

References

- Campbell, K. P., Knudson, C. M., & Imagawa, T., et al. (1987). Identification and characterization of the high affinity [³H]ryanodine receptor of the junctional sarcoplasmic reticulum Ca²⁺ release channel. *The Journal of Biological Chemistry*, 262, 6460–6463.
- Cannon, S. C. (2006). Pathomechanisms in channelopathies of skeletal muscle and brain. *Annual Review of Neuroscience*, 29, 387–415.
- Durham, W. J., Wehrens, X. H., Sood, S., & Hamilton, S. L. (2007). Diseases associated with altered ryanodine receptor activity. *Subcellular Biochemistry*, 45, 273–321.
- Engel, A. G., Shen, X. M., Selcen, D., & Sine, S. M. (2010). What have we learned from the congenital myasthenic syndromes? *Journal of Molecular Neuroscience*, 40, 143–153.
- Farrugia, M. E., & Vincent, A. (2010). Autoimmune mediated neuromuscular junction defects. *Current Opinion in Neurology*, 23, 489–495.
- Franzini-Armstrong, C. (1979). Studies of the triad. I. Structure of the junction of frog twitch fibers. *The Journal of Cell Biology*, 47, 488–499.
- Hibberd, M. G., & Trentham, D. R. (1986). Relationships between chemical and mechanical events during muscular contraction. *Annual Review of Biophysics and Biophysical Chemistry*, 15, 119–161.
- Hovnanian, A. (2007). SERCA pumps and human diseases. *Subcellular Biochemistry*, 45, 337–363.
- Huxley, H. E. (1969). The mechanism of muscle contraction. *Science*, 164, 1356–1366.
- Jones, H. R., Jr., De Vivo, D. C., & Darras, B. T. (Eds.), (2003). *Neuromuscular disorders of infancy, childhood, and adolescence—a clinician’s approach*. Philadelphia: Butterworth-Heinemann.

- Karlin, A., & Akabas, M. H. (1995). Toward a structural basis for the function of nicotinic acetylcholine receptors and their cousins. *Neuron*, *15*, 1231–1244.
- Lee, J. E., & Cooper, T. A. (2009). Pathogenic mechanisms of myotonic dystrophy. *Biochemical Society Transactions*, *37*, 1281–1286.
- Montecucco, C., Rossetto, O., Caccin, P., Rigoni, M., Carli, L., & Morbiato, L., et al. (2009). Different mechanisms of inhibition of nerve terminals by botulinum and snake presynaptic neurotoxins. *Toxicon*, *54*, 561–564.
- Pascual, J. M., & Karlin, A. (1998). State-dependent accessibility and electrostatic potential in the channel of the acetylcholine receptor. *The Journal of General Physiology*, *111*, 717–739.
- Plaster, N. M., Tawil, R., & Tristani-Firouzi, M., et al. (2001). Mutations in Kir2.1 cause the developmental and episodic electrical phenotypes of Andersen's syndrome. *Cell*, *105*, 511–519.
- Pollard, T. D., & Cooper, J. A. (1986). Actin and actin-binding proteins. A critical evaluation of mechanisms and functions. *Annual Review of Biochemistry*, *55*, 987–1035.
- Ptacek, L., Tawil, R., & Griggs, R., et al. (1994). Dihydropyridine receptor mutations cause hypokalemic periodic paralysis. *Cell*, *77*, 863–898.
- Sabater, L., Titulaer, M., Saiz, A., Verschuuren, J., Güre, A. O., & Graus, F. (2008). SOX1 antibodies are markers of paraneoplastic Lambert-Eaton myasthenic syndrome. *Neurology*, *70*, 924–928.
- Steinmeyer, K., Klocke, R., & Ortland, C., et al. (1991). Inactivation of muscle chloride channel by transposon insertion in myotonic mice. *Nature*, *354*, 304–306.
- Terlau, H., & Olivera, B. M. (2004). *Conus* venoms: a rich source of novel ion channel-targeted

peptides. *Physiological Reviews*, 84, 41–68.

Wagenknecht, T., Grassucci, R., & Frank, I., et al. (1989). Three-dimensional architecture of the calcium channel/foot structure of sarcoplasmic reticulum. *Nature*, 338, 167–170.

Zot, A. S., & Potter, J. D. (1987). Structural aspects of troponin–tropomyosin regulation of skeletal muscle contraction. *Annual Review of Biophysics and Biophysical Chemistry*, 16, 535–560.