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THE ELECTRICAL CONSTANTS OF THE SKELETAL MUSCLE FIBRES OF THE STICK INSECT, *CARAUSIUS MOROSUS*

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SUMMARY

The electrical constants of the ventral longitudinal muscle fibres of the stick insect *Carausius morosus* were determined by analysing the low-frequency cable properties of the fibre using short cable theory. At 20 °C in normal Ringer the membrane conductance (G_m) was 0·32 mmho.cm^2, the sarcoplasmic conductivity (G_i) was 6·69 mmho/cm and the membrane capacity (C_m) was 12·2 μF/cm^2. In this solution the length constant (λ) was 2·13 mm and averaged 130% of the fibre length. Hypertonic (× 2) Ringers had no significant effect on G_i but increased the membrane conductance by about 35%. The effect of temperature on the electrical constants was also investigated: the temperature coefficient (Q_{10}) of G_m was 1·44 and that of G_i was 1·41. The Q_{10} of the membrane capacity was 1·11.

INTRODUCTION

The main aim of the present study was to determine the electrical constants of the ventral longitudinal muscle fibres of the stick insect, *Carausius morosus*, and their variation with temperature and tonicity. The ventral longitudinal muscle provides a favourable preparation for a voltage clamp study of the membrane currents of insect muscle (Ashcroft, Standen & Stanfield, 1979) since the fibres have relatively few sarcolemmal invaginations (Ashcroft, 1979). However, the 3-electrode voltage clamp used in experiments (Ashcroft *et al.* 1979) requires a knowledge of the specific internal resistance (R_i) in order to calculate the membrane current density. Since hypertonic Ringer solutions were used to study electrical events in the absence of contraction it was also important to obtain a value for R_i in a hypertonic solution. The temperature coefficient (Q_{10}) of the electrical constants was also determined as membrane currents are often recorded at low temperatures where the kinetics are slower.

Analysis of the electrical constants of nerve and muscle fibres is usually based on linear cable theory which treats the fibre as a leaky cable of infinite length (Hodgkin & Rushton, 1946). This assumption is clearly untenable in the case of many insect muscles where the fibres may be only 1–2 mm long and are rarely greater than 20 mm in length. In such short fibres electrical reflections from the ends of the fibre can occur and modified equations for short cables have to be substituted (Weidmann, 1952;

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Hodgkin & Nakajima, 1972; Jack, Noble & Tsein, 1975). However, most analyses of the electrical constants of insect muscle have used the equations for an infinite cable (Henček *et al.* 1968; Malpus, 1968; Usherwood, 1969) and there are relatively few based on short cable theory (Washio, 1972; Deitmer, 1977; Patlak, 1976). A second objective therefore was to extend the present knowledge of the passive electrical properties of insect muscle fibres.

METHODS

(*a*) *Preparation*

The experiments were carried out on the isolated ventral longitudinal muscle from the third thoracic segment of the stick insect, *Carausius morosus*. This muscle consists of about 50 loosely attached fibres which insert directly into the anterior body wall and are connected to the posterior cuticle by a common apodeme. The muscle, attached to a small piece of the anterior cuticle, was isolated and mounted on a Sylgard support at rest length (Ashcroft, 1979). The fibres are approximately 1·6 mm in length and vary between 60–100 μm in diameter. Since the electrical constants of muscle fibres vary with diameter (Hodgkin & Nakajima, 1972; Henček & Zachar, 1965), the present experiments were confined to surface fibres of similar diameter. Fibre diameter was measured using an occular micrometer at 3 points and taken as the mean value. Optical measurements of fibre diameter from a whole muscle are justified in this case as the fibres are only loosely connected; values of fibre diameter obtained by single fibre dissection fell within the standard error of measurements from whole muscle. Single fibre dissection and histological examination revealed the fibres to be roughly circular in cross-section so no correction was made for fibre shape when calculating electrical constants.

The normal Ringer contained (mM/l): NaCl, 20; KCl, 20; $MgCl_2$, 50; $CaCl_2$, 20; HEPES 5 (pH 7·4); Sucrose, 100; and was continuously bubbled with 100% O_2. Hypertonic ($\times$ 2) Ringer was made by adding 400 mM sucrose to the normal Ringer. Some of the experiments were carried out at room temperature (18–23 °C); when necessary temperature was controlled with a Peltier system.

(*b*) *Electrophysiological techniques*

Standard electrophysiological recording techniques were used (Ashcroft, 1979). Three microelectrodes filled with 3 M-KCl were inserted into the fibre. One of these electrodes (resistance 10 MΩ) was placed in the middle of the fibre and was used to pass current. The other two electrodes (resistance 10–30 MΩ) were used to record the voltages V_0 and V_x at distances $d = 0$ and $d = x$ from the current electrode. The V_x electrode was always placed at the fibre end: V_0 was usually located 50 μm away from the current electrode to avoid the radial potential gradients which can arise around the current electrode tip (Eisenberg & Johnson, 1970). Membrane responses to small rectangular pulses of inward current, duration about 300 msecs, were recorded at these electrodes. Current was measured using a standard current-to-voltage transducer circuit. Hyperpolarising pulses producing potential changes of less than 10 mV were used as the input resistance is linear over this range of potential. Membrane constants were calculated independently for three separate voltage deflections and were averaged at the final stage of calculation.

Insertion of a second electrode adjacent to the current-passing electrode causes a drop in resting potential which varies between 2–10 mV depending on the proximity of the electrodes and the input resistance of the fibre. This can be attributed to a leakage conductance around the electrode. Although this leakage conductance produces a noticeable error in the measurement of the resting potential the error in the estimated input resistance is not large. The magnitude of the leakage conductance can be obtained from

$$G_L = G_m(1-(V'_m/V_m)),$$

where G_L = leakage conductance; G_m = measured input conductance; V'_m is the measured membrane potential and V_m the true membrane potential. Calculated leakage conductances ranged from 0–0·12 μmmho having a mean value of 0·03 μmho. With a mean input conductance of 1·04 μmho in normal Ringer (1·14 in hypertonic Ringer) the estimated error averaged 3% (2·5% in sucrose Ringer).

(*c*) *Symbols and definitions*

V_0 electrotonic potential produced by an injected current I_0 at $d = 0$,
V_x potential produced at a distance x from the current electrode,
x distance between the current electrode and V_x,
l $\frac{1}{2}$ length of the fibre,
a fibre diameter,
λ fibre length constant (cm). $\lambda = \sqrt{(r_m/r_i)}$, (1)
r_i internal resistance per unit length of fibre (Ω/cm),
r_m membrane resistance × unit length of fibre (Ω.cm),
R_{eff} input resistance of fibre (Ω): $R_{eff} = V_0/I_0$,
R_i specific internal resistance (Ω.cm): $R_i = r_i \frac{1}{4}\pi a^2$ (2)
R_m specific membrane resistance (Ω.cm^2): $R_m = r_m \pi a$ (3)
G_i specific sarcoplasmic conductivity (mho/cm): $G_i = R_i^{-1}$,
G_m apparent membrane conductance referred to the surface (mho.cm^2): $G_m = R_m^{-1}$,
C_m low-frequency membrane capacity referred to the surface (F.cm^{-2}): $C_m = \tau_m/R_m$, (4)
τ_m membrane time constant (s).

(*d*) *Determination of membrane constants*

Membrane constants were determined by an analysis of the low frequency cable properties of the fibre using the modifications for short cables described by Weidmann (1952) and Hodgkin & Nakajima (1972).

For a short cable the following relations hold:

$$V_x/V_0 = \frac{\cosh(l-x/\lambda)}{\cosh(l/\lambda)}, \quad (5)$$

which simplifies to equation (6) when $l = x$

$$\frac{V_x}{V_0} = \frac{1}{\cosh(l/\lambda)}. \quad (6)$$

Equation (6) was used to calculate λ, the fibre length constant. The short cable equation for determination of r_i is

$$\frac{V_0}{I_0} = \tfrac{1}{2} r_i \lambda \coth (l/\lambda). \tag{7}$$

The electrical constants r_m, R_i and R_m were then obtained from equations (1), (2) and (3) respectively.

The membrane time constant (τ_m) is obtained from the time course of the voltage change to an injected current pulse. The infinite cable model predicts that the potential at V_0 is an error function of time (t) and reaches 84% of its final steady level when $t = \tau_m$. In a spherical core conductor the equivalent value for $t = \tau_m$ is 63% of its final value (i.e. an exponential function); that for a short cable model lies somewhere between the two extremes. Stephani & Steinbach (1969) have estimated values for t in the short cable by graphically superimposing responses obtained in the infinite cable model with these estimated by assuming total electrical reflection from the ends of a short cable (their Fig. 3). The value of $t = \tau_m$ obtained from their graph for Carausius muscle fibres was 65% and was used to determine τ_m. (l/λ lay between 0·38 and 0·56 for Carausius fibres.) This low value indicates that the electrical behaviour of the muscle fibres resembles that of a sphere rather than of a cable.

In common with other studies in muscle fibres the results of cable analysis were expressed in terms of area of the surface membrane and the contribution of the transverse tubular system (T-system) was ignored.

RESULTS

(*a*) *Electrical constants in normal Ringer*

Table 1 shows the membrane constants of *Carausius* fibres determined in normal Ringer at room temperature. Fibre diameter was taken as 80 μm (measured value 81·1 ± 6·4 μm, $n = 8$). The theoretical response of a finite cable to a step change in potential was calculated using these values, and compared to the experimental results (Fig. 2). According to Hodgkin & Nakajima (1972) the potential change associated with the make of a constant current in a short cable is

$$V_m(X, T) = I_0 \frac{\sqrt{(r_m r_i)}}{4} \sum_{n=-\infty}^{\infty} \{F[\,|X + 2n(L_1 + L_2)|, T] + F[\,|X + 2L_1 + 2n(L_1 + L_2)|, T]\}, \tag{8}$$

in which

$$F(X, T) = e^{-X} \operatorname{erfc}\left(\frac{X}{2\sqrt{T}} - \sqrt{T}\right) - e^{X} \operatorname{erfc}\left(\frac{X}{2\sqrt{T}} + \sqrt{T}\right), \tag{9}$$

and where

$X = x/\lambda$ and $T = t/\tau_m$

$L_1 = l_1/\lambda$ and $L_2 = l_2/\lambda$

n = number of reflections from the ends of the fibre ($n = -\infty \ldots \infty$).

When the current electrode is inserted in the centre of the fibre $l_1 = l_2$ and therefore

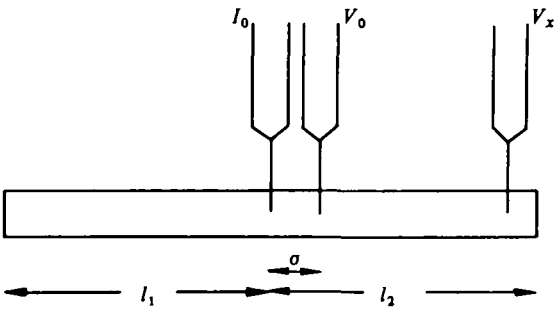

Fig. 1. Arrangement of microelectrodes for analysis of the electrical constants of a short cable. V_0 and V_x are the voltage recording electrodes, I_0 is for current injection, l_1 and l_2 are the distances from the current electrode to the end of the fibre. Since I_0 is inserted in the centre of the fibre $l_1 = l_2$. $\sigma = 50\ \mu$m.

Fig. 2. Electrotonic potential change produced at the V_0 and V_x electrodes in response to an injected current pulse of 9×10^{-9} A at I_0. $X = 0{\cdot}8$ mm. Filled circles represent the theoretical points calculated according to equation (8). Resting Potential -58 mV. Hypertonic Ringer 20 °C.

$2L_1 = 2L_2$ (Fig. 1). Equation (8) also describes the distribution of potential for the break of a constant current when the sign of X is reversed. A table of the function defined by equation (9), for various values of X and T is given by Hodgkin & Rushton (1946) and was used to evaluate the equation. In the present study the current was only considered to be reflected once from the ends of the fibre: as the fibres are so short (l/λ approximates 0·5) a better fit might have been obtained by including a larger number of reflections. The close agreement between the theoretical points derived from equation (8) and the experimental results suggests that the electrical behaviour of the muscle fibre approximates that of a short cable and provides a justification for this method of analysis.

Effect of hypertonic Ringer on electrical constants

Membrane constants determined for 17 fibres in hypertonic Ringer at room temperature are given in Table 2. These constants were calculated assuming a mean fibre diameter of 60 μm (measured values: $58{\cdot}3 \pm 2{\cdot}5\ \mu$m, $n = 11$). There is a significant drop in the length constant with increased tonicity (compare Tables 1 and 2) which is caused by a decrease in the specific membrane resistance (R_m) while the sarcoplasmic resistivity (R_i) remains constant. This constancy of R_i with hypertonic Ringer is somewhat surprising: hypertonic solutions caused a decrease in fibre diameter from

Table 1. *Electrical constants in normal ringer*

(× 10^6 means that the number must be multiplied by 10^6 to obtain the correct value. Room temperature. $n = 12$.)

	RP (mV)	$2l$ (mm)	λ (mm)	R_{eff} ($\Omega \times 10^6$)	R_i (Ω.cm)	R_m (Ω.cm^2)	r_i (Ω.cm^{-1} × 10^6)	r_m (Ω.cm × 10^4)	τ_m (msec)	C_m (μF.cm^{-2})	G_i (mmho.cm^{-1})	G_m (mmho.cm^2)
Mean	53·8	1·62	2·13	9·55	171	3668	3·81	14·6	30·8	12·2	6·69	0·32
±S.E.M. (n = 12)	±1·6	±0·04	±0·16	±0·88	±17	±460	±0·45	±1·8	±3·4	±0·58	±0·85	±0·03

Table 2. *Electrical constants in hypertonic ringer*

(× 10^6 means that the number must be multiplied by 10^6 to obtain the correct value. Room temperature. $n = 17$.)

	RP (mV)	$2l$ (mm)	λ (mm)	R_{eff} ($\Omega \times 10^6$)	R_i (Ω.cm)	R_m (Ω.cm^2)	r_i (Ω.cm^{-1} × 10^6)	r_m (Ω.cm × 10^4)	τ_m (msec)	C_m (μF.cm^{-2})	G_i (mmho.cm^{-1})	G_m (mmho.cm^2)
Mean	55·9	1·6	1·44	8·64	171	2365	6·05	12·55	34·3	14·3	6·23	0·44
±S.E.M. (n = 17)	±0·9	±0·02	±0·04	±0·54	±10·7	±120	±0·38	±0·63	±1·8	±1·0	±0·44	±0·02

Table 3. *Effect of temperature on the electrical constants*

(× 10^6 means that the number must be multiplied by 10^6 to obtain the correct value. Hypertonic Ringer. $n = 7$; Fibre length = 1·59 ± 0·04 mm; fibre diameter = 62·2 ± 1·4 μm)

	λ (mm)	R_{eff} ($\Omega \times 10^6$)	R_i (Ω.cm)	R_m (Ω.cm^2)	τ_m (msec)	C_m (μF.cm^{-2})	r_i (Ω.cm^{-1} × 10^6)	r_m (Ω.cm × 10^4)	G_i (mmho.cm^{-1})	G_m (mmho.cm^2)
20 °C	1·37 ±0·29	7·2 ±1·8	175 ±29	1994 ±529	34·3 ±4·4	16·7 ±2·1	5·55 ±0·4	10·2 ±1·2	6·08 ±0·6	0·54 ±0·05
3 °C	1·51 ±0·15	16·7 ±2·8	322 ±20	4930 ±928	54·2 ±10·7	13·4 ±1·0	10·33 ±0·5	25·2 ±5·0	3·18 ±0·2	0·25 ±0·04
Return 20 °C	1·54 ±0·12	8·9 ±0·8	172 ±13	2580 ±310	32·5 ±3·0	13·5 ±1·8	5·57 ±0·4	13·2 ±1·8	6·00 ±0·4	0·42 ±0·05

about 80 to 60 μm and internal ionic strength would be expected to increase with fibre shrinkage thus increasing G_i. The mean value of R_m decreased by 35% in hypertonic Ringer from 3668 to 2365 Ω cm^2. The hyperpolarization of the resting potential and the slight increase in the membrane capacity observed in sucrose Ringer were not statistically significant.

Effect of temperature on membrane constants

The electrical constants of a fibre were first determined at about 20 °C, then at 3 °C, and finally at room temperature again; each fibre thus served as its own control. Table 3 summarizes the results obtained for seven fibres of similar diameter. (Fibre diameter was measured individually in this case.) These results were tested for significance using the paired t test, a value for the electrical constants at 20 °C being obtained from the initial and final response of a fibre. The length constant was unchanged, or slightly increased, on cooling. Both the sarcoplasmic conductance (G_i) and the membrane conductance (G_m) were significantly decreased by low temperature ($p < 0{\cdot}01$) and the effect was reversible. The temperature coefficient (Q_{10}) of G_i was $1{\cdot}41 \pm 0{\cdot}04$ (S.E. of mean, $n = 7$) and that of G_m, $1{\cdot}44 \pm 0{\cdot}05$ ($n = 7$). Membrane capacity showed little variation with temperature, the Q_{10} being close to unity ($1{\cdot}11 \pm 0{\cdot}02$).

Membrane conductance did not return to its original level after cooling but was reduced by an average of 23 % (this difference is statistically significant, $P < 0{\cdot}05$). An artificially high initial conductance caused by a leakage conductance around the microelectrodes could cause a decrease in the apparent G_m if the electrodes were to seal in more effectively with time (cf. Methods). Fibre deterioration due to prolonged exposure to hypertonic Ringer could not have produced the observed reduction as the initial values of all fibres were low.

DISCUSSION

The length constant, λ, averaged 130% of the fibre length in normal Ringer and 90% of the fibre length in sucrose Ringer. This relatively large length constant facilitates the electrotonic spread of potential along the fibre and, in conjunction with the multi-terminal nature of the innervation, is responsible for the almost uniform membrane depolarization induced by nerve stimulation. Similar values for λ have been reported for other insect fibres (Deitmer, 1977; Washio, 1972; Henček *et al.* 1968).

The mean value of R_i, 171 Ω cm at 20 °C, lies between the values of 62 Ω cm (Deitmer, 1977) and 480 Ω cm (Patlak, 1976) previously obtained in insect muscles and is in close agreement with the sarcoplasmic resistivity of 170 Ω cm calculated by Hodgkin & Nakajima (1972) for isolated frog muscle fibres. Katz (1948) and Fatt (1964) who used measurements of transverse impedance, also give similar values for the sarcoplasmic resistivity of frog muscle. In crustacean muscle fibres, R_i appears somewhat lower, averaging around 100 Ω cm (Fatt & Katz, 1953; Dorai raj, 1964; Suarez-Kurtz & Sorenson, 1977). A variation of the internal resistance with the potassium concentration of the Ringer was reported by Schneider (1970) for the muscle fibres of *Rana pipiens*, a 5 mM increase in external potassium being associated with a 10% reduction in R_i. This may partially explain the wide range of values obtained for

R_i in insect muscles as the potassium concentration of the salines were very different. Patlak (1976) used a saline containing 4 mM-KCl, *Carausius* Ringer has a potassium concentration of 20 mM and the lowest value of R_i was obtained with a relatively high level of $[K]_0$ – 32 mM (Deitmer, 1977). The sarcoplasmic resistance of *Carausius* fibres was approximately twice that of the surrounding Ringer (60–90 Ω cm) and in accord with measurements on a variety of other tissues (red blood cells, squid axon, frog sartorius) which yield comparable ratios.

The membrane resistance of *Carausius* muscle referred to the fibre surface averaged 3668 Ω cm^2 in normal Ringer giving a membrane conductance of 0·32 mmho.cm^{-2}. Values of R_m determined by short cable analysis in other insect muscles range from 477 Ω cm^2 for the ventral longitudinal fibres of *Ephestia* larvae (Deitmer, 1977) to 4000 Ω cm^2 in *Saturniid* moth pupae (Yamaguchi, Lockshin & Woodwood, 1972). In general it appears that a high G_m is characteristic of larval skeletal muscles, such as the muscle fibres of dragonfly (Malpus, 1968) and *Ephestia* (Deitmer, 1977) larvae, whereas the later developmental stages have a lower conductance (adult *Sarcophoga* (Patlak, 1976) and *Locusta* (Washio, 1972) muscle). Conventionally membrane resistance is referred to a square centimetre of sarcolemma, the surface area of the fibre being calculated from measurements of fibre diameter. Because of the presence of caveolae, surface clefts, and an extensive network of associated tubules, the total area of membrane is therefore underestimated and the true resistance of the sarcolemma will be greater. It is therefore possible that the high G_m characteristic of larval insect muscle results from extensive sarcolemmal imaginations: significantly there is a good correlation between a low value for R_m and high membrane capacity.

The low-frequency membrane capacity of Carausius muscle fibres was 12·2 μF.cm^{-2}, and therefore contains a substantial contribution from the T-system (Peachey, 1968). Membrane capacity reported for other insect muscles ranges from 5·9 μF.cm^2 (Yamaguchi *et al.* 1972) to 47 μF.cm^{-2} (Malpus, 1968). As the correlation between the combined area of surface and tubular membranes, and the value of C_m is quite good for most muscle fibres (Selverston, 1967; Peachey, 1968) the range of capacity observed among insects of different species probably reflects variations in the extent of the TTS.

The most pronounced effect of hypertonic saline was a large increase in the specific membrane conductance. Freygang, Rapoport & Peachey (1967) interpreted a similar response of frog sartorius to sucrose hypertonic Ringer as an increase in the chloride conductance of the sarcolemma, since G_m was further increased by hypertonic NaCl. A reduction in R_m was also reported by Suarez-Kurtz & Sorenson (1977) for crustacean fibres. Fibre deterioration caused by prolonged exposure to increased tonicity, which might be reflected in an increased conductance, cannot account for the raised G_m as it would be associated with a decrease in the resting potential. The simplest explanation is that fibre shrinkage in hypertonic Ringer is associated with corrugation of the sarcolemma and that the consequent underestimation of membrane area yields a reduced value for the specific membrane resistance. This is supported by the observation that the constant r_m, whose determination is independent of fibre diameter, was not significantly reduced in sucrose Ringer. An alternative explanation for the decline in R_m is that it represents a genuine increase in the conductance of either, or both, the surface and tubular membranes.

Ringer solutions made hypertonic with an impermeant molecule such as sucrose cause dilation of the TTS elements of frog muscle fibres (Freygang *et al.* 1967; Huxley, Page & Wilkie, 1963) and there is a corresponding increase in tubular capacity (Freygang *et al.* 1967; Valdiosera, Clausen & Eisenberg 1974). Increased tonicity causes a comparable rise in the membrane capacity of crustacean fibres (Suarez-Kurtz & Sorenson, 1977) and the increased C_m of *Carausius* fibres may have a similar origin. It is also possible that the apparent rise in C_m results from an underestimation of the membrane surface area in hypertonic solution, as suggested for the specific membrane resistance.

Although the sarcoplasmic conductivity was expected to increase as a consequence of greater ionic strength, G_i was unaffected by tonicity. Similar anomalous behaviour has been reported for frog muscle (Hodgkin & Nakajima, 1972) and crab muscle (Suarez-Kurtz & Sorenson, 1977), where G_i is decreased by hypertonicity. Possible explanations include an increase in sarcoplasmic viscosity (Freygang *et al.* 1967), ion-binding and closure of an intracellular channel for current flow due to shrinkage. An increase in the viscosity of the frog fibre myoplasm is indicated by an increased resistance to stretch (Howarth, 1958) and depression of contractures due to injected calcium (April *et al.* 1968) or externally applied caffeine (Gordon & Godt, 1970). Dulhunty and Franzini-Armstrong (1975) have emphasized the importance of the SR geometry on intracellular current flow: distortions occurred by stretch, and therefore presumably shrinkage, caused an increase in R_i. Any rise in intracellular potassium concentration should also affect the resting potential and the lack of a significant hyperpolarization suggests that ion-binding or potassium efflux must occur. A combination of all these effects is probable.

Low temperature decreased both the total membrane conductance and the sarcoplasmic conductivity, the temperature coefficient for G_i being 1·41 and that of G_m, 1·44. For comparison the Q_{10} values obtained in frog muscle are G_i, 1·37; G_m, 1·49 (Hodgkin & Nakajima, 1972) and those in crustacean muscle are G_i, 1·2; G_m, 1·49 (Fatt & Katz, 1953).

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