based on an interaction of cholinergic and dopaminergic systems, particularly in the striatum[2]. Therefore, there is a need to see if neuroleptics enhance RIC and if atropine or mecamylamine diminish that effect while physostigmine enhances it.

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0014-4754/83/020228-03$1.50+0.20/0

Fast decay of fish synaptic currents[1]

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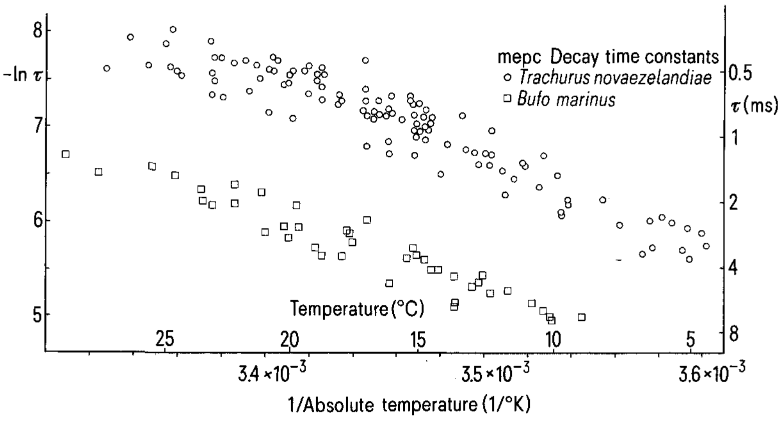
Summary. Teleost miniature end plate currents decay more rapidly than those of birds or tetrapods, with an exponential time constant of about 1 msec at 15 °C, but are otherwise normal. The rapid decay is correlated with increased unsaturation and greater fluidity of fish lipids.

The correlation between anesthetic potency and lipid solubility, which the synergistic/antagonistic effects of temperature/pressure on anesthesia, have led to hypotheses that all 3 agents interact with membrane lipids to alter fluidity, either in bulk, or in localized domains adjacent to ion channels[2,3]. The vertebrate neuromuscular junction has furnished support for this hypothesis[4], with the duration of miniature end plate currents (MEPCs) being decreased by anesthetics[5,6], higher alcohols[7,8], and elevated temperature[9]. A likely mode of action for these agents is an increase in membrane fluidity in the immediate vicinity of transmembrane channels, permitting faster relaxation of molecular gates.

Lipids extracted from the brains of fishes are more unsaturated (hence more fluid) than those of terrestrial vertebrates[10], and the fluidity of fish synaptic membranes has been correlated with lipid unsaturation[11]. Thus a comparative study of fish postsynaptic currents should serve as a test of the lipid fluidity hypotheses, which would predict that end plate currents in the more fluid fish membrane would decay more rapidly than those in tetrapods at any given temperature.

Specimens of *Trachurus novaezelandiae* Richardson, 1843 (Teleostei: Carangidae)[12] were captured in Port Jackson, Sydney, Australia, at an ambient temperature of 23 °C, and kept at 12-15 °C in a recirculating aquarium for 1 to 15 days. The inferior oblique extraocular muscle was dissected free and pinned in a perfusion chamber filled with physiological saline (231 mM NaCl, 2.7 mM KCl, 2.25 mM $CaCl_2$, 3.67 mM $MgCl_2$, 10 mM HEPES, pH 7.2). Focal extracellular recordings of spontaneous miniature endplate currents (MEPCs) were made from the white fiber band of the muscle with firepolished, blunt (30 µm) electrodes filled with 1 M NaCl or physiological saline. Because teleost motor innervation is usually distributed along the length of the muscle fiber, rather than being localized as in amphibia and mammals[13], end plates could not be located visually, but were found by sweeping the muscle surface with the



Comparison of MEPC decay time constants (τ) from fish (*Trachurus novaezelandiae*: circles) and toad (*Bufo marinus*: squares) muscle; Arrhenius plot. Each point represents the mean of 3-320 individual MEPCs, recorded by the same experimentor using identical techniques. Time constants for both animals are inverse exponential functions of temperature, but the fish MEPCs are consistentyl 3-4 times faster than toad MEPCs.

extracellular electrode. MEPCs were stored as FM tape recordings, and analyzed by averaging and/or semilogarithmic regression on a PDP-8 computer.

The fish MEPCs decayed as a single exponential function, and at all temperatures were significantly shorter than tetrapod MEPCs recorded under the same conditions (fig.) The fish MEPCs were also consistently shorter than published values for other twitch-type fibers: at 15 °C, the exponential time constant of decay (τ_D) for *T. novaezelandiae* MEPCs was approximately 1 msec, in contrast to 2 msec for mouse omohyoideus[14], 3 msec for toad sartorius[9], 5 msec for snake costocutaneous[15], and about 20 msec for chicken posterior latissimus dorsi[16], measured at the same temperature. In *Rana temporaria,* MEPCs from fast *m. pyriformis* fibers have decay time constants of 5 msec at 6 °C[17], which again is longer than τ_D for fish extraocular muscle (3-4 msec at 6 °C). Time constants reported for slow muscle fibers are generally several times longer than those of fast fibers at any given temperature[15,17], although in the chicken there is no significant difference between decay rates of fast and slow fibers[16]. Rise times for the fish MEPCs (T_G: the time elapsed between 20 and 80% of maximum deflection) were also quite fast, being about 120 µsec at 15 °C.

Apart from their short duration, *Trachurus* MEPCs were otherwise normal, with τ_D decreasing exponentially with temperature ($-\ln\tau_D = 41.66 - 83/RT$; R = gas constant, 8.32 J mole^{-1}deg^{-1}; T = absolute temperature, °K) (fig.). The Arrhenius temperature coefficient for rate of MEPC decay was 83 kJ mole^{-1}, which is within the range of other vertebrate preparations: mouse 66.2 kJ[14], toad 75.9 kJ[9], chicken 73-86 kJ[16]. There is a hint of a break around 17 °C in the Arrhenius plot of fish MEPCs, which may represent a phase change in membrane lipid, as reported for locust muscle[18].

The effect of membrane potential on τ_D was also comparable to that of tetrapod preparations[9,19,20], showing an e-fold increase in τ_D per 100 mV hyperpolarization in the voltage range between +25 and −110 mV. Voltage-clamped τ_D and T_G were somewhat longer than in extracellular MEPCs, which was probably due to non-focal clamping. In healthy cells, where MEPCs produced an extracellularly recorded voltage deflection of 350-500 µV, resting membrane potential was about −60 mV.

Drug effects on *Trachurus* MEPCs were also normal: at 15 °C, neostigmine (6 µM) increased τ_D about 2-fold, and 0.5 M ethanol increased τ_D approximately threefold.

The correlation reported here, between the fast decay of fish MEPCs and the increased unsaturation and fluidity of fish CNS lipids, is in general agreement with lipid hypotheses of anesthetic action. Provided that the increased fluidity of neural membranes is also reflected in the post-synaptic muscle membrane, it seems clear that changes in lipid fluidity do influence the time course of synaptic events; however this does not preclude additional sites or mechanisms for anesthesia.

1 The work reported here was undertaken at the University of New South Wales, Sydney, Australia, while on sabbatical leave from the University of Auckland. I wish to thank the Auckland University Council for a travel grant, and Professor P.W. Gage of the School of Physiology and Pharmacology, UNSW, for making facilities available in his laboratory. I am especially grateful to Dr R. Balnave for his generous assistance and advice, and to Dr K. Takeda for the voltage clamp measurements.
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0014-4754/83/020230-02$1.50 + 0.20/0

Reduction of fitness in *Drosophila* adults surviving parasitization by a cynipid wasp

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Summary. Drosophila adults emerging from larvae which successfully eliminated a parasite egg through the formation of a cellular melanotic capsule, were characterized by a decrease of their size and other biometrical traits and by an increase of the variability among individuals. Adults females also exhibited a significant reduction of offspring number due to lower oviposition rate and lower egg hatchability.

With respect to their impact upon natural populations, parasitic wasps are comparable to predators[1] since the development of the wasp implies the death of the host. The persistence of the host population is possible because parasite females cannot discover and attack all the possible hosts individuals and also because, in some cases, defense reactions may kill the parasite.

Natural populations are the theater of a classical coevolu-