Temperature Compensation in the Peripheral Nervous System: Antarctic vs Temperate Poikilothersms

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Summary. The effects of temperature on compound action potential velocities in peripheral nerves from Antarctic fishes and invertebrates were compared with those from temperate poikilothersms. Conduction velocity is a linear function of temperature, with sharp upper and lower limits corresponding to ‘heat-block’ and ‘cold-block’. Slope, x-intercept and cold- and heat-block temperatures were reduced in Antarctic poikilothersms. Cold-block could not be demonstrated, but heat-block occurred around 31 °C. Neuromuscular activation failed between 16 ° and 22 °C in Antarctic preparations. Fast fibres show steeper V/T slopes than slow fibres, but the two classes tend to converge on a common x-intercept; normalised velocities give nearly identical slopes, indicating that both large and small fibres are affected equally by temperature. Similarities in pattern between short- and long-term cold adaptation in both poikilothersms and endotherms suggest a common mechanism for membrane adaptations to low temperatures, and are consistent with the hypothesis of homeoviscous adaptation.

Introduction
Changes in temperature have profound effects on the function of the nervous system, which manifest themselves in a range of human experience, from paralysis of chilled extremities and cryogenic anaesthesia in minor surgery, to febrile hyperactivity. Synaptic transmission, membrane excitability, axoplasmic synthesis and transport, and conduction of the nervous impulse are all liable to the effects of temperature. Like other physiological systems, the nervous system is capable of adjusting itself for optimal function, either by the short-term process of acclimation, or long-term evolutionary changes. Local adaptations heterothermy may even occur in the nerves of the same animal (Chatfield et al. 1953; Miller 1970). Amongst endotherms, adaptive differences have also been reported for nerve function in arctic versus temperate mammals (Miller and Irving 1967), or in hibernating versus active rodents (Chatfield et al. 1948; Kehl and Morrison 1960).

One shortcoming of such work on endotherm nerves is the difficulty of accurately determining the thermal history of peripheral nerve. While deep-body nerves are maintained at a well-defined core temperature, the temperature of peripheral nerves is a compromise between the temperatures of the core and the environment. In contrast, the nerves of poikilothersms operate at very nearly the ambient temperature. Where that temperature is relatively constant, as under laboratory conditions or in offshore marine waters, variability in thermal history is greatly reduced, and the ‘normal’ operating temperature of peripheral nerves can be determined more accurately than in endotherms.

The Antarctic seas have been in equilibrium with ice for about 20 million years (Kennett 1977), and modern measurements in such high latitude deep basins as McMurdo Sound indicate an annual standard deviation of only 0.09 °C from the mean temperature of –1.87 °C (Littlepage 1965). Fishes and invertebrates from this environment are markedly stenothermal, with an upper lethal limit between 6 ° and 8 °C (Somero and DeVries 1967; Wells 1979). Attempts to acclimate Antarctic fishes to higher temperatures have been unsuccessful (DeVries, personal communication). Thus the nerves of Antarctic marine poikilothersms operate in an extremely limited and well defined low temperature domain, and may be expected to show appropriate evolutionary adaptations when compared with temperate or tropical poikilothersms. Over the past twenty years, several aspects of cold adaptation have been documented in Antarctic fishes: metabolic compensation (Wohlschlag 1964), altered
enzyme optima (Somero et al. 1968; Baldwin 1971), and biophysical cryoprotection (Raymond and DeVries 1977) have been described, but very little has been published about neurophysiological adaptations, other than a few preliminary reports (Macdonald and Ensor 1975; Macdonald and Wells 1978).

**Methods and Materials**

**Experimental Animals**

Antarctic fishes and invertebrates were captured during the summer (November through January) through the annual sea ice in McMurdo Sound. Benthic fishes (e.g. *Trematomus bernacchii* (Fam. Nototheniidae)) and arthropods (*Colossendeis robusta* (Pycnogonida; Fam. Colossendeidae), *Glyptonotus antarcticus* (O. Isopoda, Fam. Idoteidae)) were taken in baited traps. A common cryopelagic fish, *Trematomus borchgraeveni*, was taken on hand lines immediately beneath the ice, and the giant 'antarctic cod', *Dissostichus mawsoni*, was captured on set lines near the bottom in depths of about 500 m. Most animals were used within a few days of capture, but some were held for up to several weeks at −1.5 °C in aquaria continuously flushed with fresh sea water.

Temperate fishes (*Chrysophrys auratus* (Fam. Sparidae), *Nardogon scaber* (Fam. Monacanthidae)) were obtained from northern New Zealand waters at an ambient summer temperature of 16 °C, and used within two days of capture. Cane toads (*Bufo marinus*) were imported from Queensland, and introduced tree frogs (*Litoria aurea* (Fam. Hylidae)) were captured near Auckland. Both anurans were held for several winter months at 18 °C to 20 °C, with repeated force-feeding on beef liver. Earthworms (*Lumbricus terrestris*) were collected in wintertime in Auckland, and kept for 2–3 weeks at 12 °C in moist leaf mould.

**Nerve Conduction Studies**

Peripheral nerves were excised from fishes and invertebrates and transferred into appropriate physiological saline (Table 1) in a double-walled copper chamber. Temperature was controlled by adjusting the flow rate of cold (or warm) isopropanol through the outer jacket of the nerve chamber. Each measurement of temperature was made by balancing an amplified bridge circuit connected to a calibrated thermistor in the bath immediately adjacent to the nerve. Stimulation and recording were accomplished with silver wire electrodes, lifting the ends of the undivided nerve just clear of the surface film of the saline solution; between measurements the electrodes were lowered beneath the surface. A 0.1 ms square pulse was used to produce a symmetrical biphasic stimulating pulse, repeated at a frequency of 0.5 Hz. Stimulus amplitude was normally limited to just above threshold for each fibre group being tested; in most preparations only the fastest group was stimulated. Under these conditions preparations commonly lasted for as long as eight hours with minimal deterioration.

**Conduction velocity** was calculated by dividing the distance between stimulating and recording electrodes by the latency of the peak of the compound action potential. On longer nerves, velocities were calculated from conduction times between two recording electrodes, but this technique was unsuitable for very short lengths from smaller species. There was very little difference between velocities determined either way, nor was there any noticeable difference in the effect of temperature on velocity. Failure to record a compound action potential with a stimulus voltage five times threshold was used as a criterion for conduction block.

**Neuromuscular Activation**

Temperature effects on neuromuscular activation were assessed on inferior oblique extraocular muscles of *Trematomus borghreveni* and *T. bernacchii*. The muscle was exposed ventrally, but remained attached to the orbit and eyeball; a thermistor inserted between the muscle and eyeball was used to monitor temperature. The oculomotor nerve (cranial III) was stimulated supramaximally with a single biphasic pulse, and the motor response monitored (1) by visual observation of degree of contraction (recorded as strong, slight or none), and (2) by recording an electromyogram with a stainless steel needle, insulated except for the tip, inserted into the body of the muscle. Complete failure of contraction was double-checked by stimulating with a brief pulse train.

In the isopod *Glyptonotus antarcticus*, the proximal portion of the nerve was dissected free from an isolated walking leg and stimulated electrically with a brief pulse train. The leg was immobilised except for the dactylopodite, and responses were monitored visually, and by a strain gauge attached to the dactylopodite.

Except for the stimulated nerve end, all muscle preparations were completely immersed in bathing solution.

**Results**

The general pattern of changes in conduction velocity with acute changes in temperature is shown for the cane toad, *Bufo marinus*, in Fig. I. Four features should be noted for comparison between warm- and cold-adapted nerves: (A) The plot of conduction velocity versus temperature is linear, thus the effect of temperature is best expressed as the intercept and slope of this line, rather than as a Q10 value based on an exponential model. (B) The plot may be extrapolated to an x-intercept (zero velocity) at a characteristic temperature (*T0*) outside the normal physiological range of the nerve. (C) At low temperatures conduction fails ('cold block') above *T0*. (D) At high temperatures conduction velocity falls off slowly at first, then rapidly fails ('heat block'). The decrement in velocity prior to heat block may be partly due

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**Table 1. Composition of physiological solutions; original recipes or blood ion concentrations are given in cited references**

<table>
<thead>
<tr>
<th>Animal</th>
<th>NaCl</th>
<th>KCl</th>
<th>CaCl₂</th>
<th>MgCl₂</th>
<th>Tris-HCl</th>
<th>pH</th>
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<td>4.92 mmol</td>
<td>3.66 mmol</td>
<td>2.72 mmol</td>
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<td>–</td>
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<td>–</td>
<td>10</td>
<td>7.2</td>
<td>Hagiwara and Nakajima 1966</td>
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<tr>
<td>Earthworm</td>
<td>75.6</td>
<td>4.0</td>
<td>2.95</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Kamemoto et al. 1962</td>
</tr>
<tr>
<td>Marine invertebrates</td>
<td>sea water</td>
<td>–</td>
<td>–</td>
<td>–</td>
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</table>
Fig. 1. *Bufo marinus* sciatic nerve: conduction velocity versus temperature in a temperate poikilotherm. Features commonly affected by temperature adaptation include: A) Slope of the linear portion of the response. In *Bufo* the response is linear between $-1.2^\circ$ and $38^\circ$ C. B) x-intercept ($T_0$). C) Cold-block. D) Heat-block.

Fig. 2. Antarctic fish nerves: conduction velocity as a function of temperature. Heavy lines were derived from linear regression of data from a single specimen of *Dissostichus mawsoni*: Fourth spinal nerve (circles), $V=15.94+0.93 T$; lateral line nerve (triangles), $V=12.99+0.70 T$. For comparison, three additional regression lines are shown, recalculated from data in the literature: Catfish spinal cord (---), $V=1.11+1.43 T$ (Bass 1971). Beaver caudal nerve (-----), $V=4.07+1.09 T$ (Miller 1970). Cat saphenous nerve (---), $V=2.53 T-12.9$ (Franz and Iggo 1968). Note clockwise rotation of plots for cold-adapted animals: *D.mawsoni* vs catfish; beaver vs cat. ($V=$ velocity, m s$^{-1}$; $T=$ temperature, $^\circ$C).

Fig. 3. *Trematomus bernacchii*: convergence of temperature responses of fast and slow fibres in a single spinal nerve. Different symbols are used to designate velocities of consistently identifiable peaks in the compound action potential: solid lines represent estimates of the temperature response of each fibre group as determined by least square regression. Regression lines tend to converge on a point near the x-intercept ($T_0$), implying that all fibres in a given nerve are affected equally by temperature.

Examples of temperature responses of nerves from the giant antarctic cod, *Dissostichus mawsoni*, are shown in Fig. 2. Fast fibre populations conducted at velocities in excess of 10 m s$^{-1}$ at the ambient temperature of $-1.9^\circ$C. No sign of cold block was detected down to nearly $-5^\circ$C, at which temperature the supercooled saline solution suddenly froze. There was no recovery after freezing. In all antarctic preparations, both vertebrate and invertebrate, freezing occurred before any other sign of physiological failure, so that it was necessary to approach the phenomenon of antarctic cold adaptation paradoxically, by studying responses to high temperatures. In *D.mawsoni*, heat block occurred near $31^\circ$C, with no recovery.

Nerves from smaller species of nototheniid fishes (*Trematomus bernacchii*, *T.borchgrevinki*) often showed heterogeneous populations of nerve fibres with wide ranges of conduction velocities converging at zero velocity on a common $T_0$ (about $-17^\circ$C) (Fig. 3). Similarly, fast and slow fibre responses converged on $T_0$ for *D.mawsoni* $(-18^\circ$C) (Fig. 2) and for temperate poikilotherms (*Bufo marinus*, *Navodon scaber*, *Lumbricus terrestris*). Convergence of the temperature responses indicates that the influence of temperature is proportional to the normal velocity of the nerve fibres, and suggests that there are no qualitative differences in the responses of fibres of different sizes. When velocities for fast and slow fibre populations are normalised ($V_{norm}=V_f/V_o$, where $V_f=$ velocity at temperature $T$, and $V_o=$ velocity at acclimation temperature) the $V/T$ slopes become nearly identical.
Table 2. Temperature coefficients of neural conduction in antarctic and temperate poikilotherms. Regression equation: \( V = a + bT \); \( V \) = velocity in \( \text{m s}^{-1} \); \( T \) = temperature in \( ^\circ\text{C} \). \( \hat{a} \) and \( \hat{b} \) weighted mean regression coefficients; \( T_a \) ambient temperature or acclimation temperature; \( T_0 \) extrapolated temperature of zero velocity (x-intercept); \( b_{\text{norm}} \) normalized slope; \( T_{ch} \) temperature of cold-block in \( ^\circ\text{C} \); \( T_{hh} \) temperature of heat-block in \( ^\circ\text{C} \); \( s_\xi \) standard error of the mean; \( V_{\text{NC}} \) ventral nerve cord. Combined values of \( \hat{a} \) and \( T_0 \) were calculated as means of subgroups weighted by the number of samples in each subgroup. Values for \( \hat{b} \) and \( b_{\text{norm}} \) were calculated from \( \hat{a} \), \( T_0 \), and \( T_0 \) in each row.

<table>
<thead>
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<th>Organism</th>
<th>( T_a ) (°C)</th>
<th>( \hat{a} )</th>
<th>( \hat{b} )</th>
<th>( b_{\text{norm}} )</th>
<th>( T_0 ) (°C)</th>
<th>( n )</th>
<th>( T_{ch} )</th>
<th>( s_\xi )</th>
<th>( n )</th>
<th>( T_{hh} )</th>
<th>( s_\xi )</th>
<th>( n )</th>
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<td>-</td>
<td>-</td>
<td>-</td>
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<td>3</td>
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<td>0.046</td>
<td>-23.1</td>
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<td>-2.0</td>
<td></td>
<td>8</td>
<td>40.4</td>
<td>-</td>
<td>15</td>
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</table>

Table 3. Critical temperatures for neuromuscular function in antarctic poikilotherms. EMG locally recorded electromyogram; Contraction deficit amplitude of contraction decreases noticeably; Contraction failure no movement at all.

<table>
<thead>
<tr>
<th>Mean temperature (°C)</th>
<th>( s_\xi )</th>
<th>( n )</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Trematomus borchgrevinki and T. bernacchii</strong></td>
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<tr>
<td>EMG latency increases</td>
<td>15.68</td>
<td>0.84</td>
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<tr>
<td>Contraction deficit</td>
<td>15.98</td>
<td>0.67</td>
</tr>
<tr>
<td>Contraction fails</td>
<td>17.86</td>
<td>0.79</td>
</tr>
<tr>
<td>EMG, inflection of amplitude curve</td>
<td>18.06</td>
<td>1.85</td>
</tr>
<tr>
<td>EMG, amplitude decreases</td>
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<td>0.52</td>
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<tr>
<td>EMG fails</td>
<td>22.2</td>
<td>0.85</td>
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<tr>
<td>Overall mean temperature of neuromuscular failure</td>
<td>17.69</td>
<td>0.49</td>
</tr>
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</table>

**Glyptonotus antarcticus**

| Contraction deficit | 15.5 °C | - | 2 |
| Contraction fails   | 16.85   | 0.48 | 4 |

\(^a\) Visual observation through microscope
\(^b\) Strain gauge recordings

In all three antarctic fish species investigated, impulse conduction failed at an upper temperature close to 31 °C (Table 2). Neuromuscular responses proved even more sensitive to temperature. In *T. borchgrevinki*, neuromuscular activation failed consistently between 16 °C and 22 °C (Table 3).

In comparison with fish nerves, those from antarctic marine arthropods show extremely low conduction velocities (Fig. 4). Cold block could not be demonstrated in any of the antarctic invertebrate nerves. The upper temperature limits for leg nerves from a large myconid, *Colossendeis robusta* (28 °C), and from the giant isopod *Glyptonotus antarcticus* (31 °C) (Table 2), were in the same range as for fish nerves, but the ventral nerve cord of *G. antarcticus* proved to be more resistant, and continued to function up to 37 °C, with some recovery on cooling. As seen by transmission electron microscopy, axons in the leg nerves of both arthropods were very small, unmyelinated, with diameters on the order of 100 nm. In *G. antarcticus*, neuromuscular excitation failed at an upper temperature of about 16 °C (Table 3). As in
antarctic fishes, the temperature responses of isopod and pycnogonid nerves converged on a $T_0$ in the vicinity of $-20\, ^\circ C$. Despite the difference in absolute conduction velocities, normalised slopes from the arthropods are very similar to normalised slopes from the antarctic fishes (Table 2). It is interesting to note that the lowest recorded velocity in the pycnogonid nerve is about 0.03 m s$^{-1}$, 4000 times slower than that of fast mammalian motor neurons (120 m s$^{-1}$).

**Discussion**

In contrast to many temperate poikilotherms, the antarctic poikilotherms under discussion live at a remarkably stable temperature. The tremendous masses of ice and water at the southern end of the Ross Sea keep the seawater temperature constant within a few tenths of a degree Celsius throughout the year (Littlepage 1965), which is better regulation than in most so-called homeotherms. Furthermore, environmental temperature is not only constant, but homogeneous, as seawater lacks a thermal density inversion, with its resultant sharp thermoline. As in other poikilotherms, the level of thermal energy in antarctic fishes and invertebrates is under environmental, rather than metabolic, control. Very little difference has been found between environmental and deep muscle temperatures in the most massive antarctic fish, *Dissostichus mawsoni* (J.A. Raymond, personal communication). Thus, like the surrounding sea, antarctic poikilotherms are physically buffered at the seawater/ice equilibrium temperature.

As shown in Fig. 1, the effect of temperature on conduction velocity can be expressed as a straight line characterised by four parameters: slope, intercept, and upper and lower limits. It is traditional to express linear functions in the form $V = a + bT$, where $a$ is the y-intercept. In this case $a$ would also be the conduction velocity at 0 °C, which varies between fast and slow fibres in the same nerve (Fig. 3), and may even be outside the physiological range of some warm-adapted nerves. To facilitate comparison between nerves of different sizes and/or adaptation temperatures, the basic equation may be rewritten as $V = b(T - T_0)$, where $T_0$ is the x-intercept, or the temperature at which velocity extrapolates to zero. $T_0$ is always outside the nerve’s physiological range, but does not change between fast and slow fibre groups. Furthermore, it seems to remain approximately the same for nerves of different species adapted to the same temperature.

When temperature responses of nerves from antarctic species are compared with those from temperate animals, both vertebrates and invertebrates show similar patterns of adaptation. All of the features observed in Fig. 1 are affected: (A) and (B), the slope and x-intercept ($T_0$) of the $V/T$ plot, are reduced in the antarctic species; compare *D. mawsoni* with catfish in Fig. 2, and *Glyphotonus* and *Colossendeis* with *Lumbricus* in Fig. 4. As the slope of the temperature response appears to be a function of normal conduction velocity (fibre size), only the fastest populations in each animal are directly comparable. Figure 5 summarises the differences between antarctic and temperate poikilotherms. Combined temperature responses for each group (Table 2; Fig. 5) were derived from mean x- and y-intercepts of individual linear regressions ($V$ vs. $T$), weighted with the coefficient of determination ($r^2$). Similar clockwise rotations of the velocity/temperature function have been reported for cold-acclimated frogs (McDonald et al. 1963) and earthworms (Lagerspetz and Talo 1967). Temperatures of ‘cold block’ (C) and ‘heat block’ (D) are also reduced in the antarctic species (Fig. 5; Table 2).

When changes in regression coefficients and blocking temperatures of poikilotherms are compared with those reported for cold- versus warm-adapted endotherm nerves (Chatfield et al. 1948, 1953; Kehl and Morrison 1960; Miller and Irving 1967; Miller 1970), a clear pattern of neural cold adaptation emerges, its main feature being a shift of all aspects of the temperature response toward lower temperatures. When the responses are normalised (Table 2),
the apparent clockwise rotation of the \( V/T \) relationship in cold-adapted animals is reduced, so that the adaptive response appears to be primarily a ‘Precht Type 3’ partial compensation (Hazel and Prosser 1974).

The similarity in neural patterns of cold adaptation between diverse animal groups, and between short- and long-term adaptation, probably reflects an underlying similarity in mechanisms. One likely mechanism is regulation of lipid fluidity, which seems to be a universal feature of temperature adaptation. Membrane lipids from cold-adapted organisms have higher proportions of unsaturated fatty acids than those from warm-adapted organisms and are less viscous at low temperatures. Correlations between temperature and lipid fluidity have been reported for a wide spectrum of organisms, including bacteria (Sinsenky 1974), protozoa (Nozawa et al. 1974), fishes (Cossins and Prosser 1978; Cossins et al. 1980) and mammals (Meng et al. 1969). Increased unsaturation of neurally derived lipids has been reported for antarctic fishes and a crustacean (Morris and Schneider 1969; Meyer-Rochow and Pyle 1980), but no measurements of membrane fluidity have yet been made. The observed decreases in \( T_0 \) and temperatures of conduction block in antarctic poikilotherms would be consistent with a postulated increase in low temperature membrane fluidity.

Linearity of the temperature response has been reported by other investigators for both endotherms (Chatfield et al. 1948; Kehl and Morrison 1960; Paital 1965; Miller 1970; Miller and Irving 1967; Franz and Iggo 1968; de Jesus et al. 1973) and poikilotherms (Lagerspetz and Talo 1967; Bass 1971; Walker 1975), but its basis is still unexplained. A fundamental question is whether the overall linearity is a consequence of the interaction of many non-linear functions, or whether it can be ascribed to the predominance of a single limiting linear reaction?

There are several aspects of axonal propagation which respond in a quasi-linear fashion to changes in temperature, and might be candidates for a limiting reaction. Despite the complex predictions of the Debye, Hückel and Onsager equation (Hamre and DeWane 1970), the conductivities of dilute KCl and anuran saline solutions are approximately linear over the temperature range of 0° to 40°C (Macdonald, unpublished) which suggests that axoplasmic conductivity might be approximated by a linear function. Dierolf and Brink (1973) reported a significant reduction of low-temperature axoplasmic resistance in cold-acclimated earthworms. Although their measurements were made at only two temperatures, the relationship between conductivity and temperature appears to be rotated clockwise in cold-acclimated worms. Thus axoplasmic conductivity seems to show the same adaptive pattern as described for axonal conduction. Cold-adapted marine fishes show increased extracellular ion concentrations (Hazel and Prosser 1974), and antarctic fishes have both high extracellular (Dobbs and DeVries 1975) and intracellular ion concentrations (O’Grady 1980), which would increase conductivity in the cold-adapted forms.

Another parameter likely to limit the velocity of spike propagation is maximum membrane conductivity (\( g_m \)) during sodium activation. Hodgkin and Katz (1949) measured rates of rise (\( S_{sp} \)) and driving voltages (\( E_{sp} \)) of action potentials at temperatures from 5° to 30°C in squid axon. The quotient \( S_{sp}/E_{sp} \) is proportional to \( g_m \), and when calculated from their data, approximates a linear function of temperature. If electrically sensitive ion channels are influenced by membrane fluidity, as has been reported for chemically activated channels in muscle end plates (Gage 1976), one would predict that changes in membrane lipid saturation associated with cold adaptation would be reflected in channel kinetics, with a resulting shift in the temperature dependence of propagation.

Either of the above could account for the observed effects of temperature on axonal conduction. Both
seem to be linear, and both should be independent of fibre size. Acclimatory changes have been demonstrated for one (axoplasmic conductivity), but no measurements have been made of adaptive changes in the second (active membrane conductivity).

As shown in Table 3, neuromuscular responses in antarctic poikilotherms are very easily blocked by elevated temperatures, although the site of the block (pre or post-synaptic, myofibrillar) cannot be identified from the data presented. What is significant is that the train of events involved in neural activation of muscular contraction is more sensitive to temperature than is axonal propagation. This point has been made by other authors for temperate poikilotherms (Jensen 1972; Grainger 1973; White 1981). A more detailed study is currently underway to better localize the site of neuromuscular block in antarctic fish muscle (Macdonald and Montgomery, in preparation).

The relationships between normal temperature range, upper lethal limits, and the upper limits for neuromuscular function and axonal conduction are summarised in Fig. 6. It is clear that neither acute conduction block (ca. 31 °C) nor neuromuscular failure (16 ° to 22 °C) can be directly responsible for heat death in the intact animal, which occurs at far lower temperatures (6 ° to 8 °C: Somero and DeVries 1967; Wells 1979). Prosser and Nelson (1980) observed that complex integrative functions are more thermolabile than those of peripheral systems; this may be due largely to the multiplicity of central processes. Whereas axonal propagation depends basically on preserving electrical excitability, maintaining an electrochemical gradient, and ensuring sufficient longitudinal current to regenerate sodium conductivity, neuromuscular transmission and contraction add a number of steps to the basic problems of excitability. Enzymatic synthesis and degradation of transmitter, release of transmitter, receptor binding kinetics, and metabolic production of ATP are only a few of the obvious factors which may be altered by temperature. Central integrative processes will depend on these and still more factors, such as fine tuning of time and space constants, differential effects of antagonistic transmitters, and cumulative effects on networks of neuron populations, through cascading synapses. Total derangement of such a system could be precipitated by minor decrements in its component parts.

In temperate poikilotherms, temperature compensation may be induced by acclimatization or acclimation; the need for compensation, and its mechanisms, are thus a direct consequence of the animal’s poikilothermy. Antarctic poikilotherms have little or no capacity for such short-term adjustment; as shown in Fig. 6, they live within a narrow ambient temperature range (−1.9 ° to 0.5 °C over their entire circum-continental range), and rapidly succumb to temperatures only 6 ° to 8 °C above this range. In this respect they resemble homeotherms, as a similar increase in human body temperature can have disastrous consequences. The temperature compensation reported for antarctic animals is presumably of evolutionary origin. What are especially interesting are the parallels seen between short-term compensation in endotherms and temperate poikilotherms, and long-term genetic compensation in the antarctic poikilotherms.

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