Sodium Transport by the Acetylcholine Receptor of Cultured Muscle Cells

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SUMMARY

Activation of the acetylcholine receptors of cultured muscle cells by carbamylcholine increases the rate of passive $^{22}$Na$^+$ uptake into the muscle cells up to 20-fold. The Na$^+$ transport activity of the receptor desensitizes during exposure to carbamylcholine. The rate and extent of desensitization is reduced by lowering the assay temperature from 36$^\circ$ to 2$^\circ$, allowing accurate measurements of initial rates of Na$^+$ transport by the receptor.

Activation of the receptor by carbamylcholine and acetylcholine is significantly cooperative (Hill coefficients of 1.4 to 2.0). Inhibition by d-tubocurarine is not cooperative. The carbamylcholine-induced Na$^+$ transport activity of the receptor is inhibited 50% by 4 $\mu$M d-tubocurarine, 100 $\mu$M atropine, or 1.6 nM diiodo-α-bungarotoxin but is not affected by tetrodotoxin.

The initial rate of Na$^+$ transport by the receptor is temperature-independent between 2$^\circ$ and 36$^\circ$. Receptor Na$^+$ transport is saturable by Na$^+$ at 2$^\circ$ with an apparent $K_s$ of 150 ± 20 mm. Saturation by Na$^+$ is not observed at 36$^\circ$ at the concentrations tested. Saturation by Na$^+$ is observed at 2$^\circ$ both under conditions of net Na$^+$ influx and under conditions of isotopic exchange at equilibrium. The receptor does not catalyze obligatory exchange diffusion at a detectable rate. Comparison of binding of $[^{125}I]$diiodo-α-bungarotoxin with rates of Na$^+$ transport indicates a turnover number of $2 \times 10^7$ ions per min per receptor. These results are discussed in terms of the mechanism of Na$^+$ transport by the receptor.

During neuromuscular transmission of impulses, acetylcholine released from the presynaptic nerve ending binds to acetylcholine receptors of nicotinic specificity located in the postsynaptic muscle membrane and causes an increase in the passive permeability of the postsynaptic muscle membrane to Na$^+$, K$^+$, and other small cations. Electrophysiological studies have described both the presynaptic and postsynaptic events by monitoring transmembrane potentials and currents with microelectrodes placed within the cells (for a review see Reference 1). The postsynaptic response to acetylcholine is inhibited specifically by the α toxins of elapid snakes (Bungarus, Naja naja) and these toxins have been used to demonstrate the presence in the postsynaptic membrane of a specific class of macromolecules responsible for the response to acetylcholine (2).

Myoblasts obtained from embryos of a number of species can be cultured in vitro. During growth in vitro the cells divide and fuse into multinucleated myotubes (3) which are cross-striated, contain hypolemmal nuclei, contract spontaneously, and are capable of synapse formation (4, 5). Myotubes formed in vitro have a high concentration of acetylcholine receptors measured either electrophysiologically (5-7) or by binding of snake α toxins (8, 9). Cultured muscle cells therefore provide an important model system for studies of receptor function and regulation. Although microelectrode techniques involving recording from single cultured cells have yielded much useful information, convenient ion transport methods for studying directly the permeability changes due to receptor activation would be useful. Kasai and Changeux (10) have reported extensive studies of $^{22}$Na$^+$ efflux catalyzed by acetylcholine receptors in membrane vesicles from eel electroplax and demonstrated good pharmacological correlation between isotope flux measurements on membrane vesicles and electrophysiological current measurements on intact electroplax.

In the present investigation, I have applied a modification of the ion transport approach of Kasai and Changeux to a study of the Na$^+$ permeability increase due to activation of the acetylcholine receptors of cultured muscle cells.

EXPERIMENTAL PROCEDURE

Materials—Chemicals were obtained from the following sources: acetylcholine chloride, carbamylcholine chloride, d-tubocurarine chloride, atropine sulfate, n-arabinofuranosyleytosine, and ouabain from Sigma; tetrodotoxin from Calbiochem; $^{22}$NaCl from Amersham-Searle or New England Nuclear; [H]leucine from New England Nuclear; the Dubecco-Vogt modification of Eagle’s minimal essential medium from Grand Island Biological Co.; fetal bovine serum from Colorado Serum Co.; horse serum from Microbiological Associates; and re-crystallized trypsin from Worthington. Chick embryo extract and rat tail collagen were prepared as described in Refs. 11 and 12, respectively. [H]$^3$I Diiodo-α-bungarotoxin, prepared as described by Vogel et al. (8), was kindly supplied by Dr. Zvi Vogel.

Muscle Culture—Suspensions of single muscle cells from thigh muscle of 11-day-old chick embryos were prepared essentially as described by Fischbach (4). Cells were seeded at a density of 40,000/cm$^2$ in collagen-coated dishes (Falcon) or multi-wells (Linbro Chemical Co.) and grown in a medium consisting of 91% Dulbecco-Vogt modification of Eagle’s minimal essential medium,
suspended in 0.4 N NaOH, the cultured muscle cells take up only small quantities of 
Na+ that can be measured by inhibition of the active extrusion of

Carbamylcholine—Measurements of the passive Na+ permeability of cultured muscle cells have been carried out essentially as described previously (13) by inhibiting the active extrusion of 
22Na+ in exchange with the small pool of nonradioactive Na+ within the cells (Fig. 1). In the presence of ouabain, much larger quantities of 
22Na+ are taken up (Fig. 1) as the Na+ concentration

within the cells increases to the medium concentration (152 mM). The half-time for passive equilibration is 20 to 30 min and the time course is approximately exponential. Comparison of the extent of 
22Na+ uptake in the presence and absence of ouabain suggests that the cells maintain an internal Na+ concentration 12-fold lower than the medium concentration or approximately 13 mM.

Carbamylcholine (1 mM) causes a substantial increase in the initial rate of 
22Na+ uptake (Fig. 1). This increase is completely inhibited by 100 μM p-tubocurarine or 6 nM α-bungarotoxin (see below) and therefore must reflect activation of the nicotinic acetylcholine receptors of the cultured muscle cells. The increase in initial rate at early time points (Fig. 1, inset) is 18-fold in this experiment.

Although the increase in 
22Na+ uptake is very large at early times, the initial rate of uptake is not maintained. This point is illustrated more clearly in Fig. 2, in which some of the data of Fig. 1 have been plotted on semilogarithmic coordinates. If the permeability of the cells to Na+ is constant throughout the equilibration, the time course should be logarithmic and a plot of ln ([Na+]o/Na+]i) versus time should give a straight line whose slope is ln2/τdes (13). This relation is fulfilled in the absence of carbamylcholine (Fig. 2). In the presence of carbamylcholine, however, the rate of uptake is initially rapid (dashed line, Fig. 2) but decays to the control rate within 2 min. Thus the Na+ transport activity of the acetylcholine receptors of cultured muscle cells desensitizes during continued exposure to carbamylcholine or acetylcholine as has been described for receptors at adult neuromuscular junction (15).

The rate of desensitization of the Na+ transport activity of the receptor can be measured by preincubation with carbamylcholine for different times in the absence of Na+, followed by determination of the carbamylcholine-induced increase in 
22Na+ uptake (Fig. 5). The rate of 
22Na+ uptake by the receptor declined to approximately 10% of the original value during a 6-min exposure to carbamylcholine in the absence of Na+, with 50% loss of activity in 30 s in this experiment. A significant correlation between the rate of desensitization and the morphological maturity of the muscle cells was noted. Cell cultures in which myotube formation was complete, but in which no further differentiation had occurred, had high levels of receptor transport activity but desensitized relatively slowly as in Figs. 1 to 3. Cell cultures in which many myotubes were cross-striated, had

\[ \text{Unpublished results.} \]
FIG. 3. Desensitization of receptor Na+ transport activity. Muscle cells were preincubated at 36° for the indicated times in assay medium containing 1 mM carbamylcholine and Tris+ rather than Na+. 22Na+ uptake was then measured for 30 s at 36° in normal medium containing 5 mM ouabain and 1 mM carbamylcholine.

FIG. 4. Effect of temperature on receptor Na+ transport activity. 22Na+ uptake was measured at 2°, 21°, and 36° as described under “Experimental Procedure” in the presence of 5 mM ouabain and 5 mM carbamylcholine. 22Na+ uptake in the absence of carbamylcholine at each temperature has been subtracted from the results.

hypolemmal nuclei, and contracted spontaneously also had high levels of activity but desensitized more rapidly. In some experiments, complete desensitization occurred in 10 s. Since such rapid desensitization precluded accurate measurement of initial rates, conditions were sought which would slow desensitization. Removing extracellular Ca+ and adding ethylene glycol bis(β-aminoethyl ether)-N,N′-tetraacetic acid was partially effective (16), however, lowering the assay temperature proved to be more successful.

The initial rate of 22Na+ uptake catalyzed by the receptor is essentially temperature-independent between 2° and 36° (Fig. 4). At 2°, however, the initial rate is maintained for a longer period of time. Rates of uptake remained constant for at least 20 s in all cultures tested at 2° (Fig. 4). Thus, although the initial rate of 22Na+ uptake is temperature-independent, the rate and extent of desensitization are temperature-dependent. Since the initial rate of 22Na+ uptake in mature cultures can be measured more accurately at 2° than at physiological temperature, most of the experiments described here have been performed at 2°.

Dependence of Na+ Transport Activity of Acetylcholine Receptor on [Na+]in and [Na+]ext—In vivo, the acetylcholine receptor catalyzes net transport of Na+ into the muscle cell. In order to compare the rate of net transport with the rate of Na+-Na+ exchange, the rate of 22Na+ uptake was measured under conditions of net Na+ influx as in Figs. 1 to 4 and under conditions of isotope exchange at equilibrium. The results presented in Fig. 5 show that the initial rate of 22Na+ uptake into muscle cells with low internal Na+ is 1.8-fold greater than the rate of uptake into cells that have been preincubated with ouabain for 3 hours to bring the internal Na+ concentration to 152 mM. In four such experiments, the ratio of net influx to exchange averaged 1.9 ± 0.2. Control experiments confirmed that neither the number of receptors per cell (measured by toxin binding) nor the resting Na+ permeability of the cells was affected by the 3-hour exposure to ouabain. Thus, the rate of net influx exceeds the rate of exchange, demonstrating that the receptor does not catalyze an obligatory exchange diffusion reaction at a detectable rate.

The increased rate of 22Na+ uptake under conditions of net influx cannot be ascribed to an increased driving force due to the transmembrane potential (17). Although the resting potential of -40 millivolts (18) is large enough to account for a ratio of net influx to exchange of 1.9 (17), the transmembrane potential in the presence of 5 mM carbamylcholine should be in the range of 0 to -10 millivolts corresponding to a ratio of less than 1.15. The difference in rate can more likely be ascribed to a competitive inhibition of 22Na+ influx by internal unlabeled Na+ (see “Discussion”).

The initial rate of carbamylcholine-dependent 22Na+ uptake at 2° varies with [Na+]in in a manner suggesting saturation of the ion transport mechanism by Na+ (Fig. 6A). Similar results are obtained under conditions of net Na+ influx (Fig. 6A). Double reciprocal plots (Fig. 6B) show that the dependence upon [Na+]in is approximately described by the Michaelis-Menten relation with an average $K_m$ of 150 ± 20 mM in four experiments. There is a
Activation under all three of these sets of conditions is significantly cooperative. In each case, the data, when plotted on the coordinates of Brown and Hill (19) (Fig. 7B), give a line with slope greater than 1 (carbamylcholine at 2°C, n_H = 1.4; carbamylcholine at 36°C, n_H = 1.9; acetylcholine at 2°C, n_H = 2.0). Thus, as has been shown for the electroplax receptor (10, 20), activation of the acetylcholine receptors of cultured muscle cells is a cooperative process.

Activation of receptor Na+ transport activity by 1 mM carbamylcholine was inhibited by n-tubocurarine and atropine with half-maximal inhibition at 4 and 100 μM, respectively, at 2°C (Fig. 8). Similar results were obtained in experiments at 36°C. Inhibition by n-tubocurarine exhibited little if any cooperativity (n_H = 0.9 to 1.2).

Tetrodotoxin, which inhibits Na+ transport by the action potential Na+ ionophore of cultured chick muscle with a K_{JT} = 11 nM (13), has no effect on acetylcholine receptor Na+ transport at 30 μM (Fig. 8), confirming the conclusion that the action potential Na+ ionophore does not contribute to the observed Na+ movements.

Na+ Transport Activity of Single Receptor—Snake venom α-toxins have been shown to bind specifically to the acetylcholine receptors of cultured muscle cells (8, 9) by inhibition studies using nicotinic cholinergic agonists. The experiment presented in Fig. 9 compares the binding of [125I]diodo-α-bungarotoxin to muscle cells with the inhibition of the receptor Na+ transport activity by the iodinated toxin. The two titration curves coincide with 50% inhibition and binding at 1.6 nM toxin confirming the specificity of toxin binding. Binding of α-bungarotoxin therefore provides a measure of the number of acetylcholine receptors per muscle cell and Na+ uptake provides a measure of the number of Na+ ions transported into a muscle cell per min. The quotient of these two measurements is the turnover number of a single receptor. Data from experiments in which initial rates of Na+ uptake at 2°C and extents of [125I]diodo-α-bungarotoxin binding at 36°C were measured are summarized in Table I. The
cr-bungarotoxin. Muscle cells were incubated for 60 min with the a-bungarotoxin and washing as described under "Experimental Procedure." Initial rates of carbamylcholine-dependent $^{22}$Na$^+$ uptake were determined from measurements at 5, 10, 15, and 20 s at 2°C in medium containing 5 mm carbamylcholine and 5 mm ouabain. Uptake in the absence of carbamylcholine and binding of [125]$\text{I}$-toxin to dishes with no cells have been subtracted.

**Table 1**

Comparison of $^{22}$Na$^+$ uptake and [125]$\text{I}$diiodo-a-bungarotoxin binding

| Initial rate of $^{22}$Na$^+$ uptake | Toxic binding | Specific transport activity |
|-------------------------------------|---------------|---------------------------|
| nmol/min/mg                         | /mol/mg       | $^{22}$Na$^+$ ions/min/toxin site |
| 661 (520-832)                       | 207 (140-204) | $3.2 \times 10^4 \pm 0.6 \times 10^4$ |

Comparison of measurements of $^{22}$Na$^+$ uptake and bungarotoxin binding have suggested a $V_{\text{max}}$ for Na$^+$ transport by the acetylcholine receptor Na$^+$ ionophore of $2 \times 10^3$ min$^{-1}$ at $2^\circ$C (Table 1). This value is much larger than the value of $3 \times 10^4$ min$^{-1}$ derived from studies of $^{22}$Na$^+$ efflux from electrophys membrane vesicles (10). A larger estimate of $3 \times 10^5$ min$^{-1}$ has been derived from a comparison of carbamylcholine-induced conductance and binding of $\alpha$-toxin in whole electrophys (25).

Also, analysis of electrical noise produced by acetylcholine at the neuromuscular junction (26, 27) and in cultured muscle (28) has led to estimates of 0.3 to 0.9 $\times 10^{-11}$ mho for the conductance during a single noise-producing event. Equating this with the conductance of an acetylcholine receptor Na$^+$ ionophore gives an estimate of 4 to $12 \times 10^8$ min$^{-1}$ at $-40$ millivolts. The difference between the estimates derived using comparisons of toxin binding and steady state flux measurements (2 to $3 \times 10^4$ min$^{-1}$) and those derived from noise measurements (4 to $12 \times 10^8$ min$^{-1}$) may reflect the fraction of time that a given ionophore is active in the presence of activator. Thus the noise measurements estimate the instantaneous activity of a single active ionophore. On the other hand, the steady state flux measurements estimate the steady state activity of an ensemble of receptors. If the activity of a single receptor ionophore is a series of repetitive square pulses (27), at any time, only a fraction of the receptor ensemble is active. Thus, the steady state measurement must underestimate the instantaneous activity of an active receptor by a fraction that is equal to the proportion of the time that an individual ionophore is active. If this interpretation is correct, the acetylcholine receptor Na$^+$ ionophore is active only 2 to 8% of the time, even in the presence of a saturating level of activator.

Mechanisms of ion transport can be broadly divided into two classes: those involving a mobile carrier and those involving specific channels or pores. A major feature distinguishing these two classes of mechanisms is the extent to which macromolecular membrane components must move during transport of each substrate molecule. Thus, a mobile carrier must traverse or rotate through the membrane with each substrate molecule transported, while a channel must move during "opening" or activation but not during transport of each substrate molecule. These considerations predict that the rate of transport by a carrier should be: (a) relatively slow since large macromolecular components must move during transport (less than $6 \times 10^5$ min$^{-1}$ (29)) and (b) highly temperature-dependent, especially at the phase transition of the membrane lipids, since the viscosity of the membrane in which the carrier must move is highly temperature-dependent. These limitations need not apply to a channel.

Experimental support for these distinctions between carrier and channel mechanisms has been provided by experiments on ion-conducting antibiotics. Transport of valinomycin and nonactin which behave as mobile carriers (Ref. 30 and references therein) is highly temperature-dependent, especially at the lipid phase transition (30), and relatively slow ($6 \times 10^5$ min$^{-1}$) (31) while transport by gramicidin, a channel-former (Ref. 30 and references therein), is temperature-independent (30) and relatively rapid ($6 \times 10^5$ min$^{-1}$) (32). Other channel-forming antibiotics have comparably high turnover numbers. Most of the data available concern transport in biological systems suggest the involvement of carriers (reviewed in Ref. 33).

Large temperature dependences (33), especially at lipid phase transitions (34-36), and relatively slow rates (less than $10^6$ min$^{-1}$) (29) have been reported for transport of a number of metabolites.
in bacteria and for sugars and ions in mammalian erythrocytes. In addition to the characteristics of carriers described above, biological carrier systems exhibit transacceleration (33) and exchange diffusion, i.e., transport in which the rate of tracer influx is increased by increasing the internal substrate concentration (33).

The results described here provide three pieces of evidence indicating that the transport of Na\(^+\) by the acetylcholine receptor Na\(^+\) ionophore is best described by a channel or pore mechanism: (a) the transport process is essentially temperature-independent between 2\(^\circ\) and 36\(^\circ\), (b) the rate is more rapid (2 \times 10\(^7\) min\(^{-1}\)) than the limiting transport rate of even low molecular weight ion carriers such as valinomycin, and (c) the rate of uptake is not increased by increasing the internal Na\(^+\) concentration indicating that exchange diffusion does not contribute significantly to the observed transport. The action potential Na\(^+\) ionophore also appears to transport Na\(^+\) via a channel mechanism since the rate of ion movement (10\(^7\) min\(^{-1}\)) is too large to be accommodated by a carrier mechanism (37) and ion transport is not highly temperature-dependent (17). These two transport systems appear to be unique among biological systems studied to date.

Although the acetylcholine receptor appears to transport Na\(^+\) via a channel mechanism, the transport process does show saturation at 2\(^\circ\). While saturability is more often considered a property of carrier systems, channel mechanisms that incorporate saturability have been formulated (38), and the rate of ion transport by some channel-forming antibiotics has a high concentration limit (32). The results of Fig. 6 suggest that a binding site for Na\(^+\) exists within the receptor Na\(^+\) channel which at 2\(^\circ\) interacts with Na\(^+\) during the rate-limiting step in transport in a manner described approximately by the Michaelis-Menten relation with a \(K_m\) of 150 mM. If a similar interaction occurs at physiological temperature, the \(K_m\) must be higher. The binding site within the channel suggested by these data might play a role in determining the selectivity of ion transport.

The presence of a Na\(^+\) binding site within the receptor Na\(^+\) channel leads to the possibility of competitive inhibition of Na\(^+\) transport by Na\(^+\) and other transported ions. In particular, the observed inhibition of \(^{22}\)Na\(^+\) influx by internal Na\(^+\) (Fig. 5) is expected. Internal and external Na\(^+\) ions would have equal probability of occupying a Na\(^+\) binding site symmetrically oriented within the receptor Na\(^+\) channel. Since occupancy by internal Na\(^+\) would not result in \(^{22}\)Na\(^+\) uptake, the rate of \(^{22}\)Na\(^+\) uptake would be reduced 2-fold under conditions of isotope exchange. These considerations suggest that a detailed analysis of transport rates in the presence of known internal and external ion concentrations might provide considerable insight into the interaction of ions with the acetylcholine receptor Na\(^+\) channel during transport.

Acknowledgments—I thank Dr. Marshall Nirenberg for support and helpful discussions through the course of the experiments and preparation of the manuscript, Dr. Zvi Vogel for generously providing the iodinated toxin, Dr. Fred Sachs for stimulating discussions, and Ms. Joan Albert for help in preparing the manuscript.

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J. Biol. Chem. 1975, 250:1776-1781.

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