duced a series of reviews that will be standard reading for the next five years or so. I mean this as a compliment; it is a measure of how far and fast marine ornithology has come in the last two decades. The next stateof-the-art review will inevitably have this one as a cornerstone.

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Muscle Contraction

Calcium in Muscle Activation. A Comparative Approach. JOHANN CASPAR RUEGG. Springer-Verlag, New York, 1986. xiv, 300 pp., illus. \$98. Zoophysiology, vol. 19.

This book explores "the common principles that govern calcium signalling in the regulation of muscle contraction and cell motility," describing those "features of calcium regulation [that] may be common or fundamental to all muscles" and those that "are probably specializations that have evolved because of their suitability for specific functions" (p. 250). Physiology is the central focus; nevertheless, the approach is broad. Findings on intact muscles are analyzed with the aid of experiments on permeabilized preparations and isolated proteins. Relevant structural and biochemical studies are also considered in the evaluation of possible mechanisms.

The comparative approach employed by Rüegg is particularly suited for the elucidation of muscle function. In recent decades, muscle has become an outstanding example of how the convergence of different disciplines can lead to a fuller understanding of function. Identification of the muscle proteins and knowledge of their reactions have been of paramount importance in interpreting muscle structure. Structural studies have led to our present understanding of contraction at the molecular level. Kinetics of the actomyosin-ATPase cycle are closely interwoven with analysis of mechanical transients. Our understanding of the structural, biochemical, kinetic, and mechanical properties of muscles has advanced in a mutually dependent fashion.

The knowledge of control of contraction has been particularly enriched by comparative studies. Contraction of all muscles is triggered by the appearance of calcium in the myoplasm. Calcium is released from membrane-bound compartments as a result of depolarization of the cell membrane that may or may not be propagated depending

on muscle types and on species. Inwardconducting T-tubule membranes may or may not be present. The roles of specific calcium channels of the cell membrane and of secondary messengers such as cyclic AMP and inositoltrisphosphate in the production of calcium transients depend on muscle types and species. The variations that contribute to the adaptation of a particular muscle to specific functions, including speed, duration, efficiency, rhythmicity, and oscillation, are described in detail, and their effects are carefully evaluated.

Calcium is a universal triggering ion since proteins can bind it with a high affinity and it can move rapidly to and from the binding site. Thus, at rest, its myoplasmic concentration can be kept low, and relatively small amounts need be released for activation. The binding site representing a particular amino acid sequence, the E-F hand, is present in a number of calcium-binding proteins: troponin-C, calmodulin, parvalbumin, and myosin light chains. Since calmodulin is a subunit of a number of proteins, it serves as a calcium sensitizer of several different systems including myosin light-chain kinase.

Regulation of contraction is essentially the inhibition of the interaction between actin and myosin. Calcium reverses this inhibition. The "on-off" switch, however, is quite different in different muscles and species. In vertebrate striated and cardiac muscles the regulatory proteins, the tropomyosin-troponin complex, are part of the thin filaments and act on actin. In molluscan muscles, vertebrate smooth muscles, and various motile cells, myosin itself is regulated, and the light chains serve as regulatory subunits. Molluscan myosins bind calcium directly; in contrast, smooth-muscle and nonmuscle myosins are activated by phosphorylation of the regulatory light chains catalyzed by the calcium-dependent myosin light-chain kinase.

Scallop myosin is uniquely suited for the study of the role of light chains in the regulation of myosin since its regulatory light chains can be reversibly removed. The isolated myosin is controlled with the same fidelity as intact muscle, and activation by calcium is associated with the rearrangement of light chains. Activation of vertebrate smooth muscles and other motile cells by means of the myosin light-chain kinase involves a cascade of events and opens up access routes to hormones and to cyclic AMP to modulate the speed and extent of light-chain phosphorylation.

The book is a rich source of experimental background on these processes. The reference list is large (over 800 papers), up-todate, and valuable. The condensed style and large amount of material presented do not

make for rapid reading, but the book is carefully organized, with useful summaries after each chapter. The text is illustrated with a generous number of figures that help to acquaint readers with the experimental background of the conclusions and interpretations.

This is a rapidly growing field. Unanswered important questions remain. These include the mechanism of calcium release, the molecular mechanism of the action of regulatory proteins, the role of thin-filament regulation in smooth muscles, the nature and control of the maintained tension state, and "latch" and "catch" in smooth and molluscan muscles. Unresolved issues are pointed out without oversimplification, and alternatives in interpretation are presented fairly.

This is an unusually useful book for researchers and also for graduate and advanced undergraduate students interested in regulation. A less expensive paperback edition would facilitate the wide circulation it deserves.

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