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Introduction

Regulation of Avian Skeletal Muscle Development and Maturation (43057)

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The chick has served as a useful model for studies on vertebrate development. Easy access to the embryo through the shell and the ready availability of large numbers of embryos at known developmental stages have contributed to the avian egg's popularity as a research model. Early research with the avian embryo described the differentiation and morphogenesis of the single cell egg into a free living chick (1). In the case of skeletal muscle, the origin and timing of mesodermal tissue differentiation and migration into somites and then skeletal muscle was followed. More recently, embryonic skeletal muscle precursors have been removed and cultured *in vitro*. Using combinations of *in vivo* and *in vitro* approaches, it has become apparent that the progenitors of muscle, the myoblasts, divide repeatedly and then cease making DNA and fuse with adjacent myoblasts to produce extended multicellular myotubes. DNA synthesis and nuclear division are not seen in multinucleated myotubes. After fusion, the myotubes begin to synthesize muscle-specific proteins such as creatine kinase, actin, tropomyosin, and myosin heavy chain. During the maturation process, there is an orderly progression in the type of myosin expressed until adult forms predominate. Once adult-type muscle is present, a cell similar to an embryonic myoblast, the satellite cell, is responsible for adding new DNA to existing myotubes to permit growth and for forming new myotubes following injury.

Many questions remain to be answered. Our current view of skeletal muscle development is mostly descriptive, with little appreciation for the signals and mechanisms which are responsible for the steady transition in cell number, morphology and function, the diversity of muscle types, and the growth and repair of existing muscle. This symposium addresses several areas of progress in understanding muscle development at the cellular level. It should be appreciated that remarkable progress has been made in the analysis of genes involved in the commitment of cells to become myoblasts and to express muscle-specific proteins (2, 3). Increases in our understanding of the regulation of muscle development at the gene level complements much of the information reviewed in this symposium.

Frank Stockdale considers the early events in the formation of limb musculature and presents evidence for the existence of a heterologous population of embryonic myoblasts, each type giving rise to a different fiber type as distinguished by the isoform of myosin heavy chain expressed and by fiber size. These populations of myoblasts arise sequentially during development and are probably not lineal descendants of one another. Daina Ewton and James Florini examine the external cues that regulate the process of myoblast proliferation and terminal differentiation. The number of cell divisions completed is important in determining the number and size of muscle fibers in the embryo and may set the upper size limit of the skeletal musculature in the growing animal. One factor, insulin-like

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growth factor, stimulates both proliferation and differentiation of myoblasts whereas a second factor, transforming growth factor-β, inhibits all aspects of myoblast differentiation. These factors may work in concert to influence the number of cell divisions completed by myoblasts before they fuse to form myotubes.

Regulatory events that influence the size and number of skeletal muscle fibers in the postembryonic animal are considered through analysis of satellite cell function by Ronald Allen and Lucinda Rankin. They present information on factors that may determine whether satellite cells in the fully developed skeletal muscle proliferate, differentiate, or remain quiescent. Insulin-like growth factor-I, fibroblast growth factor, and transforming growth factor-β, are important regulators *in vitro* and evidence is accumulating that supports their importance *in vivo*.

The coordination of skeletal muscle development may receive inappropriate instructions as the result of genetic anomalies. Barry Wilson discusses aberrations in development and maturation processes that result in various pathologies of skeletal muscle. Genetic, anatomical, and physiologic aspects of skeletal muscle that combine to result in functional skeletal muscle are realized by the study of muscle pathology.

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