Characteristics and Metabolism of $\alpha_1$ Adrenergic Receptors in a Nonfusing Muscle Cell Line*

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The BC3H1 nonfusing muscle cell line possesses binding sites for [3H]prazosin. These binding sites are typically $\alpha_1$ adrenergic receptors as shown by their greater affinity (3700-fold) for prazosin than for yohimbine. Both kinetic and equilibrium analyses indicated that [3H]prazosin interacted with only one category of independent binding sites with the following characteristics. $K_d = 0.13 \pm 0.01$ nM, $B_{max} = 97 \pm 5$ fmol/mg of protein corresponding to 25,000 sites/cell ($n = 17$).

Biosynthesis of the $\alpha_1$ adrenergic receptor was investigated in cell culture (when the number of cells and their total protein content were constant). Phenoxybenzamine (10$^{-7}$ M) irreversibly blocked 50% of the $\alpha_1$ receptors in intact cells. More than 95% blockade of receptors was obtained with 10$^{-7}$ M phenoxybenzamine. After this blockade, new $\alpha_1$ adrenergic receptors reappeared in the cells with monoexponential kinetics. These new receptors corresponded to synthesized receptors since their appearance was blocked by cycloheximide (1 $\mu$g/ml). The cycloheximide action was reversible. If one makes the simple and probable hypothesis that the receptor production is constant and that degradation is a monoexponential process, the analysis of the kinetics of reappearance allows the determination of the rate constant for receptor degradation ($k = 0.63$ h$^{-1}$) and the rate of receptor production ($r = 3.2$ fmol/mg/h) corresponding to the synthesis of about 760 receptors/cell/h. The half-life of the receptor was 23 h.

Our knowledge of $\beta$ adrenergic receptor properties and their coupling characteristics with the adenylyl cyclase is mainly due to the use of two experimental tools, i.e., the obtention of highly labeled antagonists and agonists and the possibility of studying these systems in homogeneous cell populations such as avian erythrocytes (1-4) and cell lines without (5-7) or with different genetic deletions (8, 9). The investigation of $\alpha$ adrenergic receptors is more recent and, thus far, their characterization with labeled antagonists and agonists has only been done in whole tissues (10-14) and isolated platelets (15, 16). Therefore, we looked for a cell line having both $\alpha$ adrenergic receptors and susceptibility to genetic manipulations. BC3H1 cells were obtained from a tumor induced by nitrosoethylurea and were shown to possess many of the properties of muscle (17). In these cells, noradrenaline and acetylcholine are known to produce hyperpolarizing and depolarizing responses, respectively (17). We had previously shown that noradrenaline, through $\alpha_1$ adrenergic receptors, increases the permeability of the membrane of BC3H1 cells to $K^+$ ions (18). We show here that these cells contain highly specific $\alpha_1$ adrenergic receptors which can be labeled with [3H]-prazosin.

This cell line has also been shown to be a useful tool for studying the metabolism of acetylcholine receptors (19). It was, thus, of particular interest, in the same line, to investigate the metabolism of the $\alpha_1$ adrenergic receptor, another intrinsic membrane protein. This study was possible since we possess phenoxybenzamine (POB) which is an $\alpha$ adrenergic blocking agent with long lasting properties and has been extensively studied in biological and chemical experiments (20-24). Recently, it has been shown that POB irreversibly blocks the [3H]diiodoergocryptine binding to uterus membranes in vitro (23). The covalent interaction of POB with $\alpha_1$ adrenergic receptors is strongly suggested by the experiments of Guillaumet et al. (22) who showed that [3H]POB binding on hepatic $\alpha$ adrenergic receptors is maintained after its solubilization with Lubrol PX. We, therefore, used POB for blocking $\alpha_1$ adrenergic receptors in intact cells in order to study the kinetics of receptor repopulation in BC3H1 cells.

EXPERIMENTAL PROCEDURES

Tissue Culture—BC3H1 muscle cell line was a gift from Dr. J. Patrick, The Salk Institute, San Diego, CA. The cells were grown in Dulbecco-modified Eagle's medium containing 10% fetal calf serum, 50 IU/ml of penicillin G, and 50 $\mu$g/ml of streptomycin sulfate at 37°C in an atmosphere of 5% CO2 and 95% air. The line was maintained in the exponential growth phase by passage every 4 days. Cells used for experimentation were plated in 90-mm diameter plastic tissue culture dishes at 10$^5$ cells/cm$^2$. They were used between 14 and 21 days after seeding and the medium was changed twice a week. The cells reached confluence 6 days after plating, but differentiated properties were fully expressed only after 10 days (17, 19).

Preparation of Particulate Fractions—Cells were washed twice at room temperature with 0.15 M NaCl and scraped off with a rubber probe in 5 ml of 0.15 M NaCl/dish. They were centrifuged for 3 min at 400 x g and the pellet was homogenized at 0°C in 5 mm Tris-HCl, pH 7.6, and 1 mm MgCl2 (about 3-10$^5$ cells/ml) with a Teflon-glass homogenizer. The homogenate was diluted by one-half with the homogenization buffer and centrifuged for 15 min at 30,000 x g at 4°C. The pellet was resuspended in 50 mm Tris-HCl, pH 7.6, and 10 mm MgCl2 (about 1.5-10$^6$ cells/ml) filtered through a silk screen (150 $\mu$m pore diameter) to remove some DNA filaments which had been formed during homogenization. It was centrifuged for 15 min at 30,000 x g at 4°C. The final pellet was resuspended in 50 mm Tris-HCl, pH 7.6, and 10 mm MgCl2 at a protein concentration of about 1-2 mg/ml. The particulate fractions were kept at 0°C and used for experiments within 3 h.

Measurements of [3H]Prazosin Binding—The particulate fraction (100-200 $\mu$g) was incubated for 30 min at 20°C in a solution containing 50 mm Tris-HCl, pH 7.6, 10 mm MgCl2, and 2-10$^{-11}$ M to 2-10$^{-8}$ M

$^1$ The abbreviation used is: POB, phenoxybenzamine.
concentration of [3H]prazosin (1 ml total volume). Incubations were terminated by rapid filtration of the entire mixture through Whatman GF/B glass fiber filters and by washing three times with 5 ml of ice-cold incubation buffer. The filters were dried and counted in scintillation fluid. Figures show specific binding defined as the binding which is inhibited by 10⁻⁸ M phentolamine.

**Results**

**Binding Characteristics of [3H]Prazosin to BC₃H₃ Particulate Fractions—**[3H]prazosin, an α₁ adrenergic antagonist (25), interacted with a single category of binding site in BC₃H₃ particulate fractions (Fig. 1A). The nonspecific binding determined in the presence of phentolamine (10⁻⁸ M) was very low (9% and 50% of the total binding at 0.1 and 2 nM, respectively). The dissociation constant (K₀) was 0.12 nM (Fig. 1A) (mean value 0.13 ± 0.01 nM; n = 17). The total number of sites was 122 fmol/mg of protein (mean value 97 ± 5 fmol/mg of protein; n = 17). Cells contained 25,000 ± 3,000 α₁ adrenergic receptor sites. The time course of association was ligand concentration dependent and the binding was reversible (Fig. 1B). Both association and dissociation curves can be adequately described in terms of reversible binding of the ligand to a homogeneous population of independent binding sites since the logarithmic transformations of these curves led to a good linear relationship (Fig. 1, C and D). The kₘₐₓ (kₘₐₓ = k₊, L + k₋₁, L = [3H]prazosin concentration; k₊, L and k₋₁, L = association and dissociation rate constant, respectively) determined from the association binding curves obtained at three different ligand concentrations (Fig. 1D) allowed us to determine k₋₁, L = 0.046 min⁻¹ and k₊, L = 5.13 10⁻⁶ M⁻¹ min⁻¹. The ratio of k₊, L/k₋₁, L was thus equal to 0.09 nM, a value in good agreement with the K₀ determined at equilibrium (see above). The direct measurement of k₋₁, L gave a value of 0.338 min⁻¹ (Fig. 1C).

**Specificity of [3H]Prazosin Binding Sites—**The agonist specificity of [3H]prazosin binding sites was very typical of an α₁ adrenergic receptor. The order of specificity was (--)adrenaline > (--)noradrenaline > (--)phenylephrine > (+)adrenaline > dopamine > (+)isoproterenol (Fig. 2A). The binding was stereospecific since (--)adrenaline was 16 times more potent than (+)adrenaline. The slope of the Hill plot of the displacement curves was very close to one, indicating the absence of any heterogeneity of the sites for agonists.

**Fig. 1. Binding properties of [3H]prazosin binding to BC₃H₃ cell membranes.** BC₃H₃ cell membranes (116 μg of protein) were incubated with [3H]prazosin as indicated under "Methods." A, Scatchard analysis of specific binding of [3H]prazosin to BC₃H₃ cell membranes. [3H]Prazosin concentrations varied from 0.04 nM to 1.7 nM. B, kinetics of [3H]prazosin association with and dissociation from BC₃H₃ cell membranes. Associations were done at the three indicated concentrations of [3H]prazosin and reversibility of binding was measured after the addition of 10⁻⁵ M phentolamine 20 min after the incubation of the membranes with 0.16 nM [3H]prazosin. C, semilogarithmic plot of the dissociation process. The slope is equal to the dissociation rate constant k₋₁. [RH] = concentration of the hormone-receptor complex; [RH₀] = concentration of the hormone-receptor complex when phenolamine was added. D, semilogarithmic plots of the association processes. The slope is equal to the observed association rate constants (k₊, L). The values of k₊, L obtained at the three concentrations of [3H]prazosin permit the determination of the dissociation rate constant (k₋₁, L) and the dissociation rate constant (k₋₁). [RH] = concentration of hormone-receptor complex; [RH₀] = concentration of hormone-receptor complex at equilibrium.

The antagonist specificity clearly indicated the α₁ characteristics of the receptors labeled with [3H]prazosin. Prazosin, an α₁ specific antagonist, was 3700 times more potent than yohimbine, an α₂ specific antagonist (25) (Fig. 2B). Note that phenoxybenzamine did not give a displacement curve parallel to those of other antagonists. This is due to the nonreversible interaction of phenoxybenzamine with the binding sites (see below).

**Specific Irreversible Blockade of α₁ Adrenergic Receptors by Phenoxybenzamine on Intact Cells—**Cells were incubated for 10 min with increasing concentrations of POB (10⁻⁶ M, 10⁻⁵ M, 10⁻⁴ M, and 10⁻³ M). After extensive washing of the cells, the particulate fractions were prepared and [3H]prazosin binding measured. As seen in Fig. 3A, POB reduced the total number of sites without affecting the dissociation constant of the remaining sites. At 10⁻⁶ M concentration of POB, more than 95% of the binding sites were blocked and no Scatchard plot could be drawn. At 3 × 10⁻⁵ M concentration of POB, the irreversible blocking action of POB was very rapid since it was maximal after 5 min (Fig. 3B). Note that the blocking action of POB was not increased even when incubation lasted for 30 min and despite the fact that free POB was still present. This is probably due to a rapid inactivation of the reactive aziridinium ion of free POB (24). This rapid inactivation of POB has also been reported by Williams and Lefkowitz (23). The POB action was receptor specific since it could be suppressed by phentolamine (10⁻⁵ M) (Fig. 3B).
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Fig. 2. Comparison of relative potencies of various drugs competing with $[^3H]$prazosin binding sites. A, inhibition of $[^3H]$prazosin specific binding by adrenergic agonists. The membranes (144 pg of protein) were incubated in the presence of 0.13 nM $[^3H]$prazosin. The $K_D$ values (nanomolar) for the different agonists were: (-)-adrenaline, 328; clonidine, 420; (-)-noradrenaline, 640; (-)-phenylephrine, 2,600; (+)-adrenaline, 5,200; dopamine, 31,500; and (-)-isoproterenol, 78,000. B, inhibition of $[^3H]$prazosin specific binding by adrenergic antagonists. The membranes (152 pg of protein) were incubated in the presence of 0.15 nM $[^3H]$prazosin. The $K_D$ values (nanomolar) for the different antagonists were: prazosin, 0.08; phentolamine, 12; and yohimbine, 275.

as seen in Fig. 4A. $\alpha_1$ Adrenergic receptor sites reappeared after only 3 h, and this reappearance was time dependent. These newly detectable receptors had a $K_D$ similar to that of control cells which did not receive POB. When the receptor blockade was performed with a 10-fold higher concentration of POB (10$^{-7}$ M rather than 10$^{-8}$ M) and tested 24 h later, the receptor reappearance was similar (Fig. 4B).

Determination of Synthesis and Degradation Rates of $\alpha_1$ Adrenergic Receptors in BC3H-1 Cells—The entire time course of reappearance was studied as described in Fig. 4A. In each experiment, the cells reached confluence before the beginning of POB blockade, so that during the entire time course, the total number of cells and the total protein content per plate did not vary (Fig. 5A). Furthermore, the total number of $\alpha_1$ adrenergic receptors in control cells which did not receive POB was unchanged during the entire period of reappearance ($R_{ss}$, concentration of receptor at steady state). It was equal to 10$^7$ ± 5 fmol/mg at the beginning of the experiment and 10$^5$ ± 5 fmol/mg of protein 7 days later (Fig. 5B).

In order to demonstrate that the receptor reappearance after irreversible blockade with POB truly represents newly synthesized molecules, we studied the reappearance in the presence of cycloheximide. Cycloheximide (1 $\mu$g/ml) blocked 86% of the total protein synthesis (Table I). After 24-h treatment with cycloheximide (1 $\mu$g/ml), the cells were still alive, since a 3-h period after elimination of cycloheximide was sufficient to allow 88% recovery of protein synthesis (Table I).

When cells were treated with POB (10$^{-7}$ M) and incubated thereafter with cycloheximide (1 $\mu$g/ml), the receptor reappearance was decreased by more than 95% (Fig. 5B). In addition, after this 24-h blockade of receptor reappearance by cycloheximide, the diminution of this protein synthesis inhibitor allowed cells to synthesize new receptors with approximately the same rate as before protein synthesis inhibition (Fig. 5B). Phenoxybenzamine (10$^{-7}$ M and 10$^{-5}$ M) did not affect protein synthesis (Table I).

If one makes the following simple hypotheses, already verified for acetylcholine (26) and insulin receptors (27), that 1) the receptor production is constant during the entire period...
between the production and the degradation of new receptors:

\[ \frac{d[R_t]}{dt} = r - k[R_t] \]

giving

\[ [R_t] = \frac{r}{k} (1 - e^{-kt}) \]  

when \( t \to \infty \) \([R_t]\) approaches \( r/k \), which is, therefore, equal to \([Rss] \); \([Rss] = r/k \) (2). Therefore, Equation 1 can be written \([R_t] = [Rss] (1 - e^{-kt}) \) (3). The logarithmic transformation (Log \([Rss]/[Rss - R_t] = kt \) (4)) of the experimental repopulation curve confirms that the repopulation was a monoexponential process (Fig. 5C). The slope of the straight line obtained gave the value of the rate constant for degradation \( k = 0.03 \) h\(^{-1}\) (Fig. 5C). Since \([Rss] = 105 \) fmol/mg of protein (Fig. 5) and \([Rss] = r/k \) (2), we can calculate the receptor production rate \( r = 3.2 \) fmol/mg of protein/h. Since there were 25,000 receptor sites/cell, one can estimate that 760 receptors were synthesized/h/cell.

The half-life of the receptor calculated with Equation 4 was 23 h.

**Discussion**

**Metabolism of \(a_1\) Adrenergic Receptors**

BC\(_2\)H\(_1\) cells appear to be a useful model for studying \(a_1\) adrenergic receptors in an isolated cell line. The \(a_1\) adrenergic receptors can be easily labeled with \(^3\)H)prazosin. These receptors were typically of the \(a_1\) type, as shown by the fact that the affinity for prazosin was 3,700 times greater than the affinity for yohimbine (Fig. 2B) (25). Both kinetic and equilibrium analyses indicated that \(^3\)H)prazosin interacts with only one category of independent binding sites having a high affinity for \(^3\)H)prazosin (\(K_d = 0.13 \pm 0.01\) nM; \(n = 17\)). The total number of sites is 97 \pm 5 fmol/mg of protein corresponding to 25,000 \pm 3,000 sites/cell.

It has been shown that at confluency, BC\(_2\)H\(_1\) cells exhibit some properties of muscle such as adenylate kinase and creatine phosphokinase activities, electrical excitability, and synthesis of acetylcholine receptors (17, 19). The possibility of keeping BC\(_2\)H\(_1\) cells in a nondividing state at confluency for 7 days without variation in the number of receptors (see Fig. 5) makes this cell line particularly suitable for studying the \(a_1\) adrenergic receptor functions and biosynthesis.

POB was used for irreversibly blocking \(a_1\) adrenergic receptors in intact cells; this blockade was very rapid and concentration dependent (Fig. 3A). POB (10\(^{-7}\) M) blocked 50% of \(a_1\) adrenergic receptors with no significant change in the apparent affinity of the free binding sites for \(^3\)H)prazosin (Fig. 3A). POB (10\(^{-7}\) M) blocked at least 95% of the entire \(a_1\) adrenergic population. This blockade was receptor specific since it was suppressed when \(a_1\) adrenergic receptors were saturated with phentolamine (Fig. 3B).

The reappearance of \(a_1\) adrenergic receptors after blockade with POB (10\(^{-7}\) M) was time dependent (Fig. 4). The new receptors had the same affinity as those of control cells (Fig. 4). In previous *in vivo* studies on POB's action in rats, a debate arose about the possibility that the long lasting effects of POB might be partly due to its being trapped in cells or membranes (20, 21). The possibility that such a phenomenon might constitute a rate-limiting step for receptor reappearance was tested by blocking the \(a_1\) adrenergic receptors with 10\(^{-7}\) M and 10\(^{-6}\) M POB. Since after 24 h, the number of receptors which reappeared was the same for the two doses, we can exclude any interference of free POB with the time course of receptor reappearance (Fig. 4B).

The reappearance of \(a_1\) adrenergic receptors was due to the biosynthesis of new receptor molecules since it could be blocked by cycloheximide at a concentration which inhibited 86% of protein synthesis without killing the cells (Fig. 5). Indeed, synthesis of \(a_1\) adrenergic receptors was reinitiated after washing out of cycloheximide (Fig. 5).

**Table I**

Effects of cycloheximide and phenoxybenzamine on protein synthesis

| Pretreatment | Washing period (3 h) | Labeling period (3 h) | Radioactivity in protein |
|--------------|----------------------|-----------------------|-------------------------|
| POB          | Cycloheximide        |                        |                         |
| \( \mu M \)  | \( \mu g/ml \)       | \( \mu g/ml \)       | cpm/mg protein         |
| 1            | 1                    | 1                     | 2800                    |
| 2            | 2                    | 2                     | 327                     |
| 5            | 5                    | 5                     | 206                     |
| 0.1          | 1                    | +                     | 2450                    |
| 0.1          | 2                    | +                     | 1450                    |
|              | 5                    | +                     | 780                     |
| 10           | 1                    | +                     | 2860                    |
|              | 2                    | +                     | 2540                    |
ential (Fig. 5). This indicates that the following hypotheses are probably correct. 1) The production rate of α1 adrenergic receptors by cells is constant. 2) The degradation of receptors is proportional to the concentration of receptors in the cell. The semilogarithmic transformation of the time course of reappearance allowed the calculation of the rate constant for receptor degradation \( k = 0.03 \text{ h}^{-1} \). The half-life of the α1 adrenergic receptor was thus equal to 23 h. This value is identical with that described for acetylcholine receptors by Devreotes and Fambrough (26) in chick skeletal muscle in culture and is very similar to that generally observed for extrajunctional acetylcholine receptors (28). However, in BCaH1 cells, Patrick et al. (19) reported a higher degradation rate constant \( (0.08 \text{ h}^{-1}) \) for acetylcholine receptors. This difference in the degradation rate for α1 adrenergic receptors and acetylcholine receptors in the same cells is interesting.

In these same experiments, we were also able to estimate the production rate of α1 adrenergic receptors. This was equal to 3.2 fmol/mg of protein/h. In the study of acetylcholine receptors, several authors (19, 26) have shown that in addition to surface membrane receptors labeled with α-bungarotoxin, there are hidden receptors, some of which are precursor receptors. We did not find such a heterogeneous population of receptors in our study. However, in contrast to α-bungarotoxin, we are not sure that phenoxybenzamine is unable to enter the cell and block precursor receptors.

During the course of these experiments with \( 10^{-7} \text{ M} \) POB and even with \( 10^{-8} \text{ M} \) POB, we noted that there was a small percentage of receptors (always less than 5%) which remained in the particulate fraction of cells. It is possible that these receptors are indeed precursors, but this question remains to be investigated in more detail.

The possibility of a simple method for measuring both the rate of production and the rate of degradation of α1 adrenergic receptors should provide new insight into the problem of modulation of these receptors under different hormonal and pharmacological situations.

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