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REVIEW ARTICLE

Perinatal Maternal Malnutrition and Its Impact on Skeletal Muscle Structure in Offspring: A Narrative Review

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ABSTRACT

Background: Maternal nutrition during the perinatal period is a critical determinant of fetal development, influencing organogenesis and tissue differentiation. Adequate nutrient supply supports the proper formation of organ systems, including skeletal muscle, which plays essential roles in locomotion, posture, metabolism, and growth. Skeletal muscle development begins early in embryogenesis and continues through the perinatal stage via tightly regulated processes of myogenesis, differentiation, and growth that are highly sensitive to the intrauterine environment. Maternal malnutrition during pregnancy or lactation, whether manifested as protein deficiency, excessive fat intake, or a combination of both, can profoundly disrupt skeletal muscle development in offspring. These nutritional imbalances reduce muscle mass and alter fiber composition and diameter, resulting in impaired metabolic capacity, weaker contractile function, a great risk of obesity, resistance to insulin, and metabolic disorders in offspring. This review aimed to provide an overview of the structural changes taking place in the skeletal muscles of offspring following maternal exposure to either low-protein or high-fat diet during pregnancy and lactation.

Conclusion: Optimal Perinatal maternal nutrition is pivotal for skeletal muscle development, and malnutrition can compromise myogenesis and metabolic health. The need for focused dietary strategies during pregnancy and lactation is highlighted by the Developmental Origins of Health and Disease (DOHaD) framework, which suggests that early nutritional problems may predispose offspring to long-term musculoskeletal and metabolic diseases.

Keywords: Perinatal maternal malnutrition; Skeletal muscle development; Protein deficiency; High-fat diet.

INTRODUCTION

Prenatal and the early postnatal periods are critical windows for tissue differentiation and organogenesis, during which cells undergo rapid growth and maturation. Adverse conditions during these stages, such as poor maternal nutrition or metabolic stress, can disrupt developmental programming and lead to permanent alterations in organ structure and function. As emphasized by the DOHaD framework, such early-life influences may significantly affect adult health, increasing susceptibility to chronic diseases later in life [1,2].

A balanced maternal diet during pregnancy and lactation is essential for normal skeletal muscle development in the fetus. Adequate nutrient supply supports myoblast proliferation, differentiation, and protein synthesis, ensuring proper fiber number, type composition, and growth. In contrast, nutritional imbalances can impair these processes, leading to reduced muscle

mass, altered fiber characteristics, and compromised metabolic function in offspring. Numerous studies highlight the strong influence of maternal nutrition on intrauterine skeletal muscle development, underscoring its importance for long-term musculoskeletal and metabolic health [3-5].

Malnutrition is increasingly recognized as a major global health concern, particularly in densely populated and underdeveloped regions. While it was once primarily attributed to poverty, food scarcity, and limited access to nutritious diets, the issue has become more complex in recent decades with the proliferation and aggressive marketing of inexpensive, energy-dense, nutrient-poor foods such as fast food and junk food. Recent evidence indicates that nearly half of women of reproductive age experience malnutrition during pregnancy. Importantly, perinatal maternal malnutrition whether due to a low-protein diet, a high-fat diet, or a combination of both has been shown to induce structural alterations in the skeletal muscles of offspring [6-8] .

A maternal low-protein diet lowers amino acid levels in the fetus, leading to poor growth, small birth-weight, and impaired development of skeletal muscles. These early deficits may persist into later life, increasing the risk of reduced muscle function and metabolic disorders [9,10].

In developed nations, obesity and associated metabolic diseases have become epidemics, with children seeing the fastest rate of rise. Exposure to unfavorable intrauterine settings, especially an increased food supply during development, can result in early-onset metabolic problems, according to evidence from human studies. Within the framework of DOHaD, such intrauterine overnutrition can permanently alter metabolic programming, affecting insulin sensitivity, adiposity, and energy balance. The importance of maternal nutrition in determining long-term health outcomes is highlighted by these early-life changes, which raise a person's risk of obesity, type 2 DM, and cardiovascular disease later in life [11,12]. Further, experimental animal models show that perinatal high-fat diets increase intramuscular lipid accumulation, trigger inflammation, disrupt mitochondrial morphology, and promote fiber-type shifts from oxidative to glycolytic, impairing glucose handling [13].

It was found that, high–fat diet consumption or cafeteria diet, throughout gestational and lactational period promoted increased intramuscular lipid accumulation in cross-sectioned areas of offspring skeletal muscles [14].

The purpose of this review was to clarify the structural changes in the skeletal muscles of offspring following maternal exposure to either a low-protein (LP) diet or high-fat (HF) diet through pregnancy and lactation.

Anatomy of skeletal muscle

Skeletal muscle, a striated tissue comprising about 40% of human body weight, is distributed superficially and primarily attached to bones. Each muscle functions as an organ composed of fibers, connective tissue, nerves, and blood vessels. Skeletal muscle is highly vascularized and innervated to meet its metabolic and contractile demands. Each fiber receives input from a somatic motor neuron, while accompanying arteries, veins, and nerves branch through the epimysium to form capillary networks around the fibers [15]. Structurally, the whole muscle is covered by a connective tissue sheath called the epimysium. Within the muscle, bundles of muscle fibers called fascicles, and each fascicle is surrounded by perimysium and contains individual muscle fibers, each enveloped by endomysium. These connective tissue layers extend beyond the muscle belly to form tendons or aponeuroses, which anchor to the periosteum of bones. Muscle contraction generates tension that is transmitted through these layers to the tendon, enabling skeletal movement [16]

[Fig. 1]. The hierarchical arrangement of connective tissue layers (endomysium, perimysium, epimysium) ensures mechanical strength and coordinated contraction. These structural layers are also critical targets of pathological remodeling under malnutrition, as collagen deposition and fibrosis within these sheaths have been consistently reported in offspring exposed to maternal LP or HF diets [17].

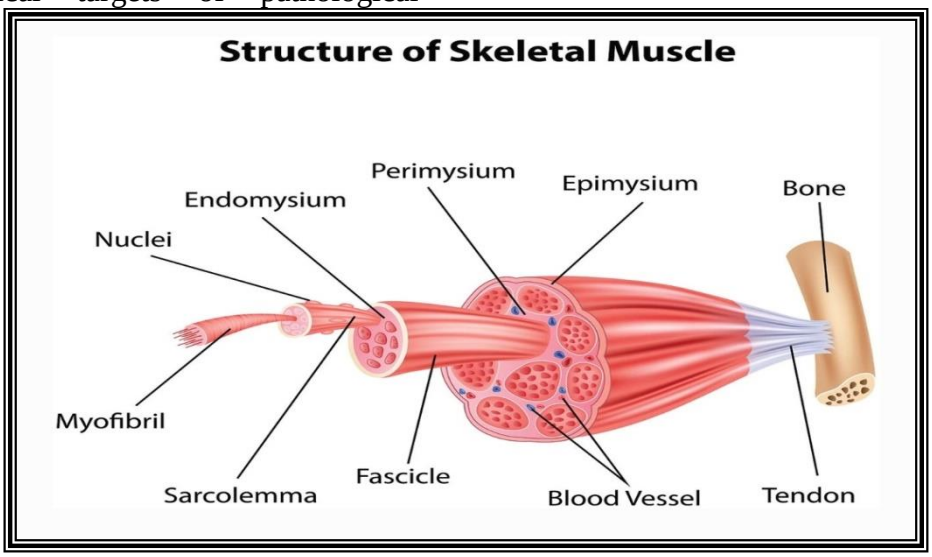


Fig. (1): A diagram of skeletal muscle shows three connective tissue layers: endomysium around individual fibers, perimysium around fascicles, and epimysium surrounding the entire muscle. These layers merge at the muscle end to form a tendon that attaches to bone. Blood vessels supply the fibers [16].

Histology of skeletal muscle

Skeletal muscle fibers are elongated, tubular, and striated cells with multiple peripheral nuclei, primarily composed of myofibrils. Each fiber is enclosed by the sarcolemma surrounded externally by a specialized basement membrane composed of a basal lamina and reticular lamina beneath which lies the sarcoplasm. The basal lamina links directly to the sarcolemma, while the reticular lamina, rich in collagen, connects to the endomysium; together they support muscle integrity, growth, and regeneration [18]. Myofibrils occupy 80–90% of muscle fiber volume and are built from myofilaments thick (myosin) and thin (actin) whose arrangement produces the characteristic light (I bands) and dark (A bands) that give skeletal muscle its striated appearance**.** The thick filaments form the A-band, with the central H-zone containing only thick filaments and the M-line linking myosin tails. The thin filaments define the I-band and are associated with regulatory proteins, tropomyosin and troponin, controlling contraction. The Z-line in the I-band connects thin filaments of adjacent sarcomeres. The sarcomere is the segment between Z-lines being the structural and functional unit of skeletal muscle. Each sarcomere contains a full A-band and halves of I-bands, and coordinated sliding of actin over myosin shortens myofibrils and the muscle fiber as a whole (Fig.2) [16,19].

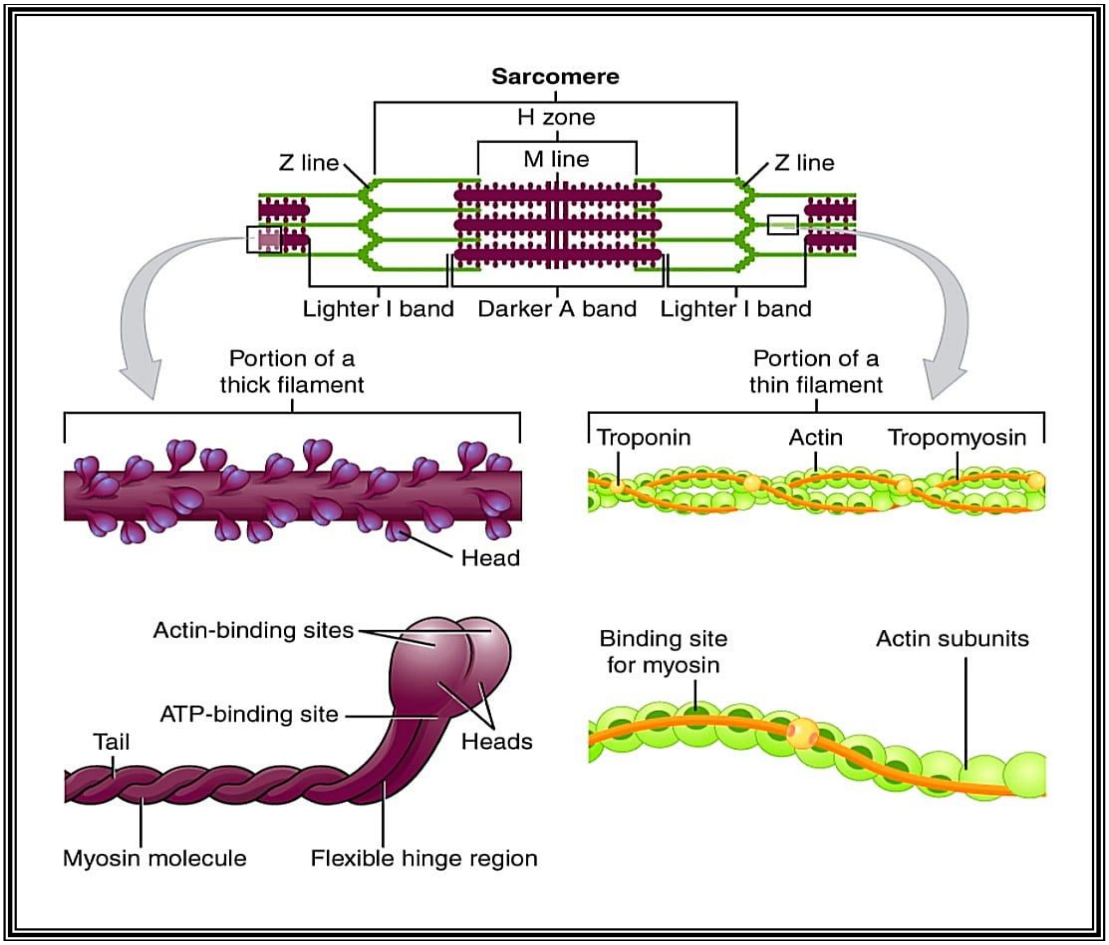


Fig. (2): A diagram showing the sarcomere of skeletal muscle fiber [19].

Myofibrils are surrounded by the sarcoplasmic reticulum (SR), which stores calcium ions and expands into terminal cisternae. Transverse tubules (T-tubules), formed by sarcolemma invaginations between terminal cisternae, create triads that transmit action potentials from the fiber surface, triggering calcium release and contraction via troponin binding. The sarcoplasm contains numerous elongated mitochondria between myofibrils, a small Golgi complex near the nucleus, scattered ribosomes, and inclusions such as glycogen granules, myoglobin, and lipofuscin pigments (Fig. 3) [16, 19]. At the microscopic level, sarcomere organization and mitochondrial density determine muscle contractile and metabolic efficiency. Disruption of these ultrastructural features such as fragmented Z-lines, swollen mitochondria, and decreased desmin expression serves as a hallmark of nutritional myopathy in offspring subjected to maternal malnutrition [20].

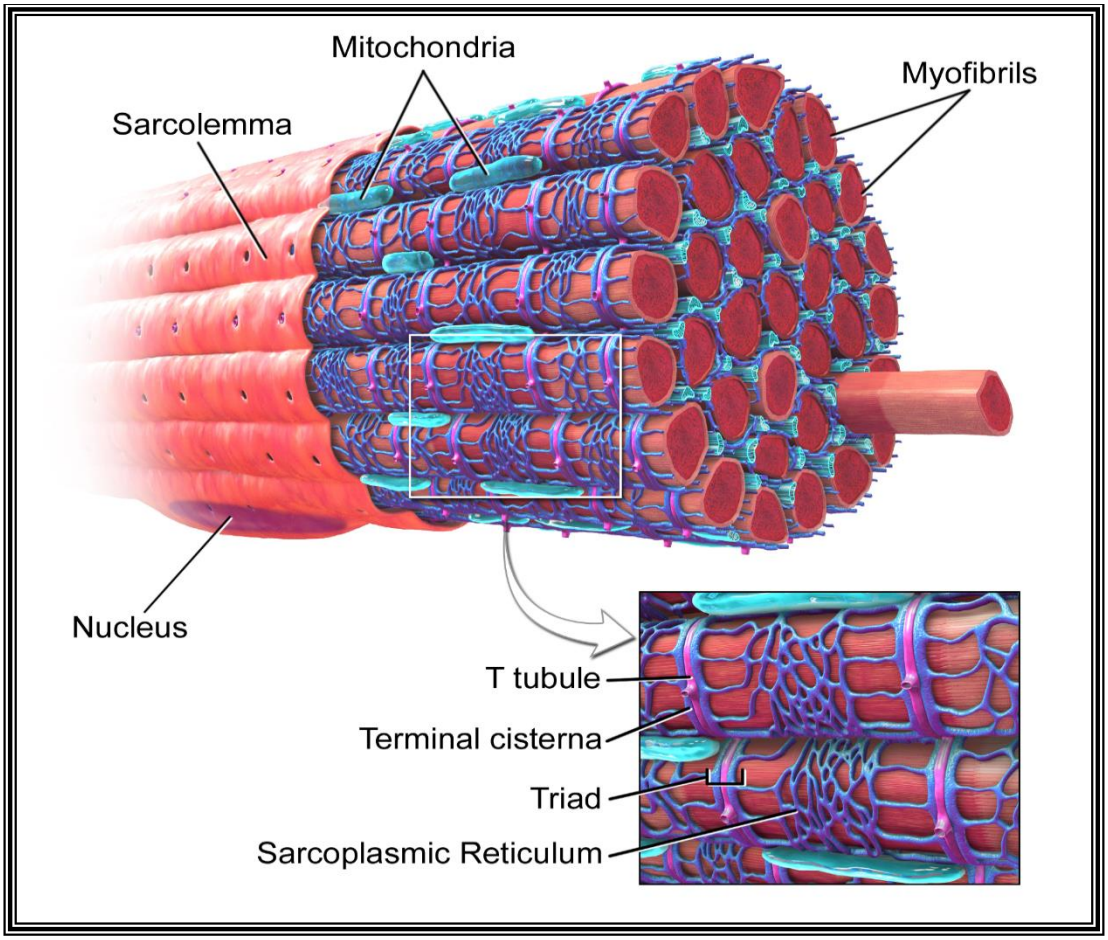


Fig. (3): Diagram of skeletal muscle fibers showing long cylindrical fibers composed of myofibril bundles surrounded by mitochondria and sarcoplasmic reticulum. The zoomed view highlights a T-tubule, an invagination of the sarcolemma between terminal cisternae [16].

Development of skeletal muscles

Skeletal muscles of the head, trunk, and limbs arise from somitomeres and somites, derivatives of the paraxial mesoderm. By the third week, the paraxial mesoderm segments into somitomeres from head to tail. Cranial somitomeres form mesenchyme that contributes to craniofacial muscles (with neural crest cells). From the occipital region caudally, somitomeres condense into paired somites, reaching 42–44 pairs by week five; some regress, while others form the axial skeleton [21]. Each somite differentiates into a sclerotome (vertebrae, ribs, sternum) and a dermomyotome, which gives rise to the myotome (skeletal muscles) and dermatome (dermis) (Fig. 4) [22]. Myotomal mesoderm gives rise to myoblasts, which elongate and fuse into multinucleated myotubes. Myotubes synthesize actin, myosin, and other proteins, forming myofilaments and myofibrils. Mature muscle fibers arise as nuclei shift peripherally, and bundles of fibers form muscles attached to skeletal elements [22]. Maternal malnutrition interferes with these tightly regulated stages, reducing myoblast proliferation, delaying differentiation, and impairing myotube fusion. Consequently, fewer mature fibers and smaller fiber diameters are observed in affected offspring [23].

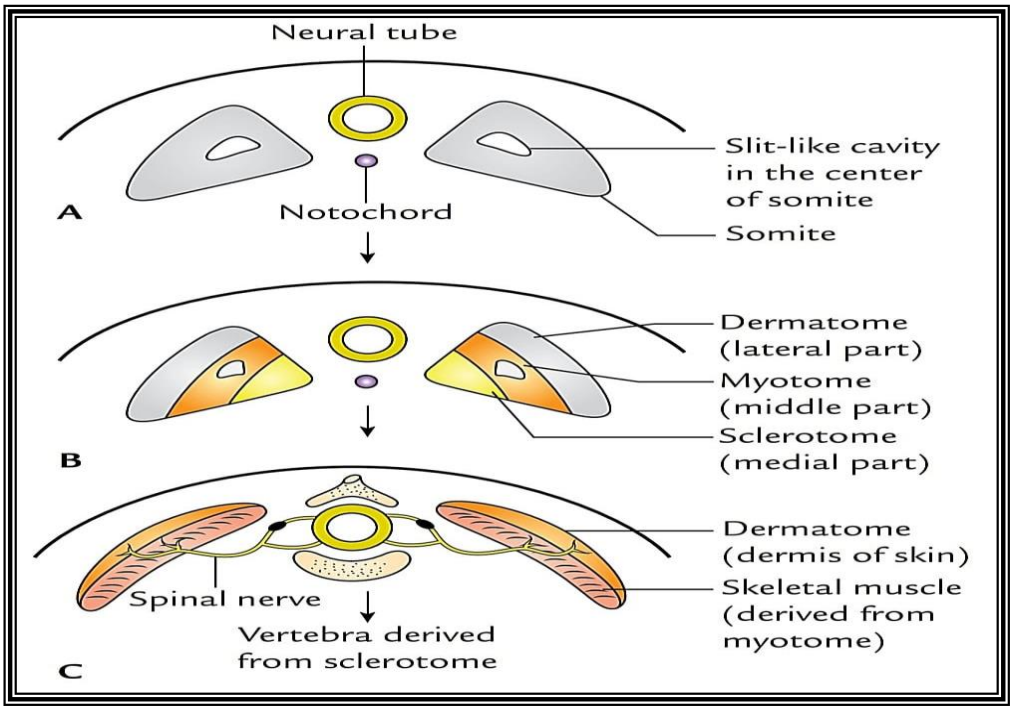


Fig. (4): A diagram showing subdivisions of the somite [22].

Structural Alterations in The Skeletal Muscles of the Offspring Induced by Maternal Low-Protein Diet during The Perinatal Period

1. Overview

Epidemiological and experimental evidence demonstrates that maternal low-protein (LP) intake during pregnancy and lactation adversely affects skeletal muscle development in offspring, predisposing them to metabolic disorders in later life [24]. Protein restriction limits the fetal amino acid supply, leading to low birth weight, reduced muscle fiber number, and altered stem cell activity [10]. Offspring nursed by LP-fed dams exhibit lower body and muscle mass, smaller fiber cross-sectional area, and persistent structural deficits even after post-weaning nutritional normalization [25].

2. Structural and Ultrastructural Alterations

Experimental models have shown that maternal LP diets disrupt skeletal muscle morphology, with offspring displaying sparse myofibrils, disorganized Z-lines, and delayed sarcomere maturation [26, 27]. These structural abnormalities are accompanied by mitochondrial swelling, fragmentation, and reduced oxidative enzyme activity, collectively impairing contractile efficiency. Prolonged exposure results in mitochondrial dysfunction, decreased mtDNA content, and downregulation of oxidative phosphorylation–related genes [9, 28]. In parallel, defective Akt–mTOR signaling, impaired insulin-stimulated protein synthesis, and diminished glycogen storage contribute to smaller muscle fibers and reduced contractile mass (Fig. 5) [29].

3. Histopathological Changes and Oxidative Stress

Perinatal protein restriction induces pronounced histological alterations in offspring skeletal muscles, characterized by thin, disorganized, and separated fibers, poorly defined striations, nuclear pyknosis, vascular congestion, and cellular infiltration [25]. LP exposure during critical developmental windows disrupts oxidative homeostasis and repair mechanisms, increasing vulnerability to oxidative stress. Elevated reactive oxygen species (ROS) and malondialdehyde (MDA) levels confirm enhanced lipid peroxidation and cellular damage [9].

4. Fibrosis and Extracellular Matrix Remodeling

Maternal LP diets also promote collagen accumulation and fibrosis in offspring skeletal muscles, attributed to impaired myogenesis and chronic low-grade inflammation. These processes hinder muscle regeneration and drive extracellular matrix (ECM) remodeling [29, 30]. Furthermore, significant glycogen depletion observed in LP-exposed muscles reflects defective insulin signaling and impaired glucose utilization [31].

5. Cytoskeletal Integrity and Desmin Expression

Desmin, the major intermediate filament protein in skeletal and cardiac muscles, preserves myofibrillar alignment by linking adjacent Z-discs and connecting the contractile apparatus to the sarcolemma, nucleus, and organelles. Desmin deficiency causes multisystem myopathies characterized by fibrosis, calcification, and structural instability [32]. Consistently, reduced desmin expression has been reported in LP diet–exposed offspring, correlating with compromised muscle architecture and impaired regenerative capacity [33].

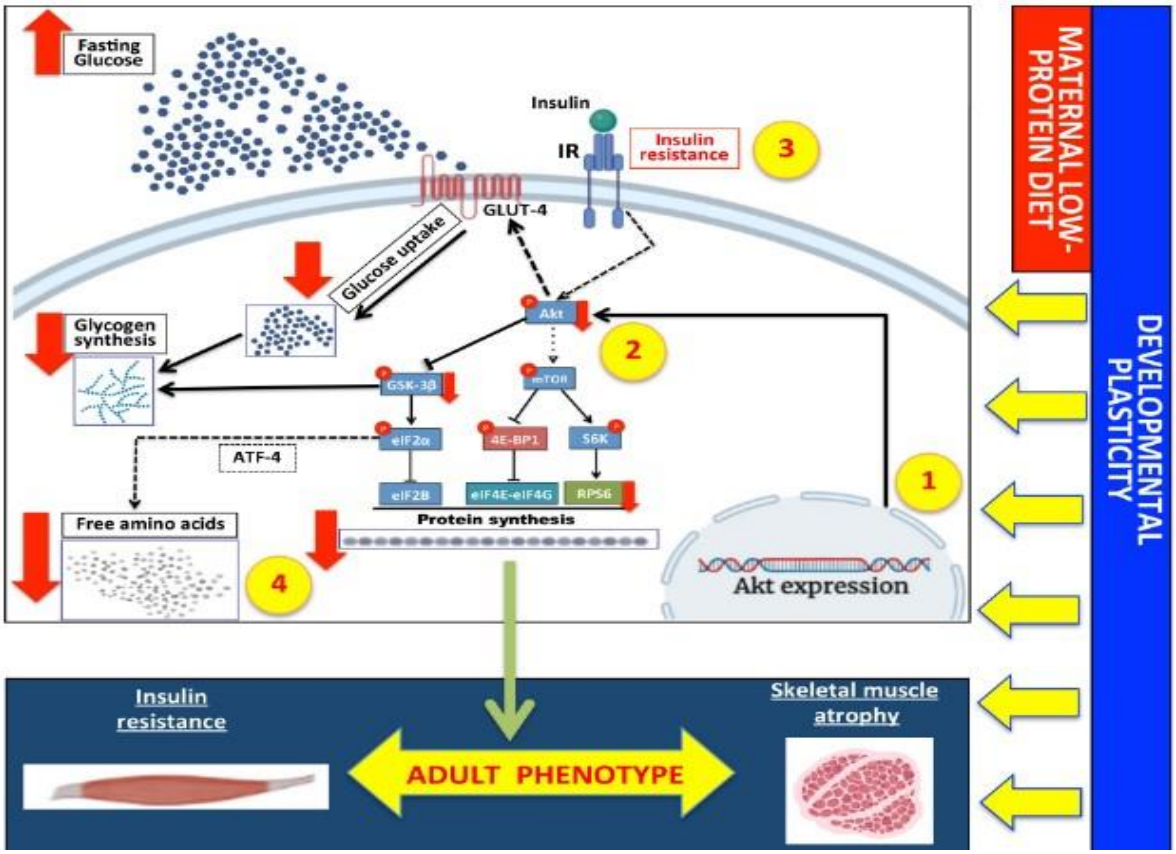
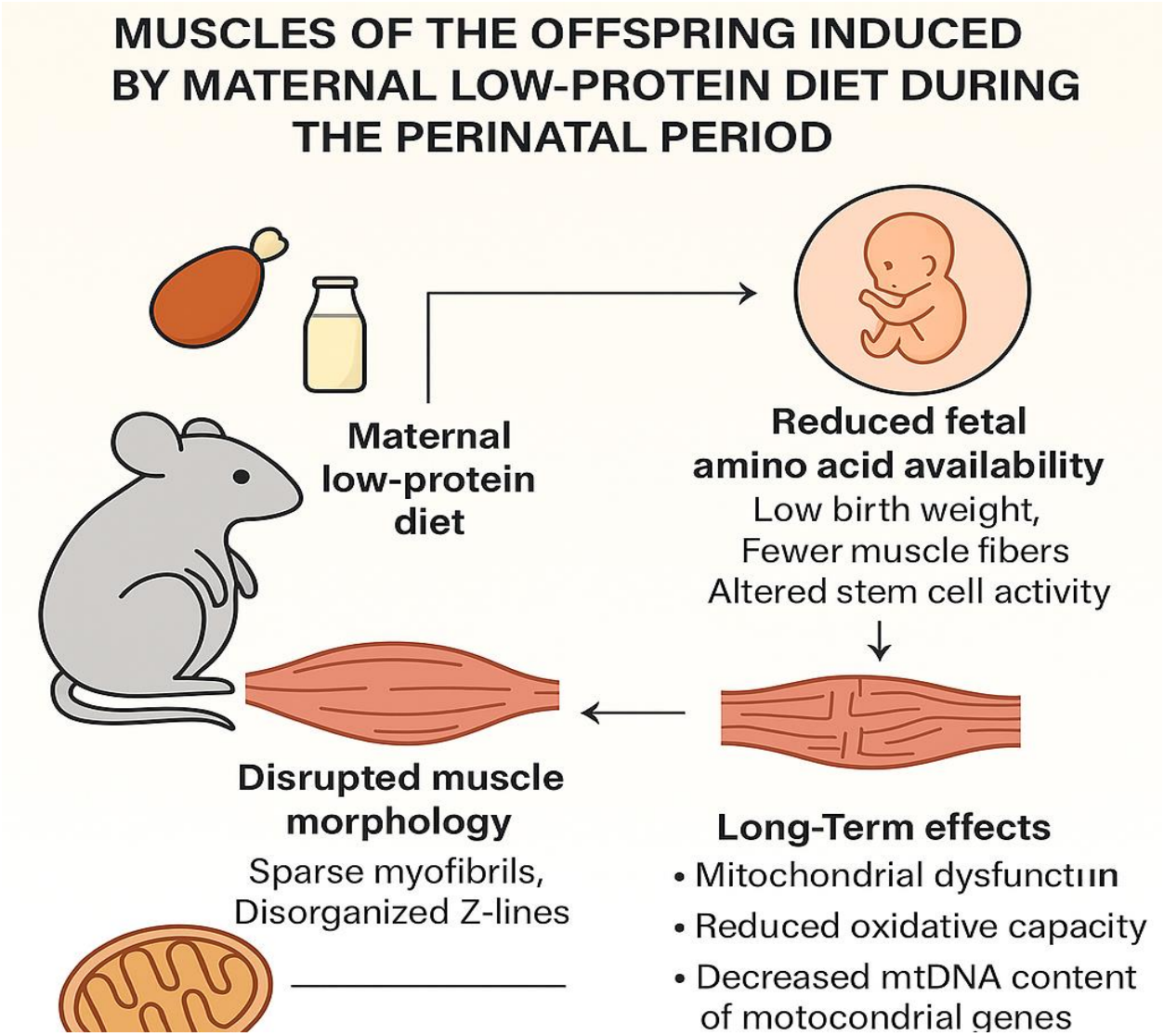


Fig. (5): Maternal low-protein diet reduces skeletal muscle protein synthesis and mass via the akt-mTOR pathway in adult rats [29].



Graphical abstract illustrating the effect of maternal Low-Protein diet on offspring skeletal muscle

Structural Alterations in The Skeletal Muscles of the Offspring Induced by Maternal High-Fat Diet during The Perinatal Period

1. Overview

Obesity and related metabolic disorders are increasing globally, particularly among younger populations. Emerging evidence indicates that adverse intrauterine environments, including maternal overnutrition, contribute to early-onset metabolic diseases in offspring [13]. Maternal obesity during conception and pregnancy elevates perinatal risks and imposes long-term health consequences on the offspring, consistent with the developmental programming hypothesis [34]. Studies have demonstrated that maternal high-fat (HF) diets during pregnancy and lactation predispose offspring to obesity, insulin resistance, cardiovascular abnormalities, and impaired skeletal muscle development [35].

2. Morphological and Metabolic Alterations

Experimental models reveal that perinatal HF intake leads to intramuscular lipid accumulation, cytokine activation, inflammation, and abnormal mitochondrial morphology in offspring skeletal muscle [36, 37]. Additional alterations include changes in fiber size and type, shifts in gene expression and metabolic pathways, and adaptive upregulation of lipid oxidation. However, such adaptations often occur at the expense of

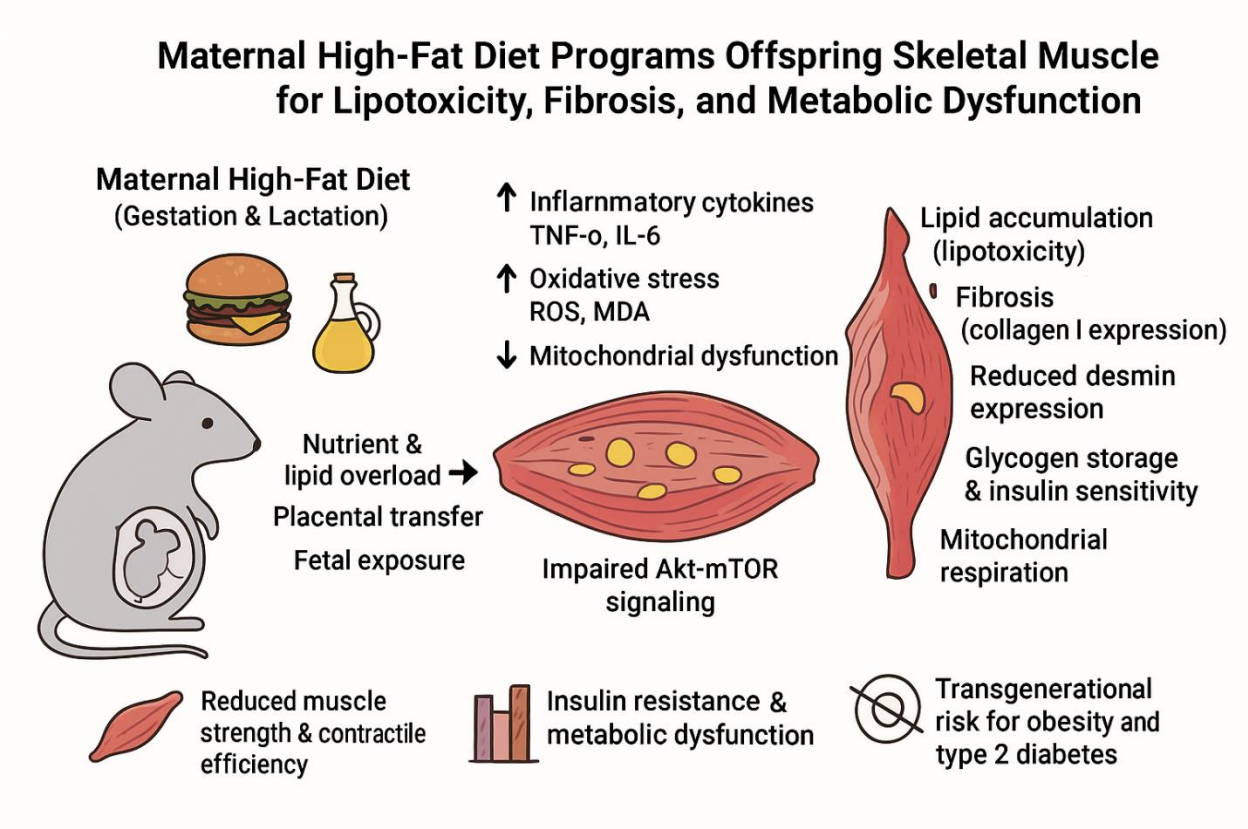
mitochondrial efficiency, ultimately compromising oxidative capacity and energy homeostasis [38, 39]. Notably, offspring of HF-fed dams exhibit aberrant mitochondrial morphology and reduced respiratory capacity in skeletal muscle [40]. The reversibility of HF diet–induced muscular and metabolic impairments remains inconsistent across studies. Some reports indicate that post-weaning dietary normalization partially restores mitochondrial function and reduces inflammation, whereas others document persistent oxidative and structural deficits [37–40]. Such discrepancies may stem from differences in fat composition, exposure duration, or offspring sex, emphasizing the need for standardized experimental protocols and human-based validation studies.

3. Structural and Biochemical Markers of Muscle Injury

Maternal HF diet exposure is associated with marked structural and metabolic disturbances in offspring skeletal muscle. These include elevated levels of muscle damage markers such as lactate dehydrogenase (LDH) and creatine phosphokinase (CPK) [41], as well as excess reactive oxygen species (ROS) production, leading to oxidative stress and mitochondrial dysfunction [42]. Histopathological findings reveal degenerative muscle alterations, lipotoxic infiltration, vascular congestion, and loss of fiber integrity [43–45].

4. Fibrosis, Insulin Resistance, and Cytoskeletal Disruption

Maternal HF intake also induces fibrotic remodeling, evidenced by increased collagen I expression and interstitial fibrosis in offspring skeletal muscle [46]. These structural changes coincide with reduced glycogen stores and enhanced insulin resistance, reflecting metabolic inflexibility and impaired glucose utilization [47]. Furthermore, elevated mast cell density and decreased desmin expression have been reported, reinforcing the link between maternal lipid overload, inflammation, fibrosis, and muscle disorganization in offspring [48–51].



Graphical abstract illustrating the effect of maternal High-Fat diet on offspring skeletal muscle

Limitations and Potential Biases

Although current evidence consistently links maternal malnutrition to structural and metabolic abnormalities in offspring skeletal muscle, several limitations must be acknowledged. Publication and selection biases may favor studies reporting positive outcomes, while variability in animal models, diet composition, and exposure periods reduces comparability and reproducibility. Confounding factors such as maternal stress, litter size, postnatal diet, and housing conditions are not always adequately controlled. Moreover, translation to humans remains limited due to interspecies differences in growth rate, metabolism, and genetic background. Addressing these gaps through well-controlled, longitudinal, and sex-specific human studies is essential to enhance the reliability and applicability of future findings.

CONCLUSION

Perinatal maternal malnutrition profoundly affects skeletal muscle development in offspring. Both low-protein and high-fat diets during pregnancy and lactation disrupt myogenesis, reduce fiber size, alter mitochondrial structure, and impair long-term muscle function. These findings reinforce the Developmental Origins of Health and Disease (DOHaD) hypothesis, highlighting how early nutritional environments shape lifelong musculoskeletal and metabolic health. Nonetheless, uncertainties persist regarding dose–response effects, reversibility of damage, and sex-specific susceptibility. Future research should prioritize longitudinal human studies linking maternal diet to offspring muscle outcomes, mechanistic investigations of epigenetic, mitochondrial, and inflammatory pathways, intervention trials to determine optimal nutrient balance, and comparative analyses across sexes and genetic backgrounds. Ensuring adequate maternal nutrition remains central to preventing lifelong skeletal and metabolic disorders.

Author contribution statement

All authors contributed equally and actively to the substantive work leading to this manuscript and accept joint responsibility for its content. Each author participated in writing, revising, and approving the final version.

Conflict of Interest: None

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Citation

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