mammary tumors in vivo during their growth and regression after hormone withdrawal or treatment with dibutyryl cyclic AMP. Table 1 shows a similar inverse relation between cyclic AMP–dependent protein kinase type II and estrogen binding in MCF-7 cells. By DEAE-cellulose column chromatography we found that the cytosols from growing MCF-7 cells exhibited two major peaks of protein kinase activity that was stimulated by cyclic AMP: one eluted at low ionic strength [type I enzyme (*12*)] and the other eluted at high ionic strength [type II enzyme (*13*)]. Both types I and II kinase activities were also detected in MCF-7 cell nuclei but in greatly reduced amounts. At 3 days after the treatment of cells with dibutyryl cyclic AMP plus arginine, type II protein kinase activity increased twofold in the cytosol whereas type I enzyme activity decreased by 40 percent (Table 1). Moreover, type II enzyme activity showed a fourfold increase in the nuclei of the growth-arrested cells, whereas type I enzyme activity did not change (Table 1). Table 1 also shows that in the growth-arrested cells estrogen binding activity decreased by 30 percent and 50 percent in the cytosol and nuclei, respectively. Thus the decreases in both estrogen binding and type I cyclic AMP–dependent protein kinase activities were inversely related with the increase of type II protein kinase activity in the growth-arrested MCF-7 cells. The results suggest that estrogen receptor and type I cyclic AMP–dependent protein kinase may serve as a positive effector for cell growth, whereas type II protein kinase serves as a negative signal.

The growth arrest of MCF-7 cells by arginine and dibutyryl cyclic AMP was accompanied by a striking change in cell morphology. As shown in Fig. 2, the cytoplasm of the treated cells was greatly enlarged without appreciable change in the size of the nuclei (compare C with B). Upon removal of the supplement the cell morphology returned to that of untreated cells and the cell number increased (Fig. 2D). Cyclic AMP–induced inhibition of cell proliferation is often associated with a change in cell morphology, synthesis of specialized cell products and cell differentiation (*14*). Since both arginine-induced and dibutyryl cyclic AMP–induced growth inhibition of MCF-7 cells was preceded by an increase in cellular cyclic AMP, the morphological change may be specifically associated with the increase in cyclic AMP.

The results of this study in vitro confirms our previous finding in vivo (*15*) that dibutyryl cyclic AMP and arginine inhibit the growth of mammary tumors. Our results, therefore, suggest that dibutyryl cyclic AMP and arginine may have therapeutic potential for breast cancer in humans.

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Skeletal Muscle: Length-Dependent Effects of Potentiating Agents

Abstract. *The ability of vertebrate skeletal muscle to contract more vigorously than normal in the presence of potentiating agents depends on the initial length of a muscle cell. Other factors such as the intracellular calcium ion transient, temperature, chemical nature of the potentiating agent, and the ratio of intrinsic twitch to tetanic force influence the degree of contractile potentiation but cannot account for the length dependence. At least part of a muscle cell seems normally less than fully active during contractions not only at short lengths but also at optimal sarcomere lengths.*

The sliding filament theory of muscle contraction adequately accounts for the progressive decline in force development when filament overlap is reduced by stretch (*1*). But when muscle is allowed to shorten below the sarcomere length at which filament overlap is optimal, there is a progressive decline in contractility that may be due to several factors (*1, 2*). A factor suggested several years ago is that the degree of activation may be progressively reduced as a muscle shortens (*3*). Activation in vertebrate skeletal muscle is known to be caused by a transient increase in intracellular free Ca^{2+}, and we sought to monitor length-dependent changes in free calcium transients and further study their influence on the contractility of complete muscle cells (*4*). If the intracellular Ca^{2+} concentration in cells with a whole assemblage of parts could be reversibly induced to approach or exceed the amount required to saturate calcium binding sites, length-dependent variations in Ca^{2+} transients should become less influential.

Live, intact cells were isolated from the tibialis anterior, semitendinosus, and iliofibularis of the frog *Rana temporaria* and studied before and after exposure to several chemical agents known to potentiate twitches (*5*). Striation spacings were measured optically before contraction as described (*2*). The cells were also microinjected with the Ca^{2+}-sensitive luminescent protein aequorin (*4*) to allow a correlation among intracellular Ca^{2+} transients, force of contraction, and some parameters (for example, temperature, ratio of initial twitch to tetanus force) previously shown to influence the degree of potentiation in whole muscle (*5–7*). The most reproducible results are obtained when contractions are preceded by a long period of rest. Aequorin responses and tetanic force are reduced when muscles are stimulated frequently, although twitch force is potentiated

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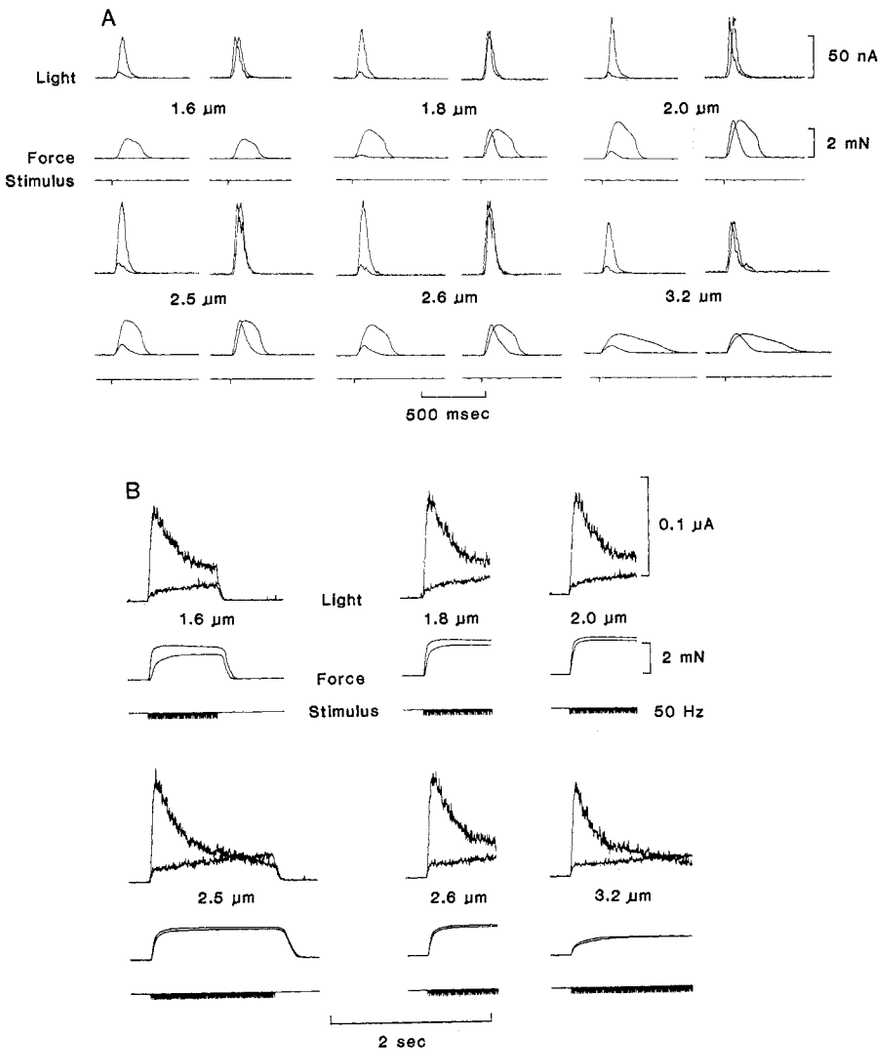

Fig. 1. (A) Effects of Zn^{2+} (50 μM) on aequorin signals and force responses in twitches at various lengths. The left-hand set of records in each of the six groups shown are the original superimposed recordings of the control and potentiated responses. The right-hand set of traces are the same responses adjusted to be equal in amplitude. The time to peak contraction is not only longer during potentiation—as is well known (*5*)—but the time to peak of the calcium transient is also shifted slightly to the right. The rate of decay of light emission is not noticeably different in control and potentiated aequorin responses, which supports the idea that potentiation primarily involves the enhancement and prolongation of Ca^{2+} release rather than selective slowing of the rate of Ca^{2+} removal. The rate of decay of force is clearly slowed in potentiated contractions, which is also well known (*5*). The observations here and previously (*4*) that aequorin transients fall much more quickly than force support the suggestion frequently made [for example, see (*9*)] that one or more features other than the rate of removal of myoplasmic free Ca^{2+} control the rate of decay of contractile activity. The initial twitch to tetanus force ratio of whole frog muscle is about 0.25 at its length in situ (about 2.5 μm) at room temperature (*5*). The ratio for the fibers illustrated in (B) and in Fig. 2, A and B, was greater than 0.3 at 2.5 μm and 15°C, which is comparable. The values for this relatively "low twitch" fiber were 0.17 at 2.0 μm, 0.25 at 2.5 μm, 0.27 at 2.6 μm, and 0.40 at 3.2 μm (tibialis anterior fiber, 5.ix.79). (B) Effects of Zn^{2+} (50 μM) on aequorin signals and force responses in tetani at various lengths. The fiber was stimulated by 1-msec d-c pulses applied transversely at 50 Hz until force ceased to rise or began to fade. The duration of a tetanus at each given length was, therefore, adjusted to assure the minimum degree of fatigue and to minimize the development of a "delta state" (*12*) in shortened fibers. Aequorin responses in potentiating solutions, nevertheless, showed considerable fade after a large initial burst of light. The fade could be accounted for by at least three possible changes: (i) a temporary local depletion of aequorin around release sites; (ii) a temporary local depletion of Ca^{2+} from intracellular release sites; or (iii) a temporary local depletion of Ca^{2+} from extracellular release sites, for example, depletion of Ca^{2+} in the T system (*13*). The first two possibilities are unlikely to be the entire explanation because high-frequency transverse alternating current produced light emission and force that was sustained for many seconds without appreciable fade (*14*). However, the cause of fade late in the tetanus was not examined further in these experiments. The initial twitch to tetanic force ratio of this fiber was 0.25 at 2.0 μm, 0.32 at 2.5 μm, 0.33 at 2.6 μm, and 0.51 at 3.2 μm and 15°C (tibialis anterior fiber, 7.ix.79).

when muscles are not in a rested state. (*4*, *6*).

The agents tested [Zn^{2+}, NO_3^-, tetraethylammonium ions (TEA^+), and caffeine] enhanced aequorin luminescence during electrical stimulation at all lengths, at cold and warm temperatures, and in fibers with a wide range of ratios of intrinsic twitch to tetanic force. The degree of enhancement was essentially independent of length. But this extra Ca^{2+} did not produce the same effect on contractility at all lengths. The degree of force potentiation in twitches or tetani was always inversely related to the cell length. We infer that Ca^{2+}-induced activation is normally less than maximum along the ascending limb and in the plateau region of the length-tension relation, and approaches maximum only along the descending limb.

The potentiating agents studied in greatest detail were small amounts of Zn^{2+} added to the bath, and NO_3^- in replacement for Cl^- present in the normal bathing solution (*5–7*). However, the addition of TEA^+ (2 to 25 mM) or caffeine (1 mM) to the normal saline solution produced qualitatively the same effects.

Figure 1A shows records of the effects of 50 μM Zn^{2+} on Ca^{2+} transients and twitch force development in an isolated skeletal (tibialis anterior) muscle fiber at 15°C. Figure 1B illustrates the effects of the same amount of Zn^{2+} on aequorin signals and the force of tetanic contractions of another fiber. The effects were qualitatively the same in either twitches or tetani and at both cold and warm temperatures. The relative increase in an aequorin response during contraction was, in a measure, independent of cell length. However, twitch force and steady-state force were enhanced more at short lengths than at the optimum sarcomere length (that is, 2.0 μm for tetani and 2.5 μm for twitches in the fibers in Fig. 1); steady-state tetanic force was essentially unaffected by a potentiating agent when a fiber was stretched to a length well along the descending limb of the length-tension relation (for example, 3.0 to 3.2 μm in Figs. 1B and 2B). Force in tetani also developed at a faster rate during the time that aequorin signals were enhanced, which agrees with the idea that the amount of activating Ca^{2+} has a marked influence on the rate of increase in the number of force-producing cross-bridges (*8*).

The length dependence of force is not readily explained by the temperature, some specific effect of Zn^{2+}, or an insufficiently long period of stimulation to

allow force to attain its highest possible level (for example, Fig. 2). In Fig. 2A the fiber was stimulated at 5-minute intervals in the sequence shown by the numbers alongside each data point. The effect in the cold was less, but clear. Similar effects of Zn^{2+} and NO_3^- on twitches of whole muscles at cold temperatures have been reported (*7*). The effect on tetanic force became progressively smaller as a fiber was cooled, but the change was smoothly graded and reversible. Thus, if the effect does ever completely vanish it presumably does so at temperatures lower than the lowest we tested (that is, 0° to 5°C).

Since striation spacing and aequorin light emission could not be measured simultaneously, it is possible that the differences could be due to changes in the degree of uniformity in striation spacing. Potentiators might reduce the internal axial translocations that occur during steady-state force development in fixed-end tetani (*9*). If sarcomere lengths became more nearly uniform at the same fiber length this might account for the changes in force shown in Figs. 1 and 2. But in other experiments the time course of shortening of the innermost myofibrils was examined with a microscope during application of current to a segment in the middle of the fibers (*2*). The potentiators did indeed increase the synchrony of shortening, but the change was along the radius of a fiber rather than along its axis. The average striation spacing and orientation of the myofibrils in the optical section through the center of a fiber was measured from each of a series of cinephotomicrographs taken during shortening before and after the addition of one of the potentiators mentioned. The entire fiber was evidently not activated maximally throughout contraction in normal saline solutions, because the shortening velocity of the central sarcomeres became less than the velocity of the peripheral sarcomeres and, therefore, the central myofibrils were pushed into waves. This change was, in all respects, the same as that previously observed (*2*).

In contrast, when a current pulse of the same strength and duration was applied to a fiber treated with one of the potentiating agents, the maximum shortening velocity of the central sarcomeres was markedly increased during the initial few tens of milliseconds, and throughout the contractions in potentiating solutions all of the myofibrils shortened at the same rate and none became wavy. Fibers also shortened more in the presence of a potentiator than they did in control solutions. This raises the possibility that there was a significant amount of internal shortening in fixed-end tetani too. Striation spacing cannot be measured or controlled by either laser diffraction or methods for following cathode-ray tube light spots (*1, 9*) if aequorin light emission is to be monitored simultaneously in a totally darkened environment (*4*). But if potentiators did cause greater internal shortening in our fixed-end tetani, the steady state for force must have been achieved at lengths shorter than those reached in the control responses, and the level of force should have been smaller rather than larger as we observed (Figs. 1B and 2B).

Fibers with an intrinsically low ratio of twitch to tetanic force (low twitch) had a maximum for twitch force at considerably longer lengths than fibers with a high twitch to tetanus force ratio, and showed more potentiation at any given length (for example, Fig. 1A). The relative increase in aequorin responses was approximately the same despite these wide-ranging differences in the effect on force. This variability of the optimum length for twitch force has already been reported for whole muscle as well as isolated fibers (*10*). The potentiators shifted the optimum length for twitch force to the same length at which the optimum for tetanic force occurred in addition to producing effects of greater magnitude on these "low twitch" fibers. These changes are consistent with the idea that a low Ca^{2+} concentration produces peak force as filament overlap is decreased by stretch, and a high Ca^{2+} concentration produces maximum force when filament overlap is optimal (*11*). In our experiments, a correlation between the degree of twitch force potentiation at

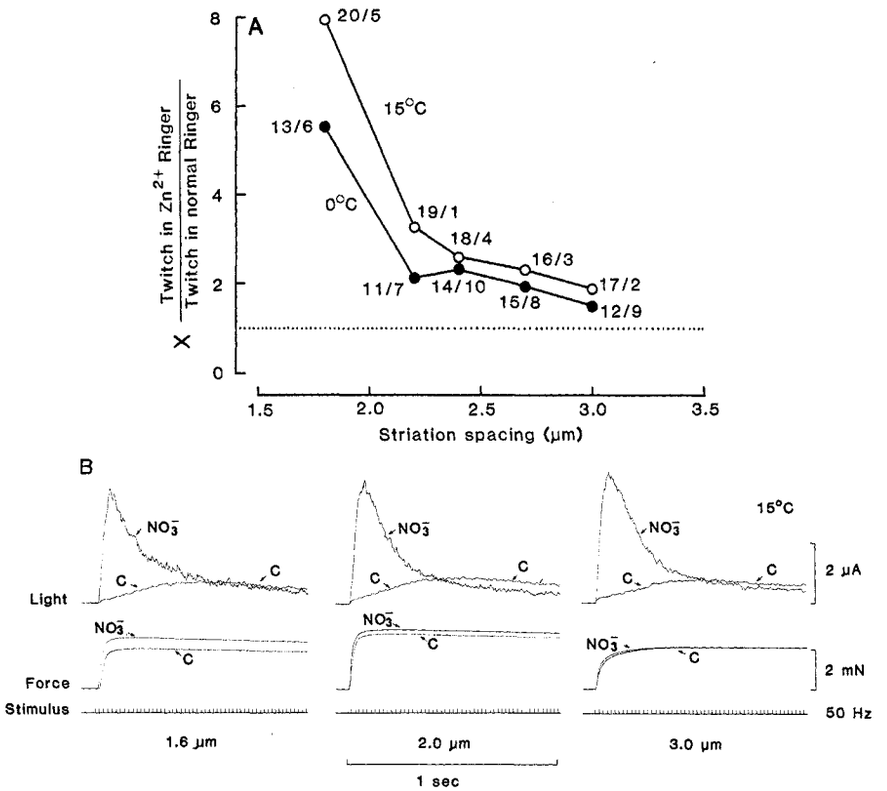

Fig 2. (A) The effect of temperature on the relative degree of twitch potentiation by 50 μM Zn^{2+}. All points were obtained from single responses of the same fiber, which had an initial twitch to tetanus ratio at 15°C of 0.32 at 2.2 μm, 0.36 at 2.4 μm, 0.43 at 2.8 μm, and 0.52 at 3.0 μm. The responses were elicited in the order indicated by the number alongside each data point. Twitch force was enhanced more as a fiber was allowed to contract to shorter lengths. But the effect was smaller at zero degrees than at 15°C. The same trend was observed in steady-state tetanic force of fibers studied at different temperatures; the absolute effects were smaller in the cold, but were distinguishable when several responses at the same length, different temperatures, and in the presence or absence of Zn^{2+} were compared (iliofibularis fiber, 20.xii.78). (B) The effect of Cl^- substitution by NO_3^- on tetanic responses. Tetanic light signals are initially enhanced early in the tetanus but fall to or below the level of the control responses. This may be due in part to the possibilities discussed in the legend of Fig. 1B. But it evidently is not related to an insufficient quantity of Ca^{2+} bound to the myofilaments, because steady-state force is enhanced in a sustained manner both before (Fig. 1B) and after (Figs. 1B and 2B) the "crossover" in light. The initial twitch to tetanus force ratio of this fiber was 0.26 at 2.0 μm, 0.32 at 2.6 μm, and 0.48 at 3.0 μm and 15°C (iliofibularis fiber, 3.x.78).

short lengths and "low twitch" values was strikingly apparent, as already reported for whole muscle (*6*). The same correlation was evident not only for twitches but also for tetanic force in the steady state. But the increased light emission was not correlated with the ratio of initial twitch to tetanus force. The unexceptionable association, therefore, was not among relative enhancement of intracellular Ca^{2+} transients, temperature, type of agent, or intrinsic twitch to tetanic force ratio. Cell length was the most consistent factor.

We conclude that these observations support and extend previous suggestions about the relative degree of activation in skeletal muscle allowed to shorten (*2*). Calcium-induced activation is progressively less than maximum at all muscle lengths shorter than those along the descending limb of the length-tension relation. In addition, the optimum length for twitch force is, by itself, neither an adequate means to assure that muscles are studied near the same average striation spacing nor is it, as the sole criterion, an adequate starting point to estimate the relative efficacy of the potentiating ability of an agent.

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Intense Natural Selection in a Population of Darwin's Finches (Geospizinae) in the Galápagos

Abstract. *Survival of Darwin's finches through a drought on Daphne Major Island was nonrandom. Large birds, especially males with large beaks, survived best because they were able to crack the large and hard seeds that predominated in the drought. Selection intensities, calculated by O'Donald's method, are the highest yet recorded for a vertebrate population.*

There are few well-documented examples of natural selection causing avian populations to track a changing environment phenotypically. This is partly because birds meet environmental challenges with remarkable behavioral and physiological flexibility (*1*), partly because birds have low reproductive rates and long generation times, and partly because it has been difficult for ecologists to quantify corresponding phenotypic and environmental changes in most field studies. In this report we demonstrate directional natural selection in a population of Darwin's finches and identify its main cause.

We studied Darwin's medium ground finch (*Geospiza fortis*) on the 40-ha islet of Daphne Major, the Galápagos, from July 1975 to June 1978. Each of more than 1500 birds was color-banded and measured for seven external morphological characters (*2*). Continuous records were kept of the banded birds and of rainfall. Each year during the breeding season (January to May) we banded nestlings and compiled nest histories. Three times a year (before, during, and after the breeding season) we collected the following data: (i) the number of seeds of each plant species in 50 randomly chosen 1.0-m^2 quadrats; (ii) a standardized visual census of finches over the entire island; and (iii) a minimum of 100 point records of feeding behavior, accumulated by noting food items eaten by banded birds encountered during nonsystematic searches (*2*).

During the early 1970's Daphne Major received regular rainfall, resulting in large finch populations and food supplies (*2*). From December through June in 1976 and 1978 we recorded rainfalls of 127 and 137 mm, respectively—sufficient for abundant production of plants, insects, and finches. However, in 1977 only 24 mm of rain fell on Daphne Major during the wet season (*3, 4*). *Geospiza fortis* did not breed at all in 1977 and suffered an 85 percent decline in population (Fig. 1A). The decline was correlated with a reduction in seed abundance ($r = .86$, $P < .01$) (Fig. 1B). Seeds form the staple diet of *G. fortis*, particularly in the dry season, when other plant matter and insects are scarce (*2*).

Between June 1976 and March 1978, the mortality, and possibly emigration (*5*), of *G. fortis* was nonrandom with respect to age, sex, and phenotype. Only one of 388 *G. fortis* nestlings banded in 1976 survived to 1978, and while the sex ratio was roughly equal in 1976, it had become skewed to six males to one female in 1978. Most significantly, the birds surviving into 1978 were considerably larger than those that disappeared (Fig. 1C). We use principal component 1 (*6*) as an index of overall body size because here, as in other avian studies (*7*), it explains a substantial portion (67 percent) of the phenotypic variance in the *G. fortis* population and has consistently high, positive correlations with the morphological variables it summarizes. The change is most obvious in the plot including all birds because it incorporates the changing sex ratio (most of the morphological characters are 4 percent larger in males than in females) and perhaps a small age effect, although all birds less than 12 weeks old were excluded from the analysis.

Small seeds declined in abundance faster than large ones, resulting in a sharp increase in the average size and hardness of available seeds (Fig. 1D). There was a corresponding change in feeding behavior. In May 1976 only 17 percent of feeding was on medium or large seeds [size-hardness index $\sqrt{DH} \geq 1.0$ (*8*)], while in May 1977 49 percent of feeding was on such seeds. During the present and related studies (*2*), large birds ate larger seeds than smaller birds, suggesting that small birds disappeared because they could not find enough food. For example, in a quantitative test of size-related feeding behavior, 198 birds that were only recorded eating seeds with a size-hardness index < 1.0 were significantly smaller than another 121 birds that routinely ate seeds with size-hardness indices ranging from 1.0 to 8.7 (*8*). In 1977, during the normally lush wet season, larger birds fed heavily on seeds extracted from the large, hard mericarps of *Tribulus cistoides* ($\sqrt{DH} = 8.68$), a food item ignored by almost all birds in earlier years (*2*). Many finches failed to molt that year, and their condition gradually deteriorated. Small

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