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Kenneth Axen, Kathleen Vermitsky Axen

The Sarcomere and Skeletal Muscle Disease Nigel G. Laing, 2009-09-29

Although best known for its role in heart disease, the sarcomere--the fundamental unit of muscle contraction--is also involved in skeletal muscle diseases. Chapters in *The Sarcomere and Skeletal Muscle Disease* provide an up-to-date review of diseases caused by mutated proteins in the different sub-compartments of the sarcomere, document the techniques currently being used to investigate the pathobiological bases of the diseases, which remain largely unknown, and discuss possible therapeutic options.

Connecting Sarcomere Protein Mutations to Pathogenesis in Myopathies Jose Renato Pinto, P. Bryant Chase, 2020-08-06

Sliding Filament Mechanism in Muscle Contraction Haruo Sugi, 2007-04-27
Sliding Filament Mechanism in Muscle Contraction: Fifty Years of Research covers the history of the sliding filament mechanism in muscle contraction from its discovery in 1954 by H.E. Huxley through and including modern day research. Chapters include topics in dynamic X-ray diffraction, electron microscopy, muscle mechanisms, in-vitro motility assay, cardiac versus smooth muscle, motile systems, and much more.

Anatomy & Physiology Lindsay Biga, Devon Quick, Sierra Dawson, Amy Harwell, Robin Hopkins, Joel Kaufmann, Mike LeMaster, Philip Matern, Katie Morrison-Graham, Jon Runyeon, 2019-09-26 A version of the OpenStax text

Myofibrillogenesis Dipak K. Dube, 2012-12-06 Myofibrillogenesis has been studied extensively over the last 100 years. Until recently, we have not had a comprehensive understanding of this fundamental process. The emergence of new technologies in molecular and cellular biology, combined with classical embryology, have started to unravel some of the complexities of myofibril assembly in striated muscles. In striated muscles, the contractile proteins are arranged in a highly ordered three dimensional lattice known as the sarcomere. The assembly of a myofibril involves the precise ordering of several proteins into a linear array of sarcomeres. Multiple isoforms in many of these proteins further complicate the process, making it difficult to define the precise role of each component. This volume has been compiled as a comprehensive reference on myofibrillogenesis. In addition, the book includes reviews on myofibrillar disarray under various pathological conditions, such as familial hypertrophic cardiomyopathy (FHC), and incorporates a section on the conduction system in the heart. Much of the information in this volume has not been described elsewhere. Presented in a manner to be of value to students and teachers alike, Myofibrillogenesis will be an invaluable reference source for all in the fields of muscle biology and heart development.

Sarcomere Behaviour of Twitch and Tonic Muscle Fibres of the Frog L. Penney, 1981

Skeletal Muscle Damage and Repair Peter M. Tiidus, 2008 Attempts to cover a wide range of both basic research and applied clinical topics related to skeletal muscle damage and repair mechanisms and their application. This book examines muscle damage and repair mechanisms and issues in specific populations including older adults and special populations.

Mechanism of Myofilament Sliding in Muscle Contraction Haruo Sugi, Gerald H. Pollack, 2012-12-06 This volume presents the entire proceedings of the symposium organized by one of us (H. S.) on November 11 to 15, 1991 at Hakone, Japan, under the title of Mechanism of Myofilament Sliding in Muscle Contraction. Among various kinds of energy transduction mechanisms in biological systems, the mechanism of muscle contraction has been studied most intensively and extensively over many years. Since the monumental discovery by the two Huxleys and coworkers that muscle contraction results from relative sliding between the thick and thin myofilaments, attention of muscle investigators has been focused on the question, what makes the filaments slide past one another. In response to the above question, A. F. Huxley and Simmons put forward a contraction model in 1971, in which globular heads of

myosin (cross-bridges) extending from the thick filament first attach to actin on the thin filament, and then change their angle of attachment to actin (power stroke) leading to force generation or myofilament sliding until they detach from the thin filament. The rocking cross-bridge contraction model seemed to be entirely consistent with the kinetic scheme of actomyosin ATPase published by Lymn and Taylor at the same time, thus giving a strong impression to the people concerned that the muscle contraction mechanism would soon be sorted out. In his review lecture in 1974, however, A. F.

Muscular Contraction Robert Malcolm Simmons, 1992-06-26 Composed of a set of chapters contributed by past and present collaborators of the Nobel laureate Sir Andrew Huxley, this book covers the areas of muscle research to which Huxley made major contributions. The purpose of the book is to discuss the way that muscles work, asking questions at a fundamental level about the molecular basis of muscle tone production and muscle contraction. The majority of the chapters are concerned with muscle physiology and the relation between structure and function. The process of activation of muscles is discussed, along with the mechanism of contraction itself. Although most of the book deals with vertebrate skeletal muscle, several chapters cover cardiac muscle. Also featured are two chapters discussing Sir Andrew's achievements in both nerve and muscle physiology.

Elastic Filaments of the Cell H.L. Granzier, Gerald H. Pollack, 2012-12-06 Elastic filaments refer mainly to titin, the largest of all known proteins. Titin was discovered initially in muscle cells, where it interconnects the thick filament with the Z-line. Titin forms a molecular spring that is responsible for maintaining the structural integrity of contracting muscle, ensuring efficient muscle contraction. More recently, it has become clear that titin is not restricted to muscle cells alone. For example, titin is found in chromosomes of neurons and also in blood platelets. This topic is fast becoming a focal point for research in understanding viscoelastic properties at the molecular, cellular, and tissue levels. In titin may lie a generic basis for biological viscoelasticity. It has become clear that titin may hold the key to certain clinical anomalies. For example, it is clear that titin-based ventricular stiffness is modulated by calcium and that titin is responsible for the altered stiffness in cardiomyopathies. It is also clear from evidence from a group of Finnish families that titin mutations may underlie some muscular dystrophies and that with other mutations chromatids fail to separate during mitosis. Thus, it is clear that this protein will have important clinical implications stemming from its biomechanical role. One aspect of this field is the bringing together of bioengineers with clinical researchers and biologists. Genetic and biochemical aspects of titin-related proteins are being studied together with front-line engineering approaches designed to measure the mechanics of titin either in small aggregates or in single molecules.

The Kinetics of Muscle Contraction David Clifford Stephen White, John Thorson, 1975

Towards an Active Matter Perspective on Sarcomere Dynamics in Cardiomyocytes Daniel Härtter, 2023

Reflections on Muscle Andrew Huxley, Sir Andrew Huxley, 1980

Anatomy and Physiology J. Gordon Betts, Peter DeSaix, Jody E. Johnson, Oksana Korol, Dean H. Kruse, Brandon Poe, James A. Wise, Mark Womble, Kelly A. Young, 2013-04-25

Skeletal Muscle Mechanics W. Herzog, 2000-10-03 Dieses Teilgebiet der Biomechanik ist für Sportwissenschaftler und Physiologen von großer Bedeutung! Die umfassende, aktuelle Abhandlung der Skelettmuskelmechanik beschäftigt sich mit drei Themenkreisen: den Mechanismen der Skelettmuskelkontraktion, der Muskelfunktion in vivo und theoretischen Modellen der Muskelfunktion. Auch ein knapper historischer Abriss und ein Ausblick auf noch offene Fragen fehlen nicht. (08/00)

Botulinum Neurotoxins Andreas Rummel, Thomas Binz, 2012-12-14 The extremely

potent substance botulinum neurotoxin (BoNT) has attracted much interest in diverse fields. Originally identified as cause for the rare but deadly disease botulism, military and terrorist intended to misuse this sophisticated molecule as biological weapon. This caused its classification as select agent category A by the Centers for Diseases Control and Prevention and the listing in the Biological and Toxin Weapons Convention. Later, the civilian use of BoNT as long acting peripheral muscle relaxant has turned this molecule into an indispensable pharmaceutical world wide with annual revenues >\$1.5 billion. Also basic scientists value the botulinum neurotoxin as molecular tool for dissecting mechanisms of exocytosis. This book will cover the most recent molecular details of botulinum neurotoxin, its mechanism of action as well as its detection and application.

Physiology Coloring Workbook Kenneth Axen, Kathleen Vermitsky Axen, 1997
 Physiology Coloring Workbook is a breakthrough approach to learning and remembering the body's processes. Written and illustrated by experts who are both research scientists and teachers, it features 250 striking, original illustrations that will give students a clear and enduring understanding of physiology. Learning interactively, through coloring, thoroughly fixes physiological concepts in the mind and takes less time than memorizing from textbooks. Physiological processes are fully explained, and complex subjects are approached through the gradual introduction of simple drawings. The authors employ a logical and consistent use of color to convey information; for example, arterial blood is always red, whereas venous blood is blue, and capillary blood is violet. Each lesson includes clearly displayed labels and specific coloring instructions. This book is an invaluable and lasting resource for students in disciplines including anatomy and physiology, biology, nursing, physical therapy and rehabilitation, medical technology, nutrition, physical education, allied health and health sciences. The 250 plates in the book are organized in the following sections: Homeostasis The Cell Transport Mechanisms Nervous System Muscle Cardiovascular System Renal System Respiratory System Gastrointestinal System Metabolism Endocrine System Reproduction

Role of Telethonin in Sarcomere Assembly and Maintenance Takrima Sadikot Pirani, Michael Bryan Ferrari, 2010
 Telethonin is found in the striated muscle, where it tethers together N-termini of two titin molecules. The kinase domain of titin (TK) phosphorylates and the C-terminal end of telethonin and this phosphorylation is thought to be important in regulating the interaction of both proteins in the sarcomere. While some studies suggest that telethonin is dispensable for sarcomere assembly, truncation mutations in telethonin are associated with a form of autosomal recessive limb-girdle muscular dystrophy type 2G (LGMD 2G). Here I used an anti-sense morpholino (MO+) approach to knockdown telethonin expression in *Xenopus* embryos. MO+ embryos are paralyzed and show severe sarcomeric disruption when compared to control embryos. In MO+ whole embryos and cell cultures stained for myosin or actin, very little or no de novo sarcomere formation is observed, suggesting a role for telethonin in sarcomere assembly. Injecting exogenous full-length telethonin mRNA (FL: 1-176) in the MO+ background restores motility and myofibrillogenesis. Several C-terminal mutant constructs were designed and tested for rescue. These included: 1) A truncation of the C-terminal phosphorylatable region (C'del: 1-163) and two different telethonin constructs encoding mutations in the C-terminal phosphorylatable residues 2) C'Ala (S164A, T166A, S168A, S170A) and 3) C'mis (163LSRTIVSRIT173). Rescue experiments with the C'del and C'Ala in the MO+ background do not relieve the paralytic phenotype or restore sarcomeric organization. However, the C'mis rescues at early stages followed by rapid and progressive degeneration of the sarcomeric structure. This result is particularly interesting since it may mimic LGMD 2G in myocytes. To determine if the phosphorylatable residues at the telethonin C-terminus are required for sarcomere organization, I tested additional constructs with individual alanine substitutions and two

constructs containing phosphorylation mimics. My results demonstrate that telethonin is required for sarcomeric organization and that phosphorylation at its C-terminal end may be essential for its function.

Starling's Law of The Heart Revisited Henk Keurs, M.I.M Noble, 2012-12-06 H. E. D. J. TER KEURS & M. I. M. NOBLE The Starling's Law of the Heart and The Frank-Starling Mechanism have long been the cornerstone of cardiac mechanical physiology. It is often forgotten that Frank and Starling carried out fundamentally different experiments. Frankl measured the isovolumic pressure developed by frog heart at different volumes. He therefore discovered the pressure-volume-volume relationship which depends directly on the force-length relationship of the 2 sarcomeres. Starling ,3 studied cardiac shortening as manifest by cardiac output and its relationship to end-diastolic conditions as manifest by right atrial pressure. Thus he was studying the ability of cardiac muscle to shorten more at a given load from a greater initial length. Starling in the promulga 4 tions of his law implied a common mechanism for these two phenomena and spoke of the energy liberated being a function of initial muscle fiber length. However, there has been much confusion about the interrelationship between the two different aspects studied by Frank and Starling. The 1960s saw the era of isolated cardiac muscle mechanics, beginning with 5 the paper of Abbott and Mommaerts. Whole muscle length-tension relations were equated with sarcomere-length-tension relations by fixation of muscle at a particular point on the curve and determination of sarcomere length by electronmicroscopy.

Mathematical Modelling of the Half-sarcomere from a Human Skeletal Muscle
Kay P. Guest, 2013

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Table of Contents Srcomjer

1. Understanding the eBook Srcomjer
 - The Rise of Digital Reading Srcomjer
 - Advantages of eBooks Over Traditional Books
2. Identifying Srcomjer
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an Srcomjer
 - User-Friendly Interface
4. Exploring eBook Recommendations from Srcomjer
 - Personalized Recommendations
 - Srcomjer User Reviews and Ratings
 - Srcomjer and Bestseller Lists

5. Accessing Srcomjer Free and Paid eBooks
 - Srcomjer Public Domain eBooks
 - Srcomjer eBook Subscription Services
 - Srcomjer Budget-Friendly Options
6. Navigating Srcomjer eBook Formats
 - ePub, PDF, MOBI, and More
 - Srcomjer Compatibility with Devices
 - Srcomjer Enhanced eBook Features
7. Enhancing Your Reading Experience
 - Adjustable Fonts and Text Sizes of Srcomjer
 - Highlighting and Note-Taking Srcomjer
 - Interactive Elements Srcomjer
8. Staying Engaged with Srcomjer
 - Joining Online Reading Communities
 - Participating in Virtual Book Clubs

- Following Authors and Publishers Srcomjer
- 9. Balancing eBooks and Physical Books Srcomjer
 - Benefits of a Digital Library
 - Creating a Diverse Reading Collection Srcomjer
- 10. Overcoming Reading Challenges
 - Dealing with Digital Eye Strain
 - Minimizing Distractions
 - Managing Screen Time
- 11. Cultivating a Reading Routine Srcomjer
 - Setting Reading Goals Srcomjer
 - Carving Out Dedicated Reading Time
- 12. Sourcing Reliable Information of Srcomjer
 - Fact-Checking eBook Content of Srcomjer
 - Distinguishing Credible Sources
- 13. Promoting Lifelong Learning
 - Utilizing eBooks for Skill Development
 - Exploring Educational eBooks
- 14. Embracing eBook Trends
 - Integration of Multimedia Elements
 - Interactive and Gamified eBooks

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