in cell culture and do not appear to involve the classical nuclear receptor. Comparison of the ability of individual iodothyronines to diminish cerebrocortical type II iodothyronine 5'-deiodinase activity in hypothaloid rats showed T3 >> rT3 >> T3 (10, 11), in sharp contrast to the rank order of potency for binding to the nuclear T3 receptor (T3 >> T3 > rT3). Thyroid hormone-induced increases in enzyme inactivation/degradation are unaffected by inhibitors of transcription or translation and can be accounted for by a selective increase in enzyme degradation (12). Comparable results have been obtained in cell culture models using dispersed fetal (13) or neonatal rat brain (14, 15), the neuroblastoma cell line NB41A3 (16), and the Bt-CAMP-stimulated astroglial cell (17, 18).

The cellular events mediating this thyroid hormone response remain to be established. Ligand-induced enzyme inhibition...
activation has been proposed to account for the ability of subcutaneous and competitive enzyme inhibitors to decrease type II iodothyronine 5'-deiodinase activity in NB41A3 cells (18), although the molecular mechanism(s) for this inactivation remains undefined. We have recently shown that the T3-dependent increase in enzyme inactivation is energy-dependent and that cytochalasins specifically block enzyme loss (18). In this study, we determined the effect of thyroid hormone on actin content and microfilament organization in astrocytes and examined some of the interrelationships(s) between the T3-dependent regulation of this short-lived membrane-bound enzyme and the actin cytoskeleton. The results demonstrate that the organization of the actin cytoskeleton in astrocytes is dynamically regulated by thyroid hormone. In addition, T3's influence on the polymerization state of the actin cytoskeleton is temporarily related to the T3-induced changes in type II 5'-deiodinase.

**MATERIALS AND METHODS**

Dulbecco's modified Eagle's medium, antibiotics, Hank's salt solution, glucose, trypsin, and fetal bovine serum (heat-inactivated) were obtained from Gibco. Culture flasks and plasticware were purchased from Nunc. L-tetraiodothyronine (L-T4), L-triiodothyronine (L-T3), bT, cAMP, and 100 nM hydrocortisone for 16 h as described previously. Iodothyronines were induced as described above. At the end of the incubation period, the hormone concentration was reduced by modulating the rate of enzyme degradation or inactivation of type II 5'-deiodinase (18). Iodothyronines were then added for 20 min as indicated, and the cells were fixed for histochemical identification of P-actin.

**Cell Culture Conditions—**Astroglial cells were prepared and grown as described previously (17). Cultures were fed every 2-3 days and were subcultured (20-30 x 10^6 cells/cm^2) every 7-10 days; in all experiments, cells were used between the 2nd and 6th passage.

Cytoskeleton-Total actin and filamentous (F-) actin were determined by the method of Weeke and Orskov (19) and purified to >95% as described previously (17, 18) with 35S as the major contaminant. Pregnant Sprague-Dawley rats were bred in the Animal Facilities at the University of Massachusetts or the Universite de Geneve. All other reagents were of the highest purity commercially available.

**Cytoskeleton Localization—**Cytoskeleton-Total actin and filamentous (F-) actin were determined by the method of Weeke and Orskov (19) and purified to >95% as described previously (17, 18). Cells were stained with rhodamine-phalloidin (Molecular Probes, Inc., Junction City, OR) for 30 min at room temperature. After washing with PBS, coverslips were mounted onto glass slides in 50% glycerol in PBS (pH 7.8) and examined using a Zeiss microscope equipped with an epi-optics UV and standard fluorescein isothiocyanate filter set. Micrographs were taken with high-speed Ektakrome ASA 400 film.

**Kinetics of Enzyme Inactivation—**Steady-state levels of type II 5'-deiodinase were induced as described above. At the start of the experiment, protein synthesis was blocked with 0.1 mM cycloheximide, and triplicate flasks (25 cm^2) of confluent cells were harvested at the times indicated. Type II iodothyronine 5'-deiodinase activity was then measured in cell sonicates as described previously (18).

The time course of T3's effect on type II iodothyronine 5'-deiodinase inactivation was studied by adding 10 nM T3 plus 1 mg/ml BSA (final concentrations) to bT, cAMP-stimulated cells in serum-free medium. Triplicate flasks were harvested at timed intervals and enzyme activity measured in cell sonicates.

**RESULTS**

Thyroid hormone levels in the culture medium were altered by replacing the serum supplement with individual iodothyronines dissolved in the serum albumin fraction (concentration 1 mg/ml in the culture medium). Iodothyronines were added in the presence of BSA to permit the free hormone levels to be measured in cell sonicates prepared in 10 mM HEPES buffer (pH 7.0), 1 mg/ml BSA, 3 mM EDTA, 1 mM MgCl2, 0.1 mM dithiothreitol, and 100 mM sodium citrate, 1 mg/ml SDS at 60 C for 15 min. Nitrocellulose blots were washed at high stringency (30 mg NaCl, 3 mM sodium citrate, 1 mg/ml SDS at 60° C for 15 min) and exposed to Kodak XAR-5 film at -70°C. Mouse β-actin cDNA was kindly provided by Dr. Joseph Majzoub, Harvard Medical School.

Type II iodothyronine 5'-deiodinase activity was determined in glial sonicates prepared from 10 nM HPLC-purified L-T3 and containing 1 mM EDTA and 10 mM dithiothreitol. Enzyme activity was determined by measuring the release of radiolabeled iodide from 2 nM L-[3' or 5' -35S]T3, in the presence of 50 mM dithiothreitol and 1 mM propylthiouracil as described previously (12, 23). Under these experimental conditions, equivalent amounts of iodide and 3,3'-T2 are released. Enzymatic activity is reported as femtomoles of iodide released per h.

**Miscellaneous Methods—**Protein was determined by the method of Bradford (26) using human γ-globulin as the protein standard. DNA was measured by the method of Hill and Whatley (27).
lectively arrest this process (18). Since type II iodothyronine 5'-deiodinase is a short-lived integral membrane protein, associated with neurilemmal membranes (28), then a likely initial step in the degradation pathway is enzyme internalization. To determine whether the steady-state levels and/or the \( t_{1/2} \) of type II iodothyronine 5'-deiodinase are influenced by the cell actin content, we varied the actin content of astrocytes by altering the plating density of the primary dispersions as described by Goldman and Chiu (29). Depicted in Fig. 1A is the effect of initial plating density on type II iodothyronine 5'-deiodinase inactivation. The rate of enzyme disappearance in cycloheximide-blocked cultures was inversely related to the initial plating density, being fastest in cells derived from the lowest initial density and slowest in cells grown from dispersions plated at the highest density.

Shown in Fig. 1B are the effects of increasing the initial plating density on composition of the Triton-insoluble cytoskeleton in astrocytes. As expected, F-actin was most abundant in cells derived from primary dispersions seeded at low density and decreased proportionally as the initial plating density increased. These changes in F-actin content were maintained through three passages indicating the density of the primary dispersion was a prime factor in determining actin levels in astrocytes. In contrast, the intermediate filament proteins, GFAP and vimentin, were much less dependent upon initial plating density with the apparent changes in quantity shown in lanes a-c (Fig. 1B) due to loading of equivalent amounts of protein on the SDS-PAGE gel.

A comparison of the effects of altered actin concentration on type II iodothyronine 5'-deiodinase activity and \( t_{1/2} \) is summarized in Table I. Steady-state enzyme levels increased proportionately from 43 to 123 units/mg protein and the \( t_{1/2} \) increased >3-fold in cells grown from dispersions in which the plating density was increased 20-fold. These increases in type II iodothyronine 5'-deiodinase were due primarily to changes in the inactivation rate since enzyme production rates, calculated from the steady-state enzyme levels and the disappearance rate constants (\( k \)), were relatively unchanged at 349 ± 34 (S.E., \( n = 4 \)) units/mg protein/h in the cells obtained from the four different initial plating densities.

F-actin levels were also inversely related to the initial plating density and remained so for at least three passages, decreasing ~4-fold as the plating density increased 20-fold (Table I). These changes in F-actin paralleled the increase in total cell actin, thus the ratio of F- to G-actin remained constant (data not shown).

**Effects of \( T_4 \) on Astrocyte Actin Content, mRNA, and Composition—**

- Enzyme half-times were determined from linear semilogarithmic plots of the disappearance of type II catalytic activity in cycloheximide-blocked cultures.
- Enzyme half-times were determined from linear semilogarithmic plots of the disappearance of type II catalytic activity in cycloheximide-blocked cultures versus time as described under "Materials and Methods." 
- F-actin content was determined as described under "Materials and Methods" and is expressed as AU/mg cell protein (AU = arbitrary absorbance units). Data are from two representative experiments, and results are presented as means of closely agreeing (+10%) quadruplicate flasks.

| Plating density (cells/cm²) × 10⁶ | Type II iodothyronine 5'-deiodinase activity (units/mg protein) | Steady state \( t_{1/2} \) (min) | F-actin (AU/mg cell protein) |
|---------------------------------|-------------------------------------------------|----------------|-----------------|
| 10                              | 123                                             | 17             | 11 ± 5          |
| 5                               | 99                                              | 11             | 22 ± 7          |
| 1                               | 64                                              | 9              | 37 ± 13         |
| 0.5                             | 43                                              | 4.5            | 49 ± 9          |
| 12                              | 17.6                                            |                |                 |
| 6                               | 12.5                                            |                |                 |
| 0.5                             | 8.36                                            |                |                 |

Fig. 1. Effects of initial plating density on the rate of type II iodothyronine 5'-deiodinase inactivation and the composition of the Triton-insoluble cytoskeleton in brGMP-stimulated astrocytes. A, the rate of type II iodothyronine 5'-deiodinase (5'D-II) inactivation was determined in triplicate (25-cm² flasks) in cells at passage three as described under "Materials and Methods." Data are presented as the percent of the starting enzyme activity of closely agreeing (+10%) triplicate cultures; SF, serum-free culture conditions; FCS, serum containing culture conditions. B, SDS-PAGE of the Triton-insoluble cytoskeleton in astrocytes. Aliquots (15 μg of protein) of the Triton-insoluble pellets obtained from cells at passage three were separated on 12.5% acrylamide slab gels as described under "Materials and Methods." Lane a, cells derived from primary cultures plated initially at 50,000 cells/cm²; lane b, cells plated at 400,000 cells/cm²; lane c, cells plated at 1,200,000 cells/cm².
actin content to control levels (11.4 versus 12.8% of total Triton-insoluble protein). Identical reductions in F-actin content were observed when 5% (v/v) hypothyroid rat serum was substituted for the calf serum in the culture medium (data not shown), indicating that other serum factors had little, if any, influence on F-actin content under these culture conditions. Total cellular actin, GFAP, and vimentin were unaffected by the presence or absence of serum (data not shown).

To examine whether T₄ and/or βcAMP treatment contributed to the changes in F-actin content by stimulating actin transcription, we determined the effects of T₄ and βcAMP on β-actin mRNA levels. As shown in Fig. 2, neither thyroid hormone nor 16 h of βcAMP treatment altered β-actin mRNA levels in astrocytes. Similarly, total actin content in the astrocyte was unaffected by 16 h of T₄ treatment of βcAMP-stimulated cells as judged by Western blot analysis with anti-actin antibodies (230 absorbance units/mg protein versus 280 absorbance units/mg protein). These data show that thyroid hormone had little or no transcriptional control of β-actin gene expression.

**Rapid Effects of Thyroid Hormone on Inactivation/Degradation of Type II 5'-Deiodinase and Actin Polymerization**—Shown in Fig. 3 is the time course for the T₄-dependent decrease of type II 5'-deiodinase in astrocytes. Addition of T₄ (~100 pM “free” hormone) to βcAMP-treated cells grown in serum-free medium resulted in a rapid 50% loss of activity during the first 8 min followed by a progressively slower loss of catalytic activity that plateaued at 35-40% of the serum-free control. The rapid effects of T₄ on the F-actin content of astrocytes are shown in Table II. F-actin levels followed a similar time course to that of type II iodothryonine 5'-deiodinase, with a rapid ~2-fold increase during the first 5-10 min and a plateau after ~10 min of treatment. Proportional decreases in G-actin were observed with no change in total actin content during this short period of T₄ treatment (data not shown). Neither cycloheximide nor actinomycin D pretreatment blocked the T₄-induced increases in F-actin in astrocytes indicating that neither translation nor transcription were required for this thyroid hormone effect.

**Fig. 3. Rapid effects of T₄ on inactivation of type II 5' deiodinase (5'D-II).** Astrocyes were grown in serum-free medium and stimulated as described under “Materials and Methods.” T₄ (final concentration 10 nM in Hank's solution containing 1 mg/ml BSA) was added at the start of the experiment and triplicate flasks of cells harvested at the times indicated. Type II 5'-deiodinating activity was determined in an aliquot of the cell sonicates as described under “Materials and Methods” and the results reported as the percent of starting enzyme activity. Data are reported as the means of closely agreeing (+10%) triplicate determinations.

**Table II**

| Pretreatment | Time (min) | T₄ | F-actin (AU/mg total cell protein) |
|--------------|------------|----|----------------------------------|
| None         | 0          | 6.6 |                                  |
|              | 20         | 7.6 |                                  |
|              | 5          | 10.4|                                  |
|              | 10         | 16.4|                                  |
|              | 20         | 14.3|                                  |
| Cycloheximide| 5          | 14  |                                  |
|              | 10         | 14.6|                                  |
|              | 20         | 18.3|                                  |
| Actinomycin D| 5          | 12.1|                                  |
|              | 10         | 19.4|                                  |
|              | 20         | 15.5|                                  |

**Fig. 2. β-Actin mRNA levels in cultured glial cells.** Confluent primary cultures (100,000 cells/cm² initial plating density) were subcultured at 1 × 10⁶ cells/cm² and grown to confluence. Cells were then treated with 0.5 mM βcAMP, 100 nM hydrocortisone for 16 h to induce type II iodothryonine 5'-deiodinase activity as indicated. 10-µg aliquots of total RNA were separated on a 1.2% agarose/formaldehyde gel, transferred to Duralose, and probed with β-actin 32P-cDNA as described under “Materials and Methods.” Lane 1, 10 µg of RNA from untreated cells grown in serum-containing medium; lane 2, 10 µg of RNA from cells grown in serum-free medium and treated with 1 mM βcAMP and 100 nM hydrocortisone for 16 h.
nucleotides and cytochalasins promote structural reorganization of actin (30), and cytochalasins antagonize the T₄-dependent regulation of type II iodothyronine 5'-deiodinase (18), we examined the effects of thyroid hormone on microfilament organization in astrocytes (Fig. 4). A complex network of actin bundles was present in astrocytes grown in serum-containing medium (Fig. 4a), and this network was markedly reduced in both amount and fiber length in the absence of thyroid hormone (Fig. 4b). T₄ replacement alone was sufficient to restore the organization of the actin cytoskeleton (Fig. 4c) to that observed in control cultures.

The rapid effects of iodothyronine replacement on the appearance of the astrocyte actin cytoskeleton are illustrated in panels d–f of Fig. 4. Cells exposed to 10 nM T₄ for 20 min showed little or no reorganization of their actin cytoskeleton (Fig. 4d), similar to T₃'s lack of effect on enzyme inactivation at these concentrations (10, 18). In contrast, cells treated for only 20 min with either 10 nM T₄ (Fig. 4e) or 10 nM rT₃ (Fig. 4f) showed nearly complete re-polymerization of the actin cytoskeleton, a time course comparable with the hormone-induced changes in the type II iodothyronine 5'-deiodinase inactivation shown in Fig. 3.

**DISCUSSION**

The ability of cytochalasins to block the T₄-dependent inactivation of type II iodothyronine 5'-deiodinase (18) raised the possibility that there was a functional relationship between the organization of the actin cytoskeleton and the inactivation of this membrane-bound enzyme. In this study, we have shown that (i) increases in the cell actin content were accompanied by decreases in the tₑ of type II iodothyronine 5'-deiodinase with no change in the enzyme production rate; (ii) T₄ and rT₃ promoted F-actin formation and increased the rate of enzyme inactivation, whereas T₃ was ineffective; (iii) the time courses for T₄-dependent formation of F-actin and the increase in type II iodothyronine 5'-deiodinase turnover were the same; and (iv) both the T₄-dependent increase in type II iodothyronine 5'-deiodinase inactivation and actin

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**Fig. 4. NBD-phallacidin-stained F-actin in astrocyte cultures.** Astrocytes were grown on coverslips in serum-supplemented medium. Forty-eight hours prior to the staining, the culture medium was changed to serum-supplemented (a), serum-free medium (b), and serum-free medium supplemented with 10 nM T₄ plus 1 mg/ml BSA (c). All cultures were stimulated for 16 h with 1 mM b[cAMP] and 100 nM hydrocortisone, followed by a 1-h incubation with 10 μM colchicine to relax the contracted cell borders. The actin cytoskeleton was stained with NBD-phallacidin as described under “Materials and Methods.” Scale bar = 10 μm. Panels d–f show serum-free cultures treated for 20 min prior to fixation with 10 nM T₃, 10 nM T₄ or 10 nM rT₃, respectively.
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polymerization did not require continued protein synthesis or transcription. These data suggest that the extranuclear T4-dependent modulation of type II 5'-deiodinase depends, in part, upon alterations in the polymerization state of the actin cytoskeleton.

Actin comprises a major fraction of the astrocyte cytoskeleton in vivo (31) and in cultures of immature and mature astrocytes (32). The ability to alter the actin content of cultured astrocytes by changing the initial plating density (29), and the observation of Ciesielski-Treska et al. (32) that a 24-h exposure to beta2cAMP had no effect on total actin content in these cells, allowed us to manipulate actin levels in cells expressing type II iodothyronine 5'-deiodinase. Steady-state enzyme levels decreased and the t1/2 of the enzyme grew progressively shorter as the actin content increased in the cultured cells. Interestingly, changes in the total actin content of glial cells had no effect on the rate of type II iodothyronine 5'-deiodinase synthesis suggesting that alterations in the composition of the cytoskeleton were responsible for the changes in the inactivation/degradation of this enzyme.

A relationship between the T4-dependent modulation of the biological half-life of type II iodothyronine 5'-deiodinase and cell actin levels was further strengthened by examination of thyroid hormone's influence on F-actin content. Cells grown in the absence of thyroid hormone contained ~40% less F-actin than cells grown in presence of T4. Addition of T4 alone normalized F-actin in cells grown in serum-free medium suggesting that thyroid hormone was essential for normal actin polymerization. Importantly, the T4-dependent changes in F-actin content were not mediated by hormone-induced changes in transcription or translation, since neither cycloheximide nor actinomycin D affected this cell response. In addition, beta2-actin mRNA levels were unaffected by T4. These data are consistent with the proposed extranuclear site of action for thyroid hormone (12, 18).

Examination of the actin cytoskeleton in cultured astrocytes confirmed thyroid hormone's influence on the microfilament network. Astrocytes grown in the absence of thyroid hormone had a poorly developed actin cytoskeleton compared with cultures grown in medium supplemented with serum or with T4 alone. Iodothyronines induced a rapid reorganization of the poorly developed actin cytoskeleton in cells grown in serum-free medium, requiring as little as 20 min of exposure to thyroid hormone. Both T4 and the metabolically inactive metabolite, rT3, promoted repolymerization of the actin cytoskeleton, while an equimolar concentration of T3 was ineffective. These results are identical to earlier reports of the effects of these iodothyronines on type II 5'-deiodinase turnover in vivo (10, 11) and in the cultured astrocyte (18).

The specific events that mediate thyroid hormone's regulation of actin polymerization remain to be established. The finding that this hormone action is not transcriptionally mediated (18, 33) suggests that T4 may bind to and modulate the activity of one or more actin-binding proteins. Several actin-binding proteins capable of depolymerizing actin filaments or anchoring the actin filament to the membrane have been identified in the brain (34-36) and these may be targets for T4 binding and subsequent dissociation from the arrested actin fiber.

T4-dependent changes in actin polymerization may be particularly important during brain development. Both neurite outgrowth and dendritic spine formation are developmentally programmed events that are highly correlated with an increase in actin filaments (37-39). These processing events depend, in part, on specific interactions between the actin cytoskeleton, integrins in the neuronal cell membrane (40-43), and the neurite promoting domain of the laminin (44). Thus, the ability of thyroid hormone to modulate actin polymerization provides an attractive model by which this hormone could regulate neuronal process formation and ultimately cell-cell interactions.

The recent work of Faivre-Sarrailh and Rabie (33) is in agreement with this idea. They reported that the cerebellum of congenitally hypothyroid rats showed 60-70% decrements in F-actin content with no change in total actin pool and that T4 administration normalized the F-actin content of the cerebellum. These findings obtained in intact rats parallel our observations on the T4-dependent changes in actin polymerization in the cultured astrocyte and demonstrate that this culture model will be useful in unraveling the molecular events that mediate the T4-dependent regulation of actin polymerization in the central nervous system.

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