Meat Science 109 (2015) 40–47

Contents lists available at ScienceDirect

Meat Science

journal homepage: www.elsevier.com/locate/meatsci

Review

Fetal programming in meat production

Min Du [a,*], Bo Wang [a], Xing Fu [a], Qiyuan Yang [a], Mei-Jun Zhu [b]

[a] Department of Animal Sciences, Washington State University, Pullman, WA 99164, United States
[b] School of Food Science, Washington State University, Pullman, WA 99164, United States

ARTICLE INFO

Article history:
Received 5 January 2015
Received in revised form 14 April 2015
Accepted 16 April 2015
Available online 23 April 2015

Keywords:
Adipogenesis
Fetal programming
Meat
Myogenesis
Progenitor cells
Quality

ABSTRACT

Nutrient fluctuations during the fetal stage affects fetal development, which has long-term impacts on the production efficiency and quality of meat. During the early development, a pool of mesenchymal progenitor cells proliferate and then diverge into either myogenic or adipogenic/fibrogenic lineages. Myogenic progenitor cells further develop into muscle fibers and satellite cells, while adipogenic/fibrogenic lineage cells develop into adipocytes, fibroblasts and resident fibro-adipogenic progenitor cells. Enhancing the proliferation and myogenic commitment of progenitor cells during fetal development enhances muscle growth and lean production in offspring. On the other hand, promoting the adipogenic differentiation of adipogenic/fibrogenic progenitor cells inside the muscle increases intramuscular adipocytes and reduces connective tissue, which improves meat marbling and tenderness. Available studies in mammalian livestock, including cattle, sheep and pigs, clearly show the link between maternal nutrition and the quantity and quality of meat production. Similarly, chicken muscle fibers develop before hatching and, thus, egg and yolk sizes and hatching temperature affect long-term growth performance and meat production of chicken. On the contrary, because fishes are able to generate new muscle fibers lifelong, the impact of early nutrition on fish growth performance is expected to be minor, which requires further studies.

Contents

1. Introduction

Forage and feed deficiency is a common occurrence in the livestock industry in many areas of the world. Such deficiency could be due to drought which results in a significant reduction in forage production subjecting cows and ewes to nutrient deficiency during gestation (Du et al., 2013). In addition, nutrient deficiency could also be due to the lack of feeds and the poor quality of feeds, rendering sows and hens to nutrient deficiency (Altmann et al., 2012).

Fetal programming, also called developmental programming or fetal developmental programming, is the response to a specific challenge to

* Corresponding author. Tel.: +1 509 335 2744; fax: +1 509 335 1082.
E-mail address: min.du@wsu.edu (M. Du).

http://dx.doi.org/10.1016/j.meatsci.2015.04.010
0309-1740/

the mammalian organism during a critical developmental time window that alters the trajectory of development qualitatively and/or quantitatively with resulting persistent effects (Nathanielsz, Poston, & Taylor, 2007). Studies linking fetal programming to animal performance in livestock and other experimental species were initiated relatively recently. In these studies, both malnutrition and over-nutrition during gestation impacts offspring growth performance (Benyshek, Johnston, & Martin, 2004; Bieswal et al., 2006; Bispham et al., 2003; Desai, Gayle, Babu, & Ross, 2005; Fernandez-Twinn, Ekizoglou, Wayman, Petry, & Ozanne, 2006; Fernandez-Twinn et al., 2005; Ford et al., 2007; King, 2006; Long, Shasa, Ford, & Nathanielsz, 2012a; Long et al., 2012b; Meyer et al., 2013; Reynolds et al., 2010; Symonds, Pearce, Bispham, Gardner, & Stephenson, 2004; Vonnahme et al., 2013; Yan et al., 2013; Zambrano et al., 2006). Therefore, in addition to genetic background, proper fetal development is also important to maximize the growth potential of animals.

Meat animals are raised for their skeletal muscle. The fetal stage is crucial for skeletal muscle development in mammalian livestock, because there is no net increase in the muscle fiber number after birth (Stickland, 1978; Zhu, Ford, Nathanielsz, & Du, 2004). A decrease in the number of muscle fibers due to fetal programming permanently reduces muscle mass and negatively affects animal performance.

Marbling (intramuscular fat) is crucial for meat palatability and the fetal and neonatal stages are major stages for generation of intramuscular adipocytes (Tong et al., 2008), which provide the sites for intramuscular fat accumulation or marbling formation during fattening. Hence, fetal programming also affects the marbling in offspring cattle. In addition, the overall fat accumulation in offspring is also affected by maternal nutrition (Zhu et al., 2006).

In this review, we summarize recent progress in the impact of maternal nutrition on fetal development, focusing on myogenesis and intramuscular adipogenesis, as well as the impacts of fetal programming on the growth performance and meat quality of offspring of mammalian livestock. In the end, as a comparison, we also discuss the possible impacts of fetal programming on poultry and fish production.

2. Programming of skeletal muscle and adipose tissue development

2.1. Fetal programming of skeletal muscle development

Muscle cells, adipocytes and fibroblasts are all derived from the mesoderm. Specifically, skeletal muscle cells are developed from dermomyotome during the early embryonic development, while adipocyte progenitors are derived from lateral mesoderm (Fig. 1). Mesoderm cells undergoing myogenic commitment initially become Pax3 + and then Pax7 +; and the subsequent expression of Myf5 commits these cells to the myogenic lineage. Myogenic differentiation involves the sequential expression of MyoD, myogenin and MYF-4 (Buckingham et al., 2003; Collins et al., 2009). These myogenic transcription factors then initiate the expression of myogenic specific genes including embryonic myosin heavy chain. Interestingly, brown adipocytes can be developed from Myf5 + cells during fetal development (Seale et al., 2008). Recently, there is a new pool of brown-like adipocytes identified in adipose tissue, so called beige adipocytes. These cells also express UCP-1, generating heat (Harms & Seale, 2013). Selected markers for satellite cells, muscle fibers, fibroblasts, brown and beige adipocytes are listed in Table 1.

Ontogenetically, the early skeletal muscle development can be separated into primary myogenesis and secondary myogenesis. Primary myofibers form first during the embryonic stage, followed by the formation of secondary myofibers which accounts for the majority of muscle fibers in adults (Beermann, Cassens, & Hausman, 1978). There is no further increase in muscle fibers after birth, and postnatal muscle growth is mainly due to increase in muscle fiber size without forming new muscle fibers (Karunaratne, Ashton, & Stickland, 2005; Stickland, 1978). Satellite cells are derived from fetal myoblasts, which become quiescent and locate surrounding mature muscle fibers in postnatal muscle. The asymmetrical proliferation and myogenic differentiation of satellite cells, and their fusion with existing muscle fibers are crucial for postnatal muscle growth (Kuang, Kuroda, Le Grand, & Rudnicki, 2007).

Since the majority of muscle fibers, if not all, form during the fetal stage and myogenesis requires large amount of nutrients (Rehfeldt &

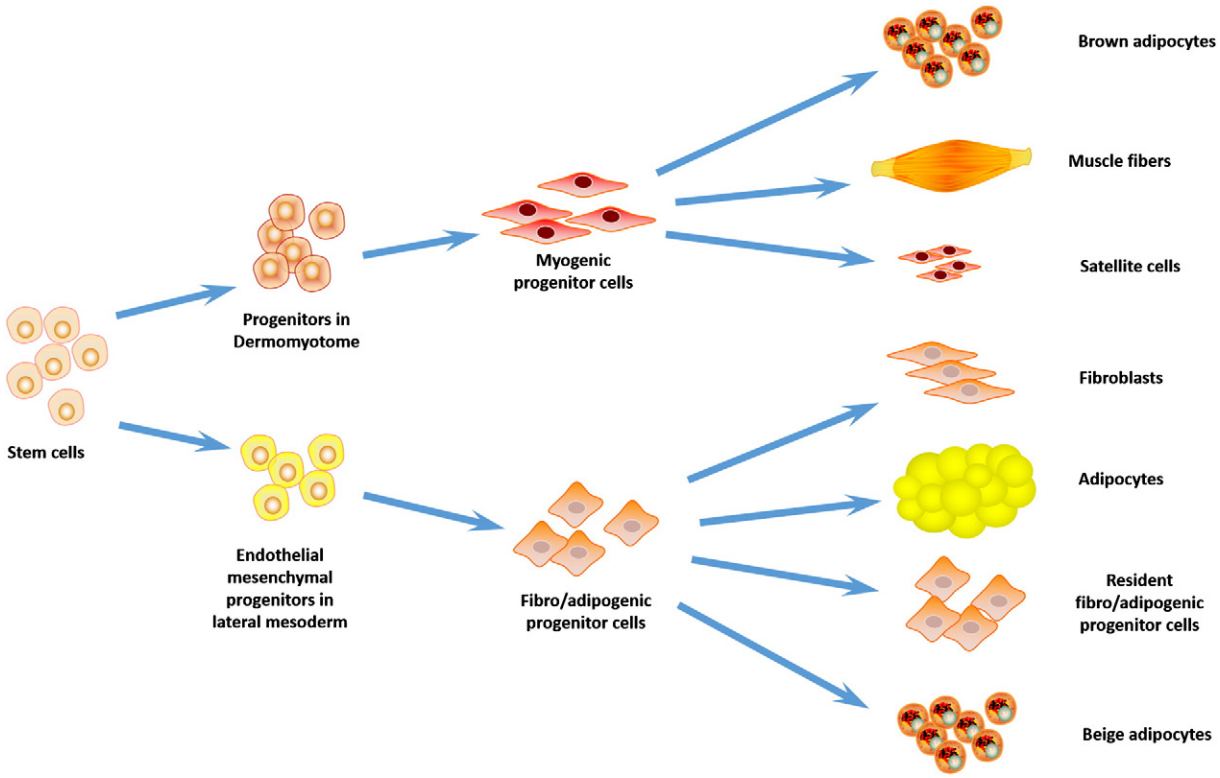

Fig. 1. Early mesoderm development and the commitment of mesenchymal progenitor cells into myogenic and fibro-adipogenic cell lineages during fetal muscle development.